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USING THE DIAMETRICAL MODEL TO EXAMINE THE RELATIONSHIP BETWEEN THE AUTISM AND PSYCHOSIS SPECTRA

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FACULTÉ DES SCIENCES SOCIALES ET POLITIQUES

INSTITUT DE PSYCHOLOGIE

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THÈSE DE DOCTORAT

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pour l'obtention du grade de

Docteur en Neurosciences

par

Guillaume SIERRO

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IMPRIMATUR

Le Décanat de la Faculté des sciences sociales et politiques de l'Université de Lausanne, au nom du Conseil et sur proposition d'un jury formé des professeurs

- Christine MOHR, directrice de thèse, Professeure à l'Université de Lausanne
- M. Ahmad ABU-AKEL, Maître d'enseignement et de recherche à l'Université de Lausanne
- Michel HERZOG, Professeur à l'Ecole Polytechnique Fédérale de Lausanne
- Thomas R. KWAPIL, Professeur à l'Université de l'Illinois à Champaign
- Jérôme ROSSIER, Professeur à l'Université de Lausanne

autorise, sans se prononcer sur les opinions du candidat, l'impression de la thèse de Monsieur Guillaume SIERRO, intitulée :

« Using the diametrical model to examine the relationship between the autism and psychosis spectra »

Jean-Philippe LERESCHE
Doyen

Lausanne, le 28 février 2018

Abstract:

Schizophrenia and autism spectrum disorders (SSD; ASD) share clinical features, although considered distinct. Theories contrast ASD and SSD social cognition. The reasoning for this thesis is based on dimensional models of personality spanning from the healthy to pathological variations. Under this scenario, do some healthy autistic traits oppose to schizotypic ones on a Mentalism continuum? Also, does this psychometric opposition correspond to a behavioural one, f.i. in processing face and gaze? First, we validated schizotypic and autistic trait questionnaires in French. Second, we identified shared and diametrical traits. Third, we conducted 3 experiments to measure face pareidolia-proneness. We expected larger pareidolia-proneness with larger positive schizotypy, and smaller autistic trait scores. Fourth, we assessed gaze direction discrimination, and gaze cueing of attention. We expected larger sensitivity to gaze with larger positive schizotypy, but a smaller one with larger autistic traits. Psychometrically, we replicated oppositions between autistic mentalizing deficits and positive schizotypic traits. Although pareidolia-proneness was unrelated to personality, configural face processing was impaired with larger positive schizotypy, but preserved with smaller autistic mentalizing deficits scores. Also, gaze sensitivity was decreased in men with larger autistic mentalizing traits, but unassociated with positive schizotypy. Our results partially support ASD-SSD opposition in social cognition, to be further confirmed by future studies. Pareidolia-proneness may be better measured using other measurement strategies. Gaze direction attribution might better contrast ASD and SSD. Comparisons of resembling disorder-related phenotypes is promising for understanding underlying aetiological mechanisms, notably using a transdiagnostic approach associating personality, cognitive styles, endophenotypes, and multidimensional or network models.

Keywords:schizotypy; personality traits; autism; face processing; gaze processing

Résumé:

Les troubles des spectres schizophréniques et autistiques (TSS; TSA) sont cliniquement ressemblants, mais catégoriellement distincts. Des théories opposent la cognition sociale des TSA et TSS. Le raisonnement de cette thèse se base sur les modèles dimensionnels de la personnalité comme reliant normal et pathologique. Aussi, certains traits autistiques s'opposent-ils aux traits schizotypiques ? Une opposition psychométrique correspond-elle à une opposition comportementale, i.e. dans le traitement des visages et du regard ? Premièrement, nous avons validé les questionnaires de personnalité schizotypiques et autistiques. Deuxièmement, nous avons identifié les traits partagés et opposés. Troisièmement, nous avons conduit 3 expériences sur la paréidolie facial, que nous attendions associée à plus de schizotypie positive et moins de traits autistiques. Quatrièmement, nous avons examiné la discrimination de la direction du regard et la redirection de l'attention par le regard, que nous attendions associées à plus de schizotypie positive et moins de traits autistiques. Au niveau psychométrique, nous avons répliqués les oppositions entre traits autistiques de mentalisation déficitaire et traits schizotypiques positifs. Bien que paréidolie et personnalité étaient sans liens, le traitement configural des informations faciales était péjoré avec plus de schizotypie positive, mais préservé avec plus de déficits autistiques de mentalisation. Aussi, la sensibilité au regard était moindre chez les hommes avec plus de déficits autistiques de mentalisation, mais sans lien avec la schizotypie positive. Nos résultats soutiennent partiellement une opposition TSA-TSS de la cognition sociale, à confirmer par de futures études. La tendance à la paréidolie gagnerait à être mesurée par d'autres stratégies. L'attribution de la direction du regard pourrait mieux distinguer TSA et TSS. La comparaison de phénotypes psychiatriques resemblants est une approche prometteuse pour comprendre des méchanismes étiologiques sous-jacents, notamment par une approche transdiagnostique associant la personnalité, les styles cognitifs, les endophénotypes, des modèles multidimensionels ou en réseau.

Mots clés:schizotypie; traits de personnalité; autisme; traitement facial; traitement du regard

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List of main abbreviations

ANOVA: analysis of variance

AQ: Autism Spectrum Quotient

ASD: autism spectrum disorders

ASp: Autistic Spectrum

BAP: Broader Autism Phenotype

EPQR-A: Eysenck Personality Questionnaire Revised and Abridged

EQ: Empathizing Quotient

CFA: Confirmatory Factor Analysis

FLO: face-like object

GD: gaze direction

GC: gaze cueing

IE: inversion effect

INV: inverted

OBJ: object

O-LIFE: Oxford-Liverpool Inventory of Feelings and Experiences

PC: principal component

PCA: principal component analysis

PCSF: Principal Component score of Shared Features

PCDF: Principal Component score of Diametrical Features

PD: personality disorder

PSp: psychotic spectrum

RT: reaction times

sO-LIFE: Short Oxford-Liverpool Inventory of Feelings and Experiences

SPQ: Schizotypal Personality Questionnaire

SPQ-B: Schizotypal Personality Questionnaire Brief

SSD: schizophrenia spectrum disorder

SSp: schizophrenia spectrum

ToM: theory of mind

UPR: upright

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1 INTRODUCTION

1.1 SHORT SUMMARY TO THE GENERAL INTRODUCTION

1.1.1 DEFINING AUTISM AND SCHIZOPHRENIA SPECTRUM DISORDERS

Currently, Schizophrenia Spectrum[1] Disorders (SSD) and Autism Spectrum Disorders (ASD) are distinct diagnostic categories (American Psychiatric Association [APA], 2013). In *DSM-5* (APA, 2013, pp. 50-51), ASD is (i) a set of persistent deficits in social communication and social interactions across multiple contexts (e.g. deficits in social-emotional reciprocity, deficits in nonverbal communicative behaviours such as eye contact), and (ii) restricted, repetitive patterns of behaviour, interests or activities (e.g. motor stereotypy, restricted and fixated interests). Importantly, the current diagnosis of ASD encompasses infantile autism, childhood autism, Kanner's autism, Asperger's syndrome, pervasive developemental disorders 'not otherwise specified' and atypical autism, but excludes Rett's syndrome. Also, language delay is no more a core symptom of ASD but a specifier, reducing the groups of symptoms from 3 to 2. ASD refers to a set of neurodevelopmental disorders (APA, 2013) with an important genetic component (Tick, Bolton, Happé, Rutter, & Rijsdijk, 2015). Throughout this thesis, we will generally refer to the broader category of Autism Spectrum (ASp), to encompass the clinical disorders *and* personality traits sharing a common liability[2]. We will explain later the relationships between ASD clinical disorders and healthy autistic personality traits.

Schizophrenia *per se* comprises symptoms such as (i) delusions, hallucinations, disorganized speech (e.g. frequent derailment or incoherence), grossly disorganized behaviour or catatonic behaviour, negative symptoms (i.e. affective flattening, alogia or avolition) during more than 1-month, (ii) social or occupational dysfunction since the onset of the disturbance (i.e. in work, interpersonal relations or self-care), (iii) persistence of symptoms for at least 6 months, (iv) with exclusion of schizoaffective and mood disorders (APA, 2013). However, schizophrenia is a paradigmatic disorder among a family of phenomenologically related disorders, such as schizoaffective, schizophreniform, or brief psychotic disorders (APA, 2013). At least three types of

[1] In psychopathology, the term "spectrum" refers to a set of closely related mental disorders, typically arranged on a dimension, featuring shared symptoms, genetic and environentmanl risk factors, possibly shared neural substrate, and overlapping aetiologies (APA, 2013; Maser et al., 2009).

[2] For further detail about the relationships between schizotypic personality traits and SSD, as well as between autistic traits and ASD, please refer to Chapter 1.2.3.

models were proposed to account for the relationships between these disorders: (i) a unitary model (only one form of psychosis diverges in multiple clinical presentations), (ii) categorical models (schizophrenia features discrete illness entities, differing in symptoms and outcomes), and (iii) dimensional models (a continuum in psychosis ranges from healthy traits to clinical symptoms; Ritsner & Gottesman, 2011). As ASD, SSD refers to a set of neurodevelopmental disorders (Weinberger, 1987; see also Fatemi & Folsom, 2009) with an important genetic component (Hilker et al., 2017). Importantly, we will consider the wider category of SSD or functional psychoses that groups disorders associated by partly common genetic liabilities on a continuum of severity ranging from schizophrenia at the extreme clinical end, through other disorders (schizoaffective disorder, but also major depressive and bipolar disorders), to personality disorders (e.g. schizotypal, paranoid and schizoid personality disorders [PDs]; Ritsner & Gottesman, 2011). Critically, SSD or functional psychoses would have a multidimensional structure, classically positive, negative and disorganized symptoms (Ritsner & Gottesman, 2011). Throughout this thesis, we will generally refer to the broader category of Psychotic Spectrum (PSp), encompassing the clinical disorders and schizotypic personality traits[3], sharing a common liability (Kwapil & Barrantes-Vidal, 2015). We will explain later the relationships between SSD clinical disorders and healthy schizotypic personality traits. Now we briefly outline ASD-SSD relationships.

1.1.2 RELATED YET SEPARATE DISORDERS

Despite considered distinct since the 70^{ies} (*DSM-III*, APA, 1980; Kolvin, 1971; Rutter, 1972), PSp and ASp share a common history and clinical features. Historically, the term "autism" has been borrowed from schizophrenia research, to describe the condition that would later become ASD (Bleuler, 1911; Kanner, 1943)[4]. Clinically, the phenomenological resemblance of social withdrawal symptoms in ASp and SSp was observed at each level of severity, i.e. acute illness, PDs and personality (e.g. Konstantareas & Hewitt, 2001). Equally relevant here, it is common that studies report on comorbidities and misdiagnoses between ASp and PSp (e.g. Davidson, Greenwood, Stanfield, & Wright, 2014; Hofvander et al., 2009; Van Schalkwyk, Peluso, Qayyum,

[3] Throughout this thesis, we will use the noun "schizotypy" and its adjective "schizotypic" to qualify non-clinical personality features, also at the base of clinical conditions (see Kwapil & Barrantes-Vidal, 2015). In the clinical domain, we will use the adjective "schizotypal" in the expression "schizotypal personality disorder", and "schizophrenia", "schizophrenic" or any term juxtaposed to the noun "disorder".

[4] Using "autism" to describe both infantile autism and schizophrenia may be misleading. Indeed, Bleulerian autism differs from Kanner's autism in that the former is an active withdrawal whereas the latter is rather a passive withdrawn state resulting from cognitive problems (see Wing & Agrawal, 2003 for a discussion). Hence, "autism" may reflect distinct underlying processes between ASp and PSp.

McPartland, & Volkmar, 2015). Hence, research into PSp and ASp faces the paradox of two spectra of disorders that are phenomenologically related, but are considered separate. In the context of this paradox, it seems unsurprising that some researchers emphasize differences between ASp and PSp (Crespi & Badcock, 2008), whereas others emphasize their similarities (King & Lord, 2011; Rausch & Johnson, 2008). Important to the current project are psychometric studies that extend the phenomenological question from clinical populations' symptoms to general populations' personality traits.

1.1.3 DISORDERS, PERSONALITY, AND THE DIMENSIONAL VIEW

Eysenck (1957) considered mental disorders as extreme manifestations of healthy personality dimensions. Eysenck claimed that mental disorders were exaggerated expressions of biologically-rooted healthy personality features or traits (Claridge & Davis, 2003). Personality can be defined as "a complex pattern of deeply embedded psychological characteristics that are expressed automatically in almost every area of psychological functioning" (Millon, Grossman, Millon, Meagher, & Ramnath, 2004, p. 3). To measure these features, personality psychologists usually use questionnaires consisting in assertions about cognitions, emotions, behaviours, and various experiences. Respondents are required to indicate whether or not, and to what extent they endorse or not these assertions as corresponding to them. These responses are coded numerically, so that they quantify the amount of personality features from a given dimension (e.g. Extraversion) endorsed by an individual. These personality features are called personality traits. A personality trait is "a long-standing pattern of behaviour expressed across time and in many different situations" (Millon et al., 2004, p. 3). Hence, personality traits are quantitative representations of associated personality features or dimensions, continuously spanning from very low to very high quantities.

Personality traits and Eysenck approach imply a dimensional view of personality and psychopathology (Claridge & Davis, 2003), notably schizophrenia (Claridge, 1997), and autism (Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, 2001). At that time, psychiatric approach of psychopathology was dominant and implied a categorical approach of mental disorders, exemplified by various editions of *DSM*: *DSM-III, DSM-IV, DSM-IV-TR, and DSM-5* (APA, 1980, 1994, 2000, 2013). In contrast, Eysenck supported a dimensional model, on which individuals were characterized by the amount of their personality traits on a continuum ranging from healthy to clinical manifestations. A dimension refers to the abstraction of specific discrete features (i.e. traits) to more general sets of these features, through statistical analyses (e.g. factor analyses). After Eysenck's attempts, similar dimensional approaches emerged for disorders like schizophrenia,

developing the notion of schizotypic personality (Claridge, 1997; Meehl, 1990). Schizotypic personality was proposed to index the liability of one individual to develop schizophrenia. Likewise, dimensional approaches proposed autistic traits representing the liability to autism in healthy population (Baron-Cohen et al., 2001; Constantino & Todd, 2003).

Dimensional approaches of schizophrenia and autism assume that disorder features and their genetic liabilities are continuously present, i.e. from the clinical population (acutely affected patients) to healthy individuals showing the respective personality expressions (Baron-Cohen et al., 2001; Claridge, 1997; Constantino & Todd, 2003). To put it clearly, not only do resembling disorders from the same spectrum share common genetic liabilities, but these genetic liabilities extend in unaffected individuals from the general population and are reflected in personality traits. Recent studies supported these claims for both autistic traits (Lundström et al., 2012), and schizotypic traits (Grant, Balser, Munk, Linder, & Hennig, 2014; Tarbox & Pogue-Geile, 2011). Indeed, dimensional models consider personality as a disorder's marker, thereby allowing investigation of *latent* mechanisms and genetic liabilities, beyond *manifest* phenomenological similarities and differences.

1.1.4 PERSONALITY AND (ENDO)PHENOTYPES

Personality may be a marker to liability to a psychiatric disorder, itself helping in identifying other disorder markers (e.g. cognitive, biological), ultimately helping in targeting better candidate genes. Over the last decades, genetic studies remained unsuccessful in the identification of candidate genes responsible for either ASp or PSp (Geschwind, 2011; McDonald & Singh, 2011). Alternative to such genetic research are indirect approaches that aim to determine behavioural or biological disorder markers or endophenotypes (Gottesman & Gould, 2003; Lainhart & Lange, 2011; Ritsner & Gottesman, 2011; Sucksmith, Roth & Hoekstra, 2011). Endophenotypes are "measurable components unseen by the unaided eye along the pathway between disease and distal genotype" (Gottesman & Gould, 2003, p. 636). They can be neurophysiological, biochemical, endocrinological, neuroanatomical, cognitive, neuropsychological, or self-report data. To be considered an endophenotype, a given feature has to meet several criteria: (i) associated with the illness in the population, (ii) heritable, (iii) state independent, (iv) co-segregating with illness within families, (v) identified in patients' unaffected relatives at higher rate than in the general population (Hasler, Drevets, Gould, Gottesman, & Manji, 2006). Specificity was proposed to be an additional criterion for an endophenotype (Tsuang, Faraone, & Lyons, 1993). Importantly, studies showed that autistic and schizotypic personality traits associated with, respectively, ASD and SSD genes.

Thereby, personality traits satisfy an important condition to be endophenotypes (Grant et al., 2014; Lundström et al., 2012; see also Grant, 2015). Cognitive and behavioural endophenotypes were reviewed and discussed in both ASD and SSD (e.g. Snitz, MacDonald, & Carter, 2006; Sucksmith et al., 2011).

1.1.5 A COMPARATIVE STRATEGY

Investigating jointly autistic and schizophrenia markers as a function of personality traits may help improving our understanding of ASD and SSD aetiologies. Studying such healthy personality expressions resembling illness allows to investigate putative endophenotypes bypassing the clinical confounds associated to illness (e.g. medication), and collecting larger samples (i.e. increasing power). Also, jointly studying autistic and schizophrenia markers and endophenotypes may help distinguishing the shared, diametrical and distinct[5] features between ASp and PSp, and disentangling the respective aetiologies of their disorders (ASD, SSD), ultimately help targeting potential candidate genes (Craddock & Owen, 2011; Ettinger et al., 2015; Sucksmith et al., 2011). In this respect, understanding the relationships between autistic and schizotypic traits is the first step to identify shared and distinct cognitive markers (Ford & Crewther, 2014).

1.1.5.1 FOCUS ON DIFFERENCES

Even if studying similarities and differences are both promising and clinically relevant research domains, we believe that studying differences is a more promising research approach (Spek & Wouters, 2010; Wouters & Spek, 2011). We present three reasons below.

First, evidence for phenomenological similarities was already established and would not lead to a major increase in scientific knowledge for our understanding of ASp and PSp (Konstantareas & Hewitt, 2001; Lugnegård, Hallerbäck, & Gillberg, 2011, 2012). Reports of genetic similarities outnumber those of differences (Craddock & Owen, 2010; Crespi & Crofts, 2012). Social brain and social cognition impairments also show overlap between ASp and PSp (Burns, 2004; McAlonan et al., 2005). To explain the paradox of phenomenologically resembling yet separate disorders, one must necessarily find a difference at a certain level of the causal pathway

[5] Throughout this work, we distinguish three types of relationships between ASp and PSp features: "shared" refers to overlapping features, "diametrical" refers to diametrically opposite features (i.e. dependent, non-orthogonal), and "distinct" refers to different features (i.e. independent, orthogonal).

between genes and symptoms (i.e. aetiological pathway). Going beyond phenomenological similarities and testing whether underlying causal mechanisms are shared, diametrical or distinct between ASp and PSp would be a progress. Indeed, shared or diametrical mechanisms would indicate that shared candidate genes sets should be targeted, and distinct mechanisms would indicate that distinct candidate gene sets should be targeted (Gottesman & Gould, 2003).

Second, from a purely logical point of view, similarity (as *absence* of differences) cannot be scientifically proven, while difference (as *presence* of at least one difference) can be. We derive this argument from popperian falsificationism (Popper, [1934] 2005). Indeed, similarity between two phenomena (e.g. A, B) implies a universal proposition (i.e. "*all* features are similar between A and B"), while difference between two phenomena implies an existential proposition (i.e. "there *exist at least one* feature for which A and B are different"). A consequence is that searching for difference allows deductive reasoning while searching for similarities only allows inductive reasoning. Scientific research favours deduction over induction, because the former but not the latter can *falsify* a proposition (i.e. prove that a proposition is false), thereby proving true the contraposition (i.e. opposite proposition). In this approach, a proposition cannot be judged *true*, but merely *not-yet-falsified* by empirical testing. A progress in knowledge occurs each time a proposition holds against test while concurrent propositions are falsified. The not-yet-rejected proposition is not getting true (because this proposition might be falsified by another empirical test) but gains *verisimilitude* (i.e. is more believable).

More precisely, in a context dominated by evidence of similarities, as ASp-PSp, showing the existence of yet another similarity between A and B will incline us to *induce* that there are other such similarities and that both A and B are similar, but will not enable us to *deduce* it. The proposition "A and B are similar" will be spurious. In contrast, showing the existence of at least one difference between A and B allows us to falsify the first proposition, i.e. to *deduce* that the proposition "all features are similar between A and B" is false. Conversly, we will be sure that the contraposition of this proposition, "*not* all features are similar between A and B", is true. We believe that this reasoning (i) applies to ASp-PSp relationships, (ii) still holds for weaker propositions such as "a given *subset* of features (e.g. negative symptoms) are similar between A and B (e.g. ASp and PSp)", (iii) is relevant in the current context in which most studies emphasize ASp-PSp similarities, and (iv) is helpful to understand whether the latent *mechanisms* underlying apparently similar clinical manifestations are or are not similar.[6]

[6] We are aware of theoretical limitations and criticisms of popperian falsificationism. However, our falsificationist argument (i.e. focusing on existential propositions instead of universal ones) is of *practical*

Third, theories proposed a distinction between ASp and PSp, grounded on testable cognitive hypotheses, potentially leading to improved understanding. Some theories emphasized descriptive and cognitive similarities at the clinical level leaving differences to unknown genetic and biological endophenotypes (Rausch & Johnson, 2008). Other theories posited partially opposite personality profiles, and cognitive styles, stemming from genetic differences (Brosnan, Ashwin, Walker, & Donaghue, 2010; Crespi & Badcock, 2010; Crespi, Stead, & Elliot, 2009). Obviously, the latter theories, and not the former ones, allow directly testable cognitive hypotheses, because they posit existence of *opposite differences*. Also, the need for theory-driven hypotheses to target meaningful differences and avoid false-positives further justifies the focus on testable differences.

1.1.6 OUR GOALS

As for personality, few studies have a priori attempted to *differentiate* autistic and schizotypic traits (e.g. Dinsdale, Hurd, Wakabayashi, Elliot, & Crespi, 2013; Ford & Crewther, 2014) and to link the outcome of such psychometric studies to behavioural and cognitive measures (Russell-Smith, Mayberry, Bayliss, & Sng, 2012). Accordingly, we pursued two major goals when conducting our studies.

The first goal was to assess the psychometric relationships between schizotypic and autistic traits in healthy populations using validated French versions of standardized questionnaires. In particular, we were interested in identifying which schizotypic and autistic dimensions are related or overlapping (i.e. "shared") and which ones differentiate or oppose between schizotypic and autistic traits (i.e. "diametrical"). To achieve this goal, we had to validate each instrument using a French-speaking undergraduate population.

The second goal was to test how shared and diametrical features of schizotypic and autistic traits would correlate with behaviour. We particularly selected cognitive measures that should be sensitive to the underlying mentalizing abilities thought to oppose ASp and PSp, i.e. face and gaze processing (Crespi & Badcock, 2008). Guided by our psychometric results and the diametrical model, we expected that social cognition performance would be unrelated to "shared" schizotypic and autistic traits, but depend on the "diametrical"/opposite schizotypic and autistic traits, in particular when traits are related to Mentalism functions. In the first set of behavioural studies, we investigated face pareidolia-proneness as a function of diametrical and shared traits. We used a

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purpose in this work. It merely orients the research on testable differences instead of non-testable similarity, i.e. absence of differences. It does not pretend to circumvent possible *theoretical* flaws of falsificationism.

classic old-new recognition paradigm with inversion of part of the stimuli, and three stimuli categories: faces, face-like objects (FLOs), and objects. Stimuli inversion at retrieval allowed to investigate participants' reliance on a configural face processing strategy, and to evaluate pareidolia effect of FLOs. In a second set of behavioural studies, we investigated gaze direction (GD) processing and gaze cueing (GC) liability as a function of diametrical and shared traits. We used a task of direction discrimination with gaze direction in whole-faces, gaze direction of eyes-only facial parts and directions of arrows. Also, we used a GC task featuring whole-faces' gaze and targets appearing either congruently or incongruently relatively to GD.

In what follows, we firstly present theories that explain the relationships between ASp and PSp. We emphasize their putative similar mechanisms and aetiologies, ultimately predicting *similarities for ASp and PSp.* Secondly, we present theories that emphasize *some* different aetiologies and mechanisms, ultimately predicting *differences between ASp and PSp*.

1.2 DIFFERENT THEORIES EXPLAINING ASP AND PSP RELATIONSHIPS

Here, we aim at providing a very partial and short summary of theories explaining ASp and PSp relationships. For the sake of simplicity and to better introduce our research, we will focus only on two group of theories[7]: those that emphasize the shared features between ASp and PSp and their allegedly shared aetiologies, versus those who emphasize diametrical features between ASp and PSp, and different aetiologies.

1.2.1 THEORIES EMPHASIZING PHENOMENOLOGICAL SIMILARITIES

Bender (1947, cited by Barneveld et al., 2011, p. 231) proposed that autism could be "an age specific expression of a developmental disorder that in adulthood is characterized by schizotypal symptoms". This theory did not survive the empirical evidence of distinction between autism and (childhood) psychosis (Rutter, 1972; Kolvin, 1971). As a result, *DSM-III* (APA, 1980) considered autism and psychosis as different diagnostic entities. Later, Volkmar and Cohen (1991) examined the prevalence of schizophrenia in individuals with autism. To put it differently, they

[7] Obviously, reality is not as binary as what we expose here. Multiple other models could be defined and compared, as admirably done by Chisholm, Lin, Abu-Akel, and Wood (2015). We will discuss these models in the 6th chapter, "General Discussion".

tested whether early autism predisposed to later schizophrenia. If both disorders were to be related, prevalence of schizophrenia should be higher in individuals with autism. However, Volkmar and Cohen found no larger prevalence of schizophrenia in individuals with autism, supporting the view both disorders were unrelated. At that time, the problem seemed to be solved.

More recently, several groups revived the idea that ASD and SSD resembling clinical manifestations reflect related aetiologies. Rausch and Johnson (2008) proposed a theory in which ASD and SSD are grouped on the same "negative symptoms spectrum". This theory is an attempt to explain similarities between autism, Asperger syndrome, schizotypal, and schizoid PDs, and schizophrenia. According to this theory, the various disorders would share common deficits in social competence (hence, the term "negative symptoms spectrum"), but also in "positive symptoms", which would consist of cognitive and motor expressions of a common liability to stereotypy. The social competence deficits would consist of either (i) afferent/perceptual deficits (e.g. deficits in emotion perception, social perception, and understanding others' intention or perception), and (ii) efferent/behavioural deficits (e.g. deficits in eye contact, facial expression, affect relatedness, speech, social interaction, nonverbal skills, postures). Cognitive stereotypies would consist of preoccupations, over-elaborate cognition, obsessions or restricted interests, but also delusions. Motor stereotypies would consist of simple (e.g. mannerism, motor tics, posturing), and complex forms (e.g. compulsions, routines, rituals). The authors raised the possibility that such stereotypies may be at the origin of deficits in social competences.

Yet, ASD and SSD are not entirely similar, and Rausch and Johnson (2008) also discussed what differentiates ASD and SSD symptoms expressions. Instead of fundamental differences between ASD and SSD, authors emphasized different symptomatic expressions of partly shared sets of genes. Overall, *different* combinations of genes (associated with negative, positive symptoms, developmental delay or pathology severity) would give rise to *different* cognitive profiles, ultimately causing *different* pathologies or subgroups. In particular, developmental delay and positive symptoms (i.e. delusional stereotypies) would play a decisive role shifting cognitive profiles either in ASD or SSD direction. Nevertheless, this model is limited relatively to the explanation of social cognition deficits. Although authors mention mentalizing deficits in autism and Asperger's syndrome, they do not discuss whether they are shared or distinct with those of schizophrenia and related PDs.

King and Lord (2011) also emphasized similarities between ASD and SSD, yet without elaborating on possible differences. To put it shortly, they considered that ASD and SSD share more features (i.e. genes, aetiological mechanisms) than the sole apparent ones (i.e. clinical features). The authors wondered whether schizophrenia would be on the autism spectrum, based on

evidence of overlaps in clinical, cognitive (i.e. theory of mind [ToM] deficits), neural (mirror neurons and connectivity deficits), pharmacological (similar treatments), and genetic domains. For these authors, these overlaps suggested *partially common* aetiologies. These authors did not conclude whether schizophrenia is on the autism spectrum, or vice versa, or whether a sub-population lays at the intersection between both spectra. However, they emphasized the importance of investigating autism and schizophrenia phenotypes together, on the ground of their similarities, yet without discussing differences. In a recent paper, De Lacy and King (2013) further developed this view. In line with Bender (1947), they considered that both ASD and SSD share similar underlying aetiology, and overlapping clinical presentations. Environmental demands and compensations during sensitive developmental periods would modulate the expression of disorders, their severity, their immediate or delayed onset. Differences between ASD and SSD would exist as epiphenomena, so that authors focus on the overlap in aetiologies and phenotypes.

More recently, various clinical and genetic accounts supported connections between ASD and SSD mostly on the ground of overlapping negative symptoms. Barneveld et al. (2011) showed that adolescents with schizotypic traits had more autistic traits in childhood than typically developing controls. About negative symptoms, Kaiser, Heekeren and Simon (2011) claimed that negative symptoms were not specific to SSDs, and extended towards healthy personality, under the form of a mood-psychotic disorders spectrum (van Os, 2009; van Os & Kapur, 2009). Other authors proposed that negative symptoms would go further than the mood-psychotic spectrum, encompassing related PDs, attention/hyper-activity disorders, and possibly also autism (Malaspina et al., 2014; Foussias, Agid, Fervaha, & Remington, 2014). Based on genetic considerations, Craddock and Owen (2010) put into question the unrelatedness of autism, schizophrenia and various other disorders such as mood and bipolar disorders (see also: Carroll & Owen, 2009).

Obviously, the aforementioned theories (De Lacy & King, 2013; King & Lord, 2011) explain similarities but not differences, impeding the generation of testable predictions. Of course, none of these authors said that ASD and SSD are totally similar and share the exact same aetiologies. Also, none of these authors claim that ASD and SSD do not show clinical or behavioural differences. Yet, their models simply focus on shared features between ASD and SSD, without proposing explanations of their clinical and behavioural differences. In contrast, research on the disorders' markers or endophenotypes would gain not only from the knowledge about similarities but also about differences (Gottesman & Gould, 2003). In addition, these theories did not extend their reflections to the healthy end of ASD and SSD continua, that is ASp and PSp. In contrast, other theories emphasizing differences opposed ASp and PSp phenotypes, including the healthy end, with regard to social brain and social cognition (Abu-Akel, 1999; Brosnan et al., 2010;

Crespi & Badcock, 2008). Importantly, the latter theories propose testable hypotheses on mechanisms underlying *apparent* similarities and differences between ASp and PSp clinical, personality and behavioural features. We present these theories below, after having defined the social brain and social cognition onto which they rest.

1.2.2 TWO THEORIES EMPHASIZING PHENOMENOLOGICAL DIFFERENCES

1.2.2.1 DIFFERENCES IN SOCIAL BRAIN AND SOCIAL COGNITION

The two models emphasizing differences are based, at least partly, on an opposition of ASp and PSp in social brain and social cognition. Notwithstanding phenomenological similarities between ASp and PSp (f.i. negative symptoms), these theories attempted to oppose at least *some* features of ASp and PSp, mostly social brain and social cognition (Brosnan et al., 2010; Crespi & Badcock, 2008). Although ASp and PSp share *apparently similar* social deficits, these may be caused by *different* mechanisms pertaining to social cognition or the social brain (equifinality; Cicchetti & Rogosch, 2002). To put it shortly, these theories consider that ASp and PSp share fewer features (i.e. genes, aetiological mechanisms) than they apparently do (i.e. clinically).

Studies reported social cognition impairments in ASD and SSD. Research on healthy end of ASp and PSp is still scarce. Social cognition refers to any kind of "processing that is elicited by, about, and directed towards other people (or, more species-general, towards conspecifics)" (Kennedy & Adolph, 2012, p. 559). Social cognition encompasses various abilities used in interaction with others, such as ToM/mentalizing, social perception[8], attribution, self-knowledge (Amodio & Frith, 2006). Important for us, social cognition's such as ToM or gaze processing show

[8] Social perception has a broad meaning and deserves some detailed definitions. Social perception is a subset of social cognition that refers to the understanding of information conveyed by face, mouth or gaze (Adolphs, 2001). Allison, Puce and McCarthy (2000, p. 275) define social perception "as part of a larger domain of cognitive skills referred to as theory of mind, mentalizing, social attention, and social cognition, which are defined as the processing of information which culminates in the accurate perception of the dispositions and intentions of other individuals." For Lieberman and Pfeifer (2005, p. 212), social perception is even wider and refers to the ability of "understanding the personality, intentions, beliefs and identity of others", that comprise recognizing others (notably face recognition), emotion recognition, attribution of intentions, beliefs, desires and enduring psychological traits to others, and stereotyping, such as categorizing based on social group membership. For the present manuscript, we will retain social perception as the perception and understanding of face and gaze cues essential to understand and attribute intentions, mental states to others.

deficits in ASD and SSD (Baron-Cohen & Belmonte 2005; Brüne, 2005; Frischen, Bayliss, & Tipper, 2007).

Social cognition relies on a set of intact brain regions belonging to the "social brain", reportedly altered in ASD and SSD. The social brain refers to certain brain areas subserving social processes, which are mostly orbital and medial prefrontal cortex, but also include amygdala and superior temporal sulcus (Kennedy & Adolph, 2012). More precisely, the social brain can be considered as a set of networks, associated with different cognitive functions (i.e. simulation/mirror/action-perception network, mentalizing network, empathy network, amygdala network; Kennedy & Adolphs, 2012). Additional brain areas are associated with the social brain, although not constituting the social brain per se (e.g. anterior cingulate cortex, temporo-parietal junction [TPJ], superior temporal sulcus [STS], and temporal poles). Congruent with social cognition deficits, ASD (McAlonan et al., 2005) and SSD (Burns, 2004) feature anatomic and functional deficits of social brain networks. Still, although both ASD and SSD share social cognition and social brain impairments, it does not follow these impairments stem from similar underlying causal mechanisms.

1.2.2.2 MENTALISM CONTINUUM AND THE DIAMETRICAL MODEL

Crespi and Badcock (2008) further proposed that ASp conditions and PSp conditions would be *diametrically opposite* disorders of the social brain and social cognition, leading to opposite cognitive profiles in healthy ends of ASp and PSp. From now on, we refer to this theory or model as the "diametrical model". The diametrical model is composed of two elements: (i) dysregulated genomic imprinting, and the resulting (ii) diametrical opposition between ASp and PSp features (Badcock, 2004). In the following two paragraphs, we will briefly explain Crespi and Badock's (2008) theory from one level to another, i.e. from gene to behaviour.

The diametrical model rests on evolutionary and genetic theories: inclusive fitness theory or conflict theory, and genomic imprinting (Crespi & Badcock, 2008). Inclusive fitness theory explains the evolution of mother-offspring interactions by taking into account the amount of resources extracted from mothers by offspring (i.e. to what extent the offspring is demanding). Obviously, the more resources a mother gives to her offspring, the better the offspring fares, but the more costly for the mother. On the other hand, the less resources the offspring demands, the better the mother fares. Genomic imprinting refers to the silencing of certain genes. The silencing of certain genes (e.g. paternal ones) will lead to relative over-expression of non-silenced ones (e.g.

maternal ones). Importantly, the imprinting or silencing of genes plays a role in the conflict theory or inclusive fitness theory. Indeed, the expression of paternal genes in the offspring leads to a more demanding phenotype (costly for the mother), whereas the expression of maternal genes in the offspring leads to a less demanding phenotype (less costly for the mother). Hence, there is a conflict between paternally and maternally imprinted genes, influencing offspring's phenotype by altering brain development.

Biologically, these paternally and maternally imprinted genes mostly express in the brain and influence brain growth. In human extreme pathological syndromes, over-expressed paternal genes cause an overgrowth, whereas over-expressed maternal genes cause an undergrowth. Other syndromes such as Angelman and Prader-Willi syndromes also show over-expression of, respectively, paternal, and maternal genes. The former shows autistic features (e.g. autistic traits, stereotyped behaviour, seizures, larger body weight), whereas the latter shows psychotic-like features (e.g. increased incidence of psychosis, enlarged ventricles, dopaminergic abnormalities). Crespi and Badcock (2008) proposed that ASp and PSp conditions would be opposed disorders of the social brain, caused respectively by a paternally-biased and maternally-biased gene expression.

In their extensive review, Crespi and Badcock (2008) attempt to substantiate this opposition of social brain at different levels: neurobiological, anatomic, functional, cognitive, and clinical. As paradigmatic examples, they focus on autism vs. psychosis comparison. For instance, in autism, we observe higher birth weight/height, faster growth, higher levels of different growth factors, larger brains, larger and more reactive amygdala, and under-development of mirror neuron system, and hypoactive dorsomedial frontal cortex (involved in mental attribution) when compared to controls. In contrast, in psychosis we observe low birth weight and length, slow growth, lower level of growth actors, smaller brain size, smaller and less reactive amygdala, dysregulated mirror neuron system, and hyperactive dorsomedial frontal cortex when compared to controls. Some of the mentioned brain areas (e.g. amygdala, mirror neuron system, dorsomedial frontal cortex) belong anatomically and functionally to the social brain, and are involved in social cognition. Hence, autism and psychosis would be opposed relatively to social brain anatomy and function subserving social cognition.

Crespi and Badcock (2008) assume that cognitive dysfunctions in autism and psychosis would be caused by altered interactions between and within components of the social brain. For instance, amygdala would be overdeveloped and hyper-activated in autism which would contribute to gaze avoidance (Dalton et al., 2005), ultimately impairing face recognition, gaze processing, and ToM. In contrast to autism, psychosis would be characterized by an opposite over-responsiveness and over-sensitivity to gaze, due to mistaken inferences about direction or mental states based on

gaze (Hooker & Park, 2005; Langdon, Corner, McLaren, Coltheart, & Ward, 2006). The consequence of social brain and social cognition deficits would be measurable social deficits, that are biased into opposite directions in autism versus psychosis spectra.

Although relating clinical extremes, the diametrical model is not limited to clinical populations (e.g. autism, psychosis) but extends towards healthy populations, manifesting in healthy personality and cognitive styles (i.e. ASp, PSp). At the cognitive level, Crespi and Badcock's theory implies corresponding diametrical or opposite cognitive styles, in particular with regard to healthy social cognition. Although there is no consensus about the definition of cognitive styles, they may be defined as "individual differences in the ways people perceive, think, solve problems, learn, and relate to others" (Witkin, Moore, Goodenough, & Cox, 1977; cited by Kozhevnikov, Evans, & Kosslyn, 2014). Also, this consideration of opposite autism and psychosis phenotypes would extend to healthy population personality, following ASp and PSp dimensional models. Indeed, autistic traits and schizotypic traits are qualitatively similar but quantitatively attenuated manifestations resembling those of autism and psychosis, respectively (see Chapter 1.3.1). Hence, Crespi and Badcock propose that the autistic cognitive style would be associated with autistic traits, whereas the psychotic one would be associated with schizotypic traits. In particular, the diametrical model posits that symptoms or deficits related to social cognition are the most relevant to distinguish between ASp and PSp, broadly.

At a clinical level, the diametrical model is based on the clinical opposition between autism and psychosis, on Mentalism and Mechanism[9] continua, as proposed by Badcock (2004). Badcock proposed that autism and psychosis would oppose with regard to hypo- or hyper-development of Mentalism (i.e. ability to understand others, adopting a human perspective) and Mechanism (i.e. ability to understand the world as a system, outside a human perspective). Defined this way, Mentalism and not Mechanism is relevant for social cognition. To put it shortly, the autistic cognitive profile would feature underdeveloped or hypo-Mentalism, whereas the psychotic one would feature overdeveloped or hyper-Mentalism (Badcock, 2004). Hypo-Mentalism would be expressed in deficits in gaze processing, ToM, understanding of metaphor, whereas hyper-Mentalism would be expressed in over-sensitivity to gaze, preserved or enhanced ToM, and paranoia. From there, the diametrical model can be extended to personality (i.e. ASp, PSp), assuming that *some* schizotypic traits represent hyper-Mentalism, and *some* autistic traits represent hypo-Mentalism, themselves associated to milder social cognition and social brain features

[9] Throughout this thesis, we capitalized "Mentalism", "Mechanism", as well as their respective adjectives "Mentalistic" and "Mechanistic" each time they refer to Badcock (2004) and Crespi and Badcock (2008) diametrical model. We wanted to avoid any confusion with similar terms with distinct meaning (e.g. "a mechanism").

(Dinsdale et al., 2013). Given the emphasis on social cognition for the diametrical model, we will only explore Mentalism.

Other views support the relevance of and relationships between ASp and PSp, and were expressed by Abu-Akel (1999), Abu-Akel and Bailey (2000), and C. Frith (2004). As for theoretical models, only another one distinguishes between ASp and PSp in a comparable way to Crespi and Badcock (2008), that is: the extended version of Empathizing-Systemizing theory (Baron-Cohen, 2002; Brosnan et al., 2010).

1.2.2.3 THE EXTENDED EMPATHIZING-SYSTEMIZING THEORY

Brosnan et al. (2010) support a similar model than Crespi and Badcock (2008), yet originating from a different tradition. Brosnan et al. (2010) essentially built on Baron-Cohen (2002) "Empathizing-Systemizing" theory (E-S theory), applied it to psychosis, resulting in a model comparable to the one of Crespi and Badcock (2008). Instead of detailing E-S theory, we will focus on the relevant part of this theory, i.e. its use of social cognition for distinguishing between ASp and PSp. Analogue to Crespi and Badcock's Mentalism, Empathizing refers to a broad ability for empathy with a cognitive component (i.e. ability to attribute intention to others) and an affective component (i.e. ability to feel others' emotion, sympathy), whereas Systemizing refers to the drive to analyse systems, predict their behaviour and construct them (e.g. technical, motoric, natural. abstract, social) Baron-Cohen, Richler, Bisarya, Gurunathan, & Wheelwright, 2003, p. 361; see also Baron-Cohen, 2002; Baron-Cohen, 2005, pp. 239–255). Indeed, E-S theory, together with "extreme male brain theory", proposed that men show lower psychometric Empathizing as compared to women (Baron-Cohen, 2002). In this context, ASp individuals' mentalizing deficits correspond to an Empathizing deficit, which "extreme male brain theory" interprets as exaggerated male features. Brosnan et al. (2010) conjectured whether psychosis would be characterized by the converse, an "extreme female brain", i.e. exaggerated female features, larger empathizing abilities over Systemizing. These authors found psychometric evidence that increased positive symptoms (i.e. paranoia and mania) associated with increased empathizing, independently from depression or anxiety symptoms. Hence, not only individuals with autism show a lack of empathizing (Wakabayashi, Baron-Cohen, & Wheelwright, 2006b), but those high in positive schizotypy and psychotic traits show more empathizing, as proposed by the diametrical model. Importantly, larger Empathizing Quotient (EQ) scores predicted better sex discrimination of degraded faces (Penton-Voak, Allen, Morrison, Gralewski, & Campbell, 2007), better identification of emotion conveyed by gaze in healthy children (Chapman et al., 2006), and larger GC (Alwall et al., 2010), and more

bilateral processing of faces (N170), and increased fusiform gyrus activation when viewing faces as compared to houses (Lazar, Evans, Myers, Moreno-De Luca, & Moore, 2014).

As a result both theories distinguish ASp and PSp cognitive profiles, notably using social cognition, from perception of simple social cues (e.g. faces) to higher-level understanding of other's intentions. Yet, these should be defined before we continue in our reasoning.

1.2.2.4 THEORY OF MIND, MENTALIZING, EMPATHIZING, AND MENTALISM: CLARIFYING DEFINITIONS

Theory of Mind, Mentalizing, Empathizing, and Mentalism have overlapping meanings but only the two latter ones are broad enough to encompass social cognition abilities, such as face and gaze processing. Theory of mind was coined by Premack and Woodruff (1978) and is synonymous for "folk psychology, mind-reading, mentalizing, meta-representation or secondary representation, [and] is the ability to understand the psychological or mental states of other individuals, such as their beliefs, desires and knowledge" (Emery, 2005, p. 118). Frith and Frith (2003) used the terms "mentalizing" and "theory of mind" (ToM) synonymously, yet preferring the former to the latter. Their definition of mentalizing is broader: the ability to understand one's and others' thoughts, feelings, and beliefs as distinct from reality. Amodio and Frith (2006, p. 273) further precise mentalizing as the "ability to represent another person's psychological perspective". More recently, Kennedy and Adolphs (2012, p. 563) focused on nonaffective components, defining mentalizing as the ability to "represent other people's intentions and beliefs".

Baron-Cohen (2002) defined the concept of Empathizing as encompassing ToM, mindreading, taking the intentional stance, yet adding an expressive dimension to Frith and Frith's mentalizing. Indeed, Empathizing involves two abilities: the ability to attribute mental states to oneself and others, as a natural way to understand agents (i.e. attributional component or ToM), and the ability to have an appropriate emotional reaction to another person's mental states (i.e. affective component or sympathy; Baron-Cohen, 2005).

Mentalism has a wider meaning than Empathizing, as it encompasses social cognition abilities broadly (Crespi & Badcock, 2008). Indeed, Mentalism refers to folk-psychology, to the set of abilities necessary for social interactions. Nevertheless, both Empathizing and Mentalism definitions are broad enough to include the social cognition functions involved in mentalizing and ToM (e.g. through joint attention; Emery, 2000), such as social perception abilities like face and gaze processing abilities.

Hence, Mentalism and Empathizing are broad concepts integrating not only ToM/mentalizing but also the more easily observable social cues processing, thought to distinguish ASp and PSp. In addition, both Mentalism and Empathizing imply an extension from clinical domain to the one of healthy personality. As a result, clinical social cognition deficits are expected to express as healthy cognitive styles (adaptive or not), and clinical symptoms would express as personality traits. Below, we discuss how the field of healthy individual differences, via personality, can be used to investigate ASp-PSp relationships.

1.3 USING PERSONALITY TRAITS TO INVESTIGATE ASP-PSP RELATIONSHIPS

Using healthy personality traits to test nonclinical ends of ASp and PSp is supported by empirical, theoretical and practical reasons. We detail some of these reasons below.

Historically, the influential personality model of Eysenck (1957) interpreted individual differences in personality as biologically rooted, and as attenuated features similar to clinical disorders. Later, the relevance of autistic traits for ASp (Baron-Cohen et al., 2001), and schizotypic traits for PSp (Claridge, 1997) was justified by similar assumptions. Recent studies support the ability of autistic and schizotypic traits to target, respectively, ASp and PSp genetic liability (Grant et al., 2014; Lundström et al., 2012).

Theoretically, Crespi and Badcock (2008) assume the diametrical model would best be tested using personality questionnaires, i.e. schizotypy (Claridge, 1997), and autistic traits (Baron-Cohen et al., 2001). Likewise, Brosnan et al. (2010) based their claims of a psychotic "extreme female brain" on the psychometric assessment of healthy undergraduate females, and did so for further experiment (Brosnan, Ashwin, & Gamble, 2013; Brosnan, Chapman, & Ashwin, 2014; see also Russell-Smith, Mayberry, & Bayliss, 2010).

Practically, testing healthy population samples (typically undergraduate samples) with individuals assessed for schizotypic and autistic traits offers several advantages, explaining the popularity of this approach. Healthy participants are more numerous, more available, and it is less costly in the case of students recruited for pedagogical or credits' requirements. This approach makes researches more feasible, facilitates recruitment, and often boosts the sample sizes, hence statistical power, as compared to other approaches. Testing healthy instead of clinical participants has additional advantages. Ettinger, Meyhöfer, Steffens, Wagner, and Koutsouleris (2014) argue that investigating schizotypy, that is SSD as a continuum (i) provides clues regarding schizophrenia

aetiology, (ii) provides clues regarding the maladaptive behaviours related to psychiatric symptoms (e.g. cigarette smoking, drugs), (iii) cheap, available and objective psychometric questionnaires, (iv) high statistical power (because of continuously distributed data), and (v) circumvent confounds of pharmacological treatments an institutionalization typical of schizophrenia. Kwapil and Barrantes-Vidal (2015) consider testing students not as a bias but as an even more conservative test, since students would benefit from protective factors. Finally, other studies studying psychiatric illnesses markers (or endophenotypes) tested undergraduate students (Cappe et al., 2012).

Yet, underlying the idea of using personality as a proxy for investigating ASp-PSp relationship, Eysenck's assumption that attenuated personality traits correspond to clinical symptoms has to be detailed and discussed. Below, we discuss Eysenck's and other dimensional approaches that attempted to bridge more or less continuously healthy personality and clinical symptoms.

1.3.1 DIMENSIONAL APPROACHES

Adopting a dimensional approach i.e. testing hypotheses about ASp-PSp in healthy populations makes sense, because healthy individuals show personality traits resembling those found in patients. Importantly, dimensional models assume that personality traits are qualitatively similar but quantitatively attenuated analogues to those of a corresponding disorder. Dimensional models were elaborated for various disorder groups such as anxiety, mood, bipolar disorder (Claridge & Davis, 2003), and crucially for our interest ASD (Baron-Cohen et al., 2001; Constantino & Todd, 2003) and SSD (Claridge, 1997; Meehl, 1990).

Personality traits corresponding to PSp and ASp are, respectively, schizotypic traits and autistic traits. For PSp, concepts of psychosis-proneness, prodromal personality, the personality of schizophrenia patients' relatives, and schizoid, schizotypal and paranoid PDs are relatively similar to the definition of schizotypy (see below; Kwapil & Barrantes-Vidal, 2015). Hence, we will globally talk about schizotypic traits to define the personality traits associated with SSD, and more generally PSp. For ASp, personality traits analogue to ASp are named autistic personality traits (Constantino & Todd, 2003). Similar autistic personality traits are found in ASp relatives (Piven, Palmer, Jacobi, Childress, & Arndt, 1997; Bailey, Palferman, Heavy, & Couteur, 1998). Yet, research generally takes care distinguishing personality features from ASp patients' relatives from those of the rest of population, by referring to the Broader Autism Phenotype (BAP; Piven et al., 1997; see also Wheelwright, Auyeung, Allison, & Baron-Cohen, 2010). In what follows we will

briefly summarize the history, and the main ideas of PSp and ASp dimensional models, respectively.

1.3.1.1.1 DIMENSIONAL APPROACHES OF PSP

1.3.1.1.1.1 AN OVERVIEW OF 3 DIMENSIONAL MODELS

The idea that certain personality traits would be attenuated versions of schizophrenia symptoms emerged early. Bleuler (1911) noticed that relatives of schizophrenia patients presented attenuated features of the illness. He considered these features as representing a "latent" type of schizophrenia, consisting in personality features resembling attenuated schizophrenia symptoms (e.g. withdrawn or exaggeratedly punctual, irritable, odd, and moody). This category can be considered as precursors of later categories of schizoid and schizotypal PDs (Kendler, 1985).

Later, Rado (1953) coined the term "schizotypy" to define a schizophrenic phenotype of individuals with a genetic liability to schizophrenia (Meehl, 1962, 1990). Meehl (1990) added that schizotypy comprised both the genetic and the environmental (i.e. psycho-social) vulnerability to schizophrenia. Meehl hypothesized that a single gene (schizogene) would cause an abnormality in neural response function (termed "hypokrisia "). The combination of this genetic neural abnormality (schizotaxia) with certain environmental factors might manifest as schizotypy, and eventually as schizophrenia. Importantly, since Meehl model assumed the role of a putative gene (schizogene), only the carrier of this gene may manifest schizotypy or schizophrenia. For this reason, Meehl's model is *quasi-dimensional* (closer from the psychiatric "categorical" approach), and not *totally-dimensional* (like Eysenck's model, see below). The latest developments of quasi-dimensional approach consider that carriers of the schizophrenia gene are more likely to develop schizophrenia (Lenzenweger & Korfine, 1992). Yet, no schizogene has yet been identified, and the number of candidate genes remains large, casting doubt onto the quasi-dimensional approach (Grant, 2015).

In contrast to quasi-dimensional models, other dimensional models conceived a more continuous relationship between schizophrenia/psychosis-liability and corresponding disorders. Eysenck (1957) is the proponent of the second model, termed *totally-dimensional*. He considered that mental disorders were exaggerated forms of biologically-rooted healthy personality features. He proposed Psychoticism as a personality dimension predicting the liability to develop psychosis, or psychosis-proneness (Eysenck, 1952). Hence, he did not conceive any discontinuity between mental disorders and healthy personality. A third model, termed *fully-dimensional*, emerged as a consensus between quasi and totally dimensional approaches. For the *fully-dimensional* approach

(Claridge, 1997; Mason & Claridge, 2006), schizotypy is a healthy personality dimension, and higher scores on its dimensions increase the likelihood for schizophrenia, as evidenced by a recent review (Nelson, Seal, Pantelis, & Philipps, 2013). Hopefully, most high scorers in schizotypy will never develop the illness (Mohr & Claridge, 2015).

Among schizotypic dimensional models, Crespi and Badcock (2008) favoured Claridge's *fully-dimensional* model of schizotypy. Indeed, the fully-dimensional model fits with the diametrical models' ASp-PSp continuum better than the quasi-dimensional model does. Below, we detail the fully-dimensional model of schizotypy.

1.3.1.1.1.2 THE FULLY DIMENSIONAL MODEL

The fully-dimensional model of Claridge (1997; see also Claridge & Davis, 2003) is adequate to deal with ASp-PSp relationships theories in that it encompasses liability to schizophrenia and psychosis, and permits correspondingly broader definitions of schizotypy, in continuity with SSD, and more broadly PSp. The fully-dimensional model attempted to unify two kinds of dimensionalities: the continuum of personality dimensions *within the non-clinical healthy domain* (i.e. the eysenckian totally-dimensional model) and the continuum of symptoms severity/numbers *within the illness domain* (i.e. the categorical quasi-dimensional model). Hence, the fully-dimensional model claims, at the same time, that schizotypic traits belong to healthy personality traits, and that they predispose to schizophrenia. A moderate amount of schizotypic traits would remain adaptive, whereas, above a threshold into illness, they would become detrimental (Claridge & Davis, 2003; see also Mohr & Clardige, 2015).

In line with this fully-dimensional view, schizotypy was recently defined as "a multidimensional unifying construct that represents the underlying vulnerability for schizophrenia-spectrum psychopathology that is expressed across a broad range of personality, subclinical, and clinical psychosis phenomenology [… assuming that the] same etiological, developmental, and phenomenological processes underlie subclinical and clinical manifestations" (Kwapil & Barrantes-Vidal, 2015, p. S368). With such a broad definition, other concepts such as prodrome, schizotypal PD, and psychotic patients can be considered as parts of the schizotypy continuum, and as specific expressions of PSp liability. Hence, self-report questionnaires based upon this definition of schizotypy are expected to capture the various aspects of SSD liability, including psychosis, thus representing the PSp.

Self-report questionnaires usually permit the measure of schizotypic traits, grouped in dimensions corresponding to schizophrenia symptoms. For instance, schizotypy self-report questionnaires include the Oxford Liverpool Inventory of Feelings and Experiences (O-LIFE, Mason, Claridge, & Jackson, 1995), the Schizotypal Personality Questionnaires (SPQ; Raine, 1991), and also the Wisconsin Schizotypy Scales (WSS: Perceptual Aberrations, Magical Thinking, Physical Anhedonia, Social Anhedonia: Chapman, Chapman, & Raulin, 1976; Chapman, Edell, & Chapman, 1980; Eckblad & Chapman, 1983; Eckblad, Chapman, Chapman, & Mishlove, 1982). Each of these questionnaires isolated a positive dimension (representing a.o. O-LIFE's Unusual Experiences [UnEx], cognitive and perceptive aberrations, magical thinking, reference thoughts), and a negative dimension (representing a.o. physical anhedonia, social anhedonia, and social interaction deficits; e.g. O-LIFE's Introvertive Anhedonia [IntAn]). O-LIFE and SPQ additionnally identified a dimension accounting for disorganization (a.o. disordered thought and language; e.g. O-LIFE's Cognitive Disorganization [CogDis]). These three personality dimensions line up nicely with the clinical evidence of three sets of symptoms or dimensions in schizophrenia patients: positive, negative, and disorganized symptoms (Arndt, Alliger, & Andreasen, 1991; Liddle 1987), although not formally including other psychotic traits (e.g. mania).

Yet, only the O-LIFE, and its short version the sO-LIFE (Mason, Linney, & Claridge, 2005), additionnally feature a dimension accounting for psychosis liability, named Impulsive Non-Conformity (ImpNon). ImpNon accounts for reckless, impulsive behaviour sometimes associated with schizotypy, as well as with hypomanic traits, and Eyesenck's Psychoticism scale (Claridge et al., 1996). ImpNon dimension was criticized as not being a genuine schizotypic dimension (Pickering, 2004). Yet, Mason and Claridge (2006) support the inclusion of this ImpNon in schizotypy, because it would account for complementary aspects related to psychosis-proneness, such as liability to psychotic (hypo-) mania (Claridge et al., 1996; Claridge & Blakey, 2009). Hence, from the theoretical point of view, this inclusion of ImpNon corresponds to the above-mentioned definition of schizotypy as representing psychosis of all severity levels (Claridge, 1997; Mason & Claridge, 2006; Kwapil & Barrantes-Vidal, 2015). Irrespectively of the issues of schizotypy factor structure, the 3 core schizotypic dimensions remain close from schizophrenia symptoms and likely index attenuated versions of symptoms, and reflect genetic associations.

Empirical data further confirm the ability of personality traits measured by O-LIFE and sO-LIFE to account for clinical symptoms, in various populations. Cochrane, Petch and Pickering (2010) comparatively assessed healthy controls and schizophrenia patients with the O-LIFE and the Scale for Assessment of Negative Symptoms (SANS; Andreasen, 1983) and the Scale for Assessment of Positive Symptoms (SAPS; Andreasen, 1984). O-LIFE's UnEx scale corresponded

to SAPS scale score, whereas CogDis and IntAn did not correspond to SANS. Lin et al. (2013), however, showed correspondence between SANS score and sO-LIFE's IntAn in an ultra-high risk sample. Also, higher IntAn associated with lower functioning and quality of life, and indicated need for care, whereas higher CogDis associated with higher depression and anxiety, and higher UnEx higher positive symptoms only (Lin et al., 2013). Cella et al. (2013) showed that adolescents with high scores in all sO-LIFE dimensions featured high distress, and family history of psychosis, suggesting a genetic component.

Schizotypic traits reflect genetic liability to psychosis, because they are heritable, they aggregate in families with SSD individuals, and represent schizophrenia or psychosis risk. For instance, Kendler (1985) showed that schizophrenia relatives' personality traits could be described by schizotypal PD. Also, Siever et al. (1990) showed that relatives of individuals diagnosed with schizotypal and/or paranoid PDs showed an increased risk for schizophrenia-related disorders. A recent twin study using O-LIFE questionnaire showed that schizotypic traits are heritable (Mason & Claridge, 2006). Tarbox and Pogue-Geile (2011) evaluated that 50% of schizotypic traits variance was genetic. Grant et al. (2014) suggested that O-LIFE Short scores are an endophenotype of schizophrenia, because of their associations with dopamine-related polymorphisms (e.g. Val/Met), and genes (e.g. COMT, MAOA; see also Grant, 2015).

1.3.1.1.2 DIMENSIONAL APPROACHES OF ASP

Likewise, for ASp, dimensional approaches were hinted from the very first descriptions of infantile autism and what would become Asperger's syndrome. Kanner (1973; cited by Frith, 1991) evoked the possibility that infantile autism feature different grades of severity, as observed for other disorders. Also, Ousley and Cermak (2014) considered that Asperger ([1944] 1991) assumed the syndrome he described was dimensional, because of the attenuated autistic symptoms he observed in his patients' relatives. These observations of Kanner and Asperger corresponds to the two meanings of ASD distinguished by Lai, Lombardo, Chakrabarti and Baron-Cohen (2013): one meaning of "spectrum" refers to a continuity *within the clinical domain*, and the other one to a continuity *extending from clinical domain towards healthy personality one*. This second meaning corresponds to what we termed ASp.

Inside the clinical domain, the notion of "autism continuum" (Wing & Gould, 1979), to become "autism spectrum disorders" (ASD; coined by Allen, 1988), formalized the idea of different degree of severity *within autism spectrum*, that is, between autistic-like disorders sharing a common triad of deficits (but variable in its expression). For instance, infantile autism and Asperger's

syndrome may be seen as differing in severity, although both belonged to *DSM-IV-TR*'s "pervasive development disorders". Indeed, the former's diagnostic criteria feature a language acquisition delay[10], whereas the latter did not (APA, 2000). Nowadays, infantile autism and Asperger's syndrome are considered to be highly related (Sanders, 2009), though distinguished by specifiers (APA, 2013). Current ASD diagnosis is closer to Asperger's syndrome, since language delay is no more an inclusion criteria (APA, 2000), but a specifier (APA, 2013).

Beyond the clinical domain, ASp refers to attenuated clinical autistic symptoms found in healthy autistic personality (Lai et al., 2013). Attwood (2007), and Hippler and Klipcera (2003) noted that Asperger himself considered the existence of an "autistic PD" that was part of a normal range of abilities, extending toward normality. Asperger's reference to personality stresses the early intuition of (i) an association between autistic personality and disorder, and (ii) the relevance of autistic personality (i.e. adaptive features) to understand a disorder, sometimes accompanied by exceptional achievements (Asperger, [1944] 1991). As for schizotypy, research extended the ASD into healthy population, in the form of personality traits corresponding to attenuated version of ASD symptoms (Baron-Cohen et al., 2001; Constantino & Todd, 2003; also in the BAP: Piven et al., 1997).

Several self-report questionnaires[11] exist, that claim to measure autistic traits corresponding to ASp clinical symptoms. The best studied and the most well-known is the Autism Spectrum Quotient (AQ; Baron-Cohen et al., 2001). Other questionnaires were developed, notably for use in more specific populations (e.g. BAP): the Autism Screening Questionnaire (ASQ; Berument, Rutter, Lord, Pickles, & Bailey, 1999), the Autism Spectrum Disorder in Adults Screening Questionnaire (ASDASQ; Nylander & Gillberg, 2001), the Broader Autism Phenotype Questionnaire (BAPQ; Hurley, Losh, Parlier, Reznick, & Piven, 2007), the Broad Autism Symptoms Scale (BPASS; Dawson et al., 2007), the Subthreshold Autistic Traits Questionnaire (SATQ; Kanne, Wang, & Christ, 2012), and the Social Response Scale (SRS; Constantino, Davis et al., 2003). But do these instruments measure autistic traits the same way?

10 Interestingly, the recent *DSM-5* (APA, 2013) suppressed the distinctions between Asperger's syndrome and infantile autism, and favoured the term ASD over PDD. Hence, language delay is no more necessary for diagnosing an ASD and became an optional feature (a specifier). Yet, ASD core symptoms still consist in social/communication, which keeps the emphasis on Mentalism/Empathizing relevant with regard to *DSM-5*.

11 Obviously, responding to self-report questionnaires is suitable for adult respondents with a normal IQ, be it in the clinical (e.g. high-functioning autistic or aspergers) or in the healthy domain (e.g. participant at least with a normal IQ). Self-report questionnaire are inadequate for lower ends of ASp featuring social communication deficits.

Unlike schizotypic traits, analysis of autistic traits' instruments shows no clear consensus as to the number, identity, and structure of autistic traits' dimensions. For instance, the BAPQ identified 3 dimensions (Rigid, Aloof, Pragmatic Language deficits; Hurley et al. 2007), the BPASS 4 dimensions (Social Motivation, Expressiveness, Conversational skills, Flexibility/Range of Interests). The SATQ, merging AQ and BAPQ, identified 5 dimensions (Social Interaction & Enjoyment, Oddness, Reading Facial Expression, Expressive Language, and Rigidity). The AQ originally was created to measure 5 dimensions (Attention to Detail [AttDet], Attention Switching deficits [AttSwi], Communication deficits [Comm], Imagination deficits [Ima], Social Skills deficits [SocSki]; Baron-Cohen et al., 2001). More than others, this instrument underwent translations in several languages (e.g. Italian: Ruta et al., 2013; Dutch: Hoekstra, Bartels, Cath, & Boomsma, 2008), constituting many occasions to verify and question its factor structure and psychometric properties.

Most independent studies on AQ factor structure did not support the original AQ factor structure and proposed alternative ones. While the AQ received most attention in terms of validation studies, it also received criticism regarding the reliability of its scales, and its factor structure (e.g. Hurst, Mitchell et al., 2007). Here, we focus on the factor structure of AQ. Instead of confirming this original factor structure, the majority of studies proposed alternative factor structures. For instance, Austin (2005) and Hurst, Mitchell et al. (2007) supported a 3-dimensions model (Social Skills deficits, Communication/Mindreading, and Details/Patterns), resembling the one of BAPQ and the triad of autistic symptoms of *DSM-IV* (APA, 1994). Stewart and Austin (2009) supported a 4-dimension model (Socialness, Patterns, Understanding Others/Communication deficits, Imagination deficits). Later studies supported factor structures with more dimensions, such as the model by Hoekstra et al., (2011) featuring 5-dimensions on 2-levels model (Social Behaviour: Social Skills deficits, Routine, Switching deficits, Imagination deficits; Numbers/Patterns), or the one by Kloosterman, Keefer, Kelley, Summerfeldt and Parker (2011) 5-dimensions model (Social Skills deficits, Communication/Mindreading deficits, Attention to Details, Imagination deficits, Routines/Repetitive Behaviours). Yet, in most of these studies, modern psychometric tools were applied and provide some certainties about AQ factor structure. We further discuss issues about AQ factor structure and psychometric properties later. Disregarding the issues of AQ traits factor structure, several studies supported autistic traits ability to account for attenuated versions of autistic symptoms, and reflect genetic associations.

Like schizotypic traits, autistic traits reflect genetic liability to ASD, because they are heritable, they aggregate in families with ASD individuals and represent ASD risk. Unaffected relatives of individuals with ASD show more autistic traits as compared to control groups (Bishop

et al., 2004; Bolton, Pickles, Murphy, & Rutter, 1998; Piven et al., 1997) or individuals of the general population (Baron-Cohen et al., 2001; Pisula et al., 2013). Using the SRS in twin studies, Constantino and Todd (2003, 2005) showed that autistic traits were heritable. Parents with children with ASD show increased scores at AQ as compared to parents without ASD child (Bishop et al., 2004; Kose, Bora, Erermiş, Özbaran, Bildik, & Aydın, 2013; Ruta et al., 2011; Wheelwright et al., 2010). Interestingly, parents of ASD children scored higher most of the time in Comm, SocSki, and sometimes Ima, whereas AttDet, or AttSwi were not discriminant (Chapter 6.3.1.4.). In the general population, autistic traits as measured by AQ are also heritable (Hoekstra, Bartels, Verweij, & Boomsma, 2007). As for schizotypy, autistic traits seem to be able to capture ASD liability, hence representing the ASp (Lundström et al., 2012; Murray, Booth, Kuenssberg, & O'Donnell, 2014). Yet, is it the case in all populations, in particular in different languages?

1.3.2 VALIDATION OF PERSONALITY TRAITS QUESTIONNAIRES

A prerequisite for using personality traits is their reliability and validity in a given cultural and linguistic environment. As part of a validation study, psychometric features such as factor structure and reliability of a questionnaire have to be examined, in particular after a translation from a language to another one (e.g. Chinese: Lau, Gau et al., 2013). Obviously, acceptable reliability and validity are of paramount importance when the recruitment of an imaging or behavioural study relies on personality traits dimension scores (e.g. Cappe et al., 2012). Personality questionnaires by definition include noise in their measure. The maximum has to be done to reduce this part of variance originating from bad translation, biased items (e.g. relatively to socio-demographic variables such as gender roles), unreliable or useless items, and unsuitable factor structures (e.g. Winterstein, Ackerman, Silvia, & Kwapil, 2011). Technically, a validation study does not only include reliability (or internal consistency) measured by Cronbach's alphas, and descriptive norms of questionnaire scores. A validation study should also include factor structure assessment, with methods such as Factor Analyses (FAs), Principal Component Analyses (PCAs), and Confirmatory Factor Analyses (CFAs; Brown, 2006). Since validation studies of French version of sO-LIFE and AQ questionnaires were not performed (i.e. not with CFAs or factor/principal component analyses; for AQ see Sonié et al., 2011, 2013), the very first part of this thesis consisted in validating these measures. Also, correlational analyses between an instrument dimensions and other instruments' dimension measuring analogue or associated constructs allow further assessment of validity (e.g. Asai, Sugimori, Bando, & Tanno, 2011; Ingersoll, Hopwood, Wainer, & Donnellan, 2011). We also used several instruments in order to offer further evidence in support of sO-LIFE and AQ validity

(Chapters 2 and 3). In what follows, we will briefly summarize the properties of sO-LIFE and AQ questionnaires reported by validation studies, to clarify our expectations for their respective French validations.

1.3.2.1 THE SO-LIFE QUESTIONNAIRE

The factor structure of a schizotypic traits questionnaire like sO-LIFE is expected to consist of 3 to 4 dimensions. The sO-LIFE questionnaire (Mason et al., 2005) is the shortened version (43 items) of the original O-LIFE questionnaire (104 items; Mason et al., 1995). The sO-LIFE consists of 4 dimensions assessing positive schizotypy (Unusual Experiences), negative schizotypy (Introvertive Anhedonia), cognitive disorganization (Cognitive Disorganization), and impulsive/reckless and anti-conformist behaviours (Impulsive Nonconformity). The first three dimensions closely match those of another schizotypy instrument, the SPQ (Raine, 1991), as demonstrated by Asai et al. (2011). Contrary to the WSS, both SPQ and sO-LIFE include a disorganized dimension. Yet, contrary to the SPQ, the sO-LIFE stems from the fully-dimensional tradition, which considers psychosis liability, and not only schizophrenia liability. For this reason, it includes an Impulsive Nonconformity dimension accounting for psychosis-related traits associated with schizotypy, i.e. impulsive/antisocial borderline or (hypo-) manic features (Claridge, 1997; Mason & Claridge, 2006). Also, the sO-LIFE does not feature any paranoia dimension, whereas paranoia was identified in some validation studies of the SPQ (Stefanis, Smyrnis, Avramopoulos, & Stefanis, 2004). Despite its originality as compared to other schizotypy scales, several studies replicated most of the (s)O-LIFE factor structure.

Although studies supported the 4-factor structure of the original O-LIFE, recent studies on sO-LIFE hesitated between 4- and 3-factor structures. Both exploratory analyses (FAs; Claridge et al., 1996), and CFAs (Mason, 1995) validated the original O-LIFE 4-dimension factor structure. Correspondingly for sO-LIFE, CFAs supported a 4-factor solution in Italian (Cella et al., 2013). Yet, in English, Lin et al. (2013) supported a 3-factor model: ImpNon was discarded on the ground of nonsignificant loadings. Hence, studies are split between the original 4-factor structure and an alternative 3-factor structure, excluding ImpNon, and getting closer to SPQ one. Therefore, our French validation study should expect comparable means and properties than those of English version (Mason et al., 2005), as well as a similar dilemma between 4- and 3-factor structures (Cella et al., 2013; Lin et al., 2013; see Chapter 2).

1.3.2.2 THE AQ QUESTIONNAIRE

Autistic traits as measured by the AQ did not give rise to a consensual factor structure, although the rejection of original factor structure is quasi-systematic. Originally, the AQ was devised as a 50-item 5-dimension instrument (Baron-Cohen et al., 2001). However, several independent studies criticized the low reliability, and the factor structure of the AQ, and proposed alternative factor structures (Austin, 2005; Hurst, Mitchell et al., 2007; Lau, Gau et al., 2013; Lau, Kelly et al., 2013; Stewart & Austin, 2009). Among studies challenging AQ factor structure, opinions are globally split between those expecting factor structure with 3-, 4- and 5-dimensions. The first ones' factor structures mirror dimensions close from clinical diagnostic criteria (e.g. Austin, 2005; Hurst, Mitchell et al., 2007; Russell-Smith, Mayberry, & Bayliss, 2011), in line with other instruments (BAPQ: Hurley et al., 2007). The second ones supported 4-dimension structures on the ground of PCAs (Stewart & Austin, 2009; Russell-Smith et al., 2011). Finally, several recent studies chose 5-dimension models, backed by results of parallel analyses and CFAs (e.g. Hoekstra et al., 2008; Kanne et al., 2012; Lau, Gau et al., 2013; Lau, Kelly al., 2013).

To our knowledge, studies on AQ factor structure using CFAs never reported acceptable fit indices for the original AQ factor structure (Baron-Cohen et al., 2001), nor 3-dimension models, but favoured an alternative 5-dimension models (Lau, Kelly et al., 2013). One could have believed that 3-dimension factor structures models would have fared better than others, on the ground of the *triad* of autistic symptoms (APA, 2000). Yet, most recent studies' CFAs considered a minimum of 5 dimensions, notably after parallel analyses (e.g. Kloosterman et al., 2011). Also, factor structures with 2, 3 or 4 dimensions fared more poorly than their 5-dimension ones (e.g. Lau, Gau et al., 2013). So far, only two studies reported AQ models with acceptable factor structures, relatively to current fit criteria (Brown, 2006). These alternative factor structures were obtained through shortening and reorganising items in different dimensions (Chinese/Taiwanese AQ: Lau, Gau et al., 2013; original English AQ: Lau, Kelly et al., 2013; see Chapter 3).

Although AQ validation studies using CFAs were performed for several linguistic versions, no published paper reported either PCA or CFAs with the French version of AQ. Indeed, the French version of AQ was published and norms were described (Lepage, Lortie, Taschereau-Dumouchel, & Théoret, 2009; Sonié et al., 2011, 2013). Sonié et al. (2013) showed AQ scores were sensitive to Asperger's syndrome diagnosis, reported adequate reliability, and established a cut-off score. Yet, to our knowledge, the French AQ factor structure was neither validated with PCAs/FAs nor with CFAs. Instead, authors using the French AQ assumed the original 5-dimension 50-item factor structure would suit a French-speaking population. Since numerous AQ validation studies

challenged AQ original factor structure and proposed alternative ones, we should expect that the validation of French AQ version makes no exception.

Based on the aforementioned AQ validation studies, we expected the French version of the AQ to feature a 5-factor structure, with more or less probability to contain the following dimensions: (i) *certainly* a dimension analogue to SocSki, yet reoriented towards social anhedonia rather than lack of social skills (e.g. Austin, 2005; Hoekstra et al., 2008, 2011; Hurst, Mitchell et al., 2007; Kloosterman et al., 2011; Lau, Gau et al., 2013; Lau, Kelly et al., 2013; Russell-Smith et al., 2011; Stewart & Austin, 2009), (ii) *certainly* a dimension analogue to AttDet (e.g. Austin, 2005; Hoekstra et al., 2008, 2011; Hurst, Mitchell et al., 2007; Kloosterman et al., 2011; Russell-Smith et al., 2011; Stewart & Austin, 2009), (iii) *probably* a revised and redefined dimension analogue to Comm, accounting for mentalizing deficits or Mindreading (Hurst, Mitchell et al., 2007; Kloosterman et al., 2011; Lau, Gau et al., 2013; Lau, Kelly et al., 2013; Russell-Smith et al., 2011; Stewart & Austin, 2009), (iv) *possibly* an independent Imagination dimension (e.g. Hoekstra et al., 2008, 2011; Kloosterman et al., 2011; Russell-Smith et al., 2011; Stewart & Austin, 2009), and (v) *possibly,* instead of keeping AttSwi dimension (but see: Hoekstra et al., 2008; Lau, Gau et al., 2013), a revised and redefined dimension accounting for Routines/Repetitive Behaviours or Resistance to Change (Hoekstra et al., 2011; Kloosterman et al., 2011; Lau, Kelly et al., 2013).

1.4 AUTISTIC AND SCHIZOTYPIC TRAITS RELATIONSHIPS

1.4.1 RELATIONSHIPS OF AUTISTIC AND SCHIZOTYPIC TRAITS WITH BROADER PERSONALITY TRAITS

Correlational analyses provide an indirect way to validate the French version of sO-LIFE and AQ, replicating known relationships with other instruments measuring similar or close constructs. In particular, we aimed at better characterizing and replicating the relationships of autistic and schizotypic traits with regard to a broader personality model. Indeed, even if autistic and schizotypic traits may appear peculiar and alien to “normal” personality, they indeed can be explained, at least partly, by general personality models. Concretely, we used the French SPQ-B (Ortuño-Sierra et al., 2013; Raine & Benishay, 1995) as a specific instrument to validate sO-LIFE. To validate both sO-LIFE and AQ, we also used the Eysenck Personality Questionnaire Revised and Abridged (EPQR-A), a broader measure of personality (Bouvard, Aulard-Jaccod, Pessonneaux, Hautekeete, & Rogé, 2010; Francis, Brown, & Philipchalk, 1992). The SPQ-B features three dimensions akin to schizophrenia symptoms and the 3 core schizotypic dimensions of sO-LIFE: Cognitive-Perceptive traits (Cog-Per), Interpersonal deficits (Int), and Disorganization (Dis).

Relationships between these constructs and AQ and sO-LIFE ones can be hypothesized from previous literature. EPQR-A measures 4 dimensions: Neuroticism (i.e. negative affects and cognitions), Extraversion (i.e. sociability, enjoyment of social situations), Psychoticism (i.e. lack of consideration for others, social norms, impulsivity), plus a Lie scale (i.e. social desirability or liability to lie).

Previous literature suggests some precise relationships of EPQR-A with sO-LIFE and AQ, as well as between sO-LIFE and SPQ-B. Based on previous studies (Burch, Hemsley, Pavelis, & Corr, 2006; Claridge et al., 1996; Ross, Lutz, & Bailley, 2002), we would expect the following relationships between EPQR-A and sO-LIFE dimension scores (see also Chapter 3): (i) positive correlations between Neuroticism and each sO-LIFE dimension, strongest with disorganized one (CogDis), (ii) negative correlations between Extraversion and negative (IntAn) and disorganized schizotypy (CogDis), and, (iii) positive correlation between Psychoticism and ImpNon. Based on a previous study (Asai et al., 2011), we would expect the following relationships between corresponding SPQ-B and sO-LIFE dimension scores: (i) positive correlations between positive dimensions (respectively UnEx and Cog-Per), (ii) positive correlations between negative dimensions (IntAn and Int), and, (iii) positive correlations between disorganized dimensions (CogDis and Dis). Based on previous studies (Austin, 2005; Wakabayashi, Baron-Cohen, & Wheelwright, 2006a), we expect the following relationships between EPQR-A and AQ dimension scores: (i) positive correlations between Neuroticism and some AQ subscales, such as Communication and Attention Switching deficits dimensions, and, (ii) negative correlations between Extraversion and AQ social deficits dimensions (e.g. SocSki, Comm, Ima).

1.4.2 RELATIONSHIPS BETWEEN ASP AND PSP IN CLINICAL AND HEALTHY POPULATIONS

The validation of autistic and schizotypic traits questionnaires' French versions (AQ, sO-LIFE) allows us to address the first important question of this work: "what are the relationships between autistic and schizotypic personality traits ?" Of course, data exist about the relationships between ASp and PSp, either at the symptoms or at the personality traits level. Studies can roughly be divided into three types: (i) studies comparing *symptoms in patients* diagnosed with ASD and SSD (e.g. Lugnegård et al., 2011, 2012), (ii) studies comparing *personality traits in patients* with ASD and SSD (Konstantareas & Hewitt, 2001), and (iii) studies comparing autistic and schizotypic *personality traits in non-clinical* (putatively) healthy population (e.g. Hurst, Nelson-Gray et al., 2007). Since we are interested in the healthy personality part of ASp and PSp, we will mostly be

concerned by the third type of studies, i.e. those between autistic and schizotypic traits in healthy controls. Nevertheless, we will briefly review these three types of studies, before emphasizing the third one. We want to underline the continuity between ASp-PSp relationships across severity levels, irrespectively of instruments (i.e. clinical diagnoses, self-reports).

1.4.2.1 CO-OCCURRENCE OF ASD AND SSD SYMPTOMS IN CLINICAL POPULATIONS

Numerous studies investigated ASD and SSD comorbidity, and recent ones not only show an overlap in negative symptoms (Konstantareas & Hewitt, 2001), but also another one in positive symptoms. Interestingly, these associations pertain to schizophrenia, but also to bipolar disorder, and PDs. Below, we briefly summarize some studies.

SSD diagnoses/symptoms in ASD populations: Some studies found SSD diagnoses or symptoms in ASD populations. Lugnegård et al. (2011) investigated the psychiatric comorbidity in young adults with Asperger's syndrome. In a second paper, Lugnegård et al. (2012) showed that around half of individuals with Asperger's syndrome in their sample met criteria for at least one PD. Most met the criteria for schizoid PD, few for schizotypal PD, but also for avoidant and obsessive-compulsive PD. More intriguing, 13% of individuals with Asperger's syndrome reported hallucinations. In ASD individuals, Hofvander et al. (2009) reported life-time prevalence of comorbid psychotic disorders (12%), as well as comorbidity with, in particular, schizoid, paranoid and schizotypal PDs (see also: Attwood, 2007 pp. 341–343, for a discussion; Billstedt, Gillberg, & Gillberg, 2005; Stahlberg, Soderstrom, Rastam, & Gillberg, 2004).

ASD diagnoses/symptoms in SSD populations: Other research found ASD diagnoses or symptoms in SSD, be it with current or retrospective assessment. With respect to childhood symptoms, Esterberg, Trotman, Brasfield, Compton and Walker (2008) investigated the links between childhood and current autistic features in adolescents with schizotypal PD. Although neither childhood nor current autistic features predicted conversion to schizotypal PD, adolescents with schizotypal PD showed more severe past and current social impairment, unusual interests and behaviours as compared to control groups (i.e. normal and other PDs). Hallerbäck, Lugnegård and Gillberg (2012) found that half of their cases with SSD met criteria for ASD according to retrospective parental reports. In a larger study, Davidson et al. (2014) showed that prevalence of Asperger's syndrome in a population of first-episode psychosis was considerably higher than in the general population.

Hence, evidence suggests that ASD and SSD symptoms overlap using clinical assessment tools (for a review: Chisholm et al., 2015). Yet, importantly for us, personality questionnaires assessment in clinical populations showed comparable overlaps, as presented below.

1.4.2.2 COMPARISON BETWEEN AUTISTIC AND SCHIZOTYPIC TRAITS IN CLINICAL POPULATIONS

Several studies assumed sensitivity of personality traits to account for corresponding symptoms in clinical populations, and reported traits differences between ASD and SSD patients. Below we detail results for studies measuring jointly autistic and schizotypic traits in ASD and SSD patients, studies examining schizotypic traits in ASD populations, and those examining autistic traits in SSD populations.

Autistic and schizotypic traits in ASD and SSD: Konstantareas and Hewitt (2001) reported an overlap in negative traits between ASD and SSD patients, using autistic and schizotypic traits questionnaires (AQ, SPQ), and conjectured on the relatedness of the disorders. Spek and Wouters (2010) showed that individuals with schizophrenia were more likely to report positive traits at SPQ, whereas individuals with high-functioning autism were more likely to report impairments in AQ's SocSki and Comm. However, both groups overlapped with regard to negative traits (Int), disorganization (Dis), AttDet, and Ima deficits. In a second paper, Wouters and Spek (2011) added that the high-functioning autism group reported larger scores in AQ's SocSki, Comm and AttSwi dimensions, as compared to the schizophrenia group. Despite shared social deficits traits, high-functioning autism groups showed larger social deficits than schizophrenia one.

Schizotypic traits in ASD: Using the SPQ, Barneveld et al. (2011) showed that adolescent with a childhood diagnosis of ASD not only showed higher negative traits but also higher positive and disorganized ones, suggesting ASD are at higher risk for developing psychosis.

Autistic traits in SSD: Jones, Thapar, Lewis and Zammit (2012) showed that psychotic experiences in adolescence associated with childhood autistic traits, in particular speech problems, odd rituals, and unusual habits.

Further than merely replicating clinical assessments' findings, personality traits' questionnaires clarify that mostly negative, disorganized, and to a certain extent positive traits, overlap between ASD and SSD. In healthy populations, analogue personality traits questionnaires should show a similar overlap pattern.

1.4.2.3 COMPARISON BETWEEN AUTISTIC AND SCHIZOTYPIC TRAITS IN HEALTHY, NON-CLINICAL POPULATIONS

Although studies on healthy participants also emphasized the existence of shared or overlapping personality traits between autistic and schizotypic ones, recent evidence suggest the existence of opposite traits. Since we provided validation data of AQ and sO-LIFE, we will focus on results previously obtained with these instruments (e.g. Russell-Smith et al., 2011), and comparable ones (e.g. SPQ, Hurst, Mitchell et al., 2007). Across studies, two main sets of overlapping or shared traits, and recently one set of diametrical traits emerge: (i) shared or overlapping negative schizotypy, negative and disorganization traits, and autistic social skills and communication deficits traits, (ii) shared or overlapping positive schizotypy, and autistic attention to details traits, and, (iii) positive schizotypic traits diametrically opposite to some autistic traits. Below, we will briefly review evidence supporting the shared (i, ii) and the (iii) diametrically opposite personality trait sets.

1.4.2.3.1 SHARED PERSONALITY TRAITS BETWEEN AUTISTIC AND SCHIZOTYPIC SETS

Using either SPQ or O-LIFE together with AQ, several studies showed large overlap between negative/disorganized traits dimensions. Most studies showed moderate to large positive correlations between negative schizotypic dimension (e.g. SPQ: Int; sO-LIFE: IntAn) and autistic social deficits dimensions, mostly SocSki and Comm deficits dimensions (Hurst, Nelson-Gray et al., 2007; Claridge & McDonald, 2009; Russell-Smith et al., 2011; see also Ford & Crewther, 2014). Moreover, autistic Communication and Imagination deficits also showed small to large positive correlations with schizotypy negative dimensions (Ford & Crewther, 2014; Hurst, Nelson-Gray et al., 2007; Russell-Smith et al., 2011). Disorganized schizotypic dimensions (e.g. SPQ: Dis; SPQ: Odd Behaviour; SPQ: Odd Speech) showed small to large positive correlations with autistic Comm and SocSki deficits dimension (AQ: Comm; Hurst, Nelson-Gray, 2007; Ford & Crewther, 2014). Using sO-LIFE, cognitive disorganization (sO-LIFE: CogDis) showed significant small to medium correlations with autistic communication deficits analogues, but equally with autistic social skills deficits. Using PCAs and FAs on AQ and SPQ dimensions, two studies extracted a component with strong loadings of negative and disorganized schizotypic traits (SPQ: Int, SPQ: Dis) together with autistic social deficits (AQ: SocSki, AQ: Comm; Dinsdale et al., 2013; Ford & Crewther, 2014). Hence, the first overlap in negative and disorganized traits has strong support in healthy populations, in line with results in clinical populations, and irrespectively of instruments used (SPQ or sO-LIFE). The same goes for positive traits.

As for the overlap in positive traits, the same studies in healthy populations showed a weaker but robust overlap between positive schizotypy and mainly autistic attention to details (AttDet). Schizotypy positive dimensions (UnEx, Pos) showed positive associations with autistic attention to details (AQ: AttDet) or analogues. For instance, SPQ positive dimensions showed small to large correlations with autistic attention to details (Hurst, Nelson-Gray, 2007; Ford & Crewther, 2014). Similarly, with sO-LIFE, positive schizotypy (sO-LIFE: UnEx) and impulsive non-conformity (sO-LIFE: ImpNon) showed significant small to medium positive correlations with autistic attention to details (AQ: AttDet; Russell-Smith et al., 2011). In PCAs by Dinsdale et al. (2013), positive schizotypic dimension and autistic attention to details loaded congruently on the same component (PC2) different from the first one (PC1) grouping negative and social deficit traits. To put it shortly, the second overlap in positive traits (i.e. positive schizotypy and autistic attention to detail) also has a strong support in almost all studies (but see Ford & Crewther, 2014), in line with results in clinical populations (Chapters 1.4.2.1. and 1.4.2.2.), and irrespectively of questionnaire used. Yet, positive schizotypy (UnEx) also opposes to another set of autistic traits.

1.4.2.3.2 DIAMETRICAL PERSONALITY TRAITS BETWEEN AUTISTIC AND SCHIZOTYPIC SETS

Despite emphasis on overlapping or shared features between autistic and schizotypic traits, diametrical traits exist. Indeed, in the results of Hurst, Nelson-Gray et al. (2007), several autistic social deficits dimensions (AQ: Comm, AQ: SocSki, AQ: Ima) were significantly *negatively* associated with positive schizotypy (SPQ: Cog-Per). Although the amounts of shared variance were small, they were significant. Interestingly, Russell-Smith et al. (2011) report similar relationships, but using the sO-LIFE: autistic Imagination deficits (AQ: Ima) show small but significant negative correlations with positive schizotypy (sO-LIFE: UnEx) and Psychoticism-analogue Impulsive Non-conformity (sO-LIFE: ImpNon). Using PCAs/FAs, Dinsdale et al. (2013) identified potentially diametrical or specific traits, between schizotypy and autistic domain. Aside from a negative/disorganized component (PC1), Dinsdale et al. extracted a second component (PC2), in which, mostly, positive schizotypy loading oppositely to several *undefined* autistic social deficits traits (i.e. SocSki, Ima). These authors could replicate twice the aforementioned opposition, but their interpretation of this dimension and the autistic traits involved remained elusive.

Finally, an investigation of autistic and schizotypic traits relationships in a French-speaking population should uncover both shared/overlapping sets of traits, and diametrical/opposite ones. First, we should identify an overlap consisting in a shared set of traits between autistic and

schizotypy domain. This would group, mostly, negative, but also disorganized schizotypy, and autistic social anhedonic dimensions (mostly SocSki). Second, we should identify a dimension featuring diametrical traits between autistic and schizotypy domains, as in Dinsdale et al. (2013). To do so, we used correlations, and PCAs, to extract 2 dimensions, for the sake of replicability and parsimony (Dinsdale et al., 2013). We will put emphasis on interpreting the eventual diametrical traits in light of Mentalism/Empathizing theories (Brosnan et al., 2010; Crespi & Badcock, 2008). Corresponding to these psychometric relationships between autistic and schizotypic traits, we will hypothesize relationships of shared and, eventually, diametrical sets of traits with behavioural correlates of social cognition or Mentalism (see Chapter 3).

1.5 BEHAVIOURAL CORRELATES OF AUTISTIC AND SCHIZOTYPIC TRAITS

So far, we discuss self-reported experiences, feelings, and cognitions associated to autistic and schizotypic traits, notably related to social deficits. Yet, do they correspond to similar, or different, and even possibly opposite, ways of processing social information? If autistic and schizotypic personality traits are expressions of an underlying genetic liability to illness (e.g. Grant et al., 2014; Lundström et al., 2012), autistic and schizotypic personality traits may be related to attenuated behavioural features resembling those of ASD and SSD (Ettinger et al., 2015; Snitz et al., 2006; Sucksmith et al., 2011). Indeed, cognitive theories were proposed to account for symptoms and traits in either ASp or PSp (e.g. Hill & Frith, 2003; O'Flynn, Gruzelier, Bergman, & Siever, 2003). Many different behavioural markers exist for either ASD or SSD, some of which meet criteria for endophenotypes (Gottesman & Gould, 2003). We present some examples from ASD and SSD research, in turn, and evaluate suitable candidates for ASp and PSp.

1.5.1 BEHAVIOURAL CORRELATES OF ASD

In ASD research, we can find roughly three main categories of behavioural tasks, related to three major cognitive theories explaining symptoms (Hill & Frith, 2003). First, there would be experiments targeting mindblindness (Baron-Cohen, 2002; Baron-Cohen, Leslie, & Frith 1985), or empathizing/mentalizing deficits (Happé et al., 1996). Although the first hypotheses proposed an absent ToM in ASD, Hill and Frith (2004) proposed that the *intuitive* understanding that people have mental states is impaired, whereas the acquisition of a *conscious* ToM is possible. Yet, Hill and Frith observed that the mentalizing theory accounts less well for face recognition impairment, more generally "social perception". On the contrary, the difficulties with face recognition may play

a role in explaining developmentally the ToM deficits. Also, ASD may be characterized by absent innate preferences for attending to social stimuli, related to lack of reward value or social motivation (Dawson, Webb, & McPartland, 2005; Klin, Jones, Schultz, Volkmar, & Cohen, 2002), and/or active or passive gaze avoidance (Senju & Johnson, 2009; Tanaka & Sung, 2013). Yet, ASD does not consist only in social deficits, but also routines/repetitive behaviour, insistence on sameness and restricted interests. Hill and Frith (2004) present two cognitive theories attempting to account for these deficits: i.e. weak central coherence, and executive functioning deficits.

Second, the "weak central coherence" theory attempted to account for the local rather than global bias of individuals with ASD. "Central coherence" refers to an information processing style characterized by integration of information into a higher-level meaning. These pieces of information can be verbal or nonverbal. Notorious examples feature block design IQ subtest (Wechsler, 1981), the embedded figure task (Witkin, Oltman, Raskin, & Karp, 1971), or visual search tasks (Mottron, Dawson, Soulières, Hubert, & Burack, 2006). For instance, the Embedded Figure Task (EFT) consists in localizing a visual shape embedded in another larger, more complex, visual shape. Individuals with ASD showed advantage in EFT as compared to controls, likely due to a weak central coherence or a local bias (Happé & Frith, 2006). In this task, a style with a "strong coherence" may be disadvantaged as compared with the autistic style characterized by weak central coherence. Indeed, the former process information as a function of the context, at the expense of attention to and memory for details, whereas the latter works at the expense of contextual meaning and in favour of piecemeal processing (Hill & Frith, 2003). Authors hypothesized that this would be caused by a dysconnectivity between brain regions subserving more basic perceptual processes and those subserving top-down modulation of these processes. Although weak central coherence initially referred to an *inability to integrate contextual information*, it is now viewed as a style or *preference* for details, a local processing *bias* (Happé & Frith, 2006). Another theory extended weak central coherence theory to explain autistic "enhanced perceptual functioning", disregarding previous claims of information integration deficits (Mottron et al., 2006).

A third group of tasks pertains to executive dysfunctions. Executive function refers to various functions subserved by frontal lobes: e.g. planning, working memory, initiation and monitoring of action, inhibition (Hill & Frith, 2003). The executive dysfunctions theory in ASD attempts to explain autistic rigidity and perseverations on the model of executive functions failures seen after frontal lobe lesions (Hill & Frith, 2003). For instance, deficits in planning were observed in the Tower of Hanoi or Tower of London tasks (Shallice, 1982). Attention set shifting deficits were assessed with Trail Making Task (Reitan, 1958). Also, perseveration was investigated as lack of cognitive flexibility at Wisconsin Card Sorting task (WCST; Berg, 1948). This task consisted in

classification of cards according to abstract rules (e.g. colour, shape or number). Yet, the participant is not instructed about the classification rule and has to deduce it using feedback information (i.e. correct/incorrect). Also, the rule changes unpredictably several times throughout the experiment, and the participant has to re-deduce it and keep it. These difficulties challenge the participant "cognitive flexibility", i.e. ability to recognize and update a behaviour quickly, which oppose to perseveration (see Hill, 2004 for a review).

Although well studied in autistic individuals, these deficits are problematic for several reasons. Deficits in these tasks may not provide much information about ASp aetiology, mechanisms and genetics for several reasons: (i) the replication of these deficits within ASp is sometimes difficult, (ii) certain of these deficits are not specific to ASp and can be found associated to other neurological or psychiatric conditions, (iii) social cognition may explain more directly straightforwardly autistic social deficit symptoms than executive dysfunctions and weak central coherence, and (iv) certain of these deficits did not appear in first degree relatives of individuals with ASp (i.e. BAP), making them unlikely endophenotypes candidates (Sucksmith et al., 2011).

Sucksmith et al. (2011) reviewed endophenotypes of ASp, and concluded that the best *cognitive* endophenotype candidates pertained to deficits in social cognition rather than executive dysfunctions, weak central coherence or any other. To do so, Sucksmith et al. (2011) investigated the cognitive deficits in BAP populations, i.e. relatives of individuals with ASD, that is the healthy end of ASp. In either children or older relatives of individuals with ASD, the best cognitive traits of the BAP pertained to social cognition. In BAP children, social cognition deficits pertained to: initiation of and response to joint attention, reduced gaze towards caregiver's eyes vs. mouth, difficulties automatically orienting to targets/forming visual expectations to environment, and face processing abnormalities (increased attention to mouth relative to eyes). Importantly, ToM was not supported as a BAP cognitive marker in siblings of children with ASD. Similarly, in older relatives, Sucksmith et al. (2011) also note various traits pertaining to social cognition such as ToM deficits, as measured by inference of mental states based on gaze pictures (RMET; Baron-Cohen & Hammer, 1997; Losh et al., 2009), deficits in detecting trustworthiness of faces, basic emotion recognition, discerning emotional content of complex social scenes, differences in face processing strategy (e.g. Adolphs, Spezio, Parlier, & Piven, 2008), and eye gaze processing and social orienting difficulties (Wallace, Sebastian, Pellicano, Parr, & Bailey, 2010). Interestingly face recognition and memory deficits found only partial support (Dalton, Nacewicz, Alexander, & Davidson, 2007; Wallace et al., 2010). Hence, social cognition deficits, and in particular face and gaze processing deficits, not only represent the behavioural expression of ASD genetic liability (i.e. endophenotypes), but also permit testing of ASp hypo-Mentalism or Empathizing deficits at the

behavioural level. Interestingly, SSD also features such social cognition deficits, notably for face and gaze processing, suggesting they may apply to PSp.

1.5.2 BEHAVIOURAL CORRELATES OF SSD

In SSD research, many different cognitive deficits were proposed to explain schizophrenia symptoms and functional deficits. The existence of cognitive deficits associated with schizophrenia dates back from the origins of the term. For instance, Bleuler (1911) observed the schizophrenic tendency for uncommon associations, echoing contemporary research about cognitive deficits (i.e. loosening of associations, distant associations in fluency tasks).

Globally, schizophrenia patients feature impairments in three main domains: episodic memory, attention, and executive function and working memory (Braff & Freedman, 2002; Goldberg, David, & Gold, 2003; Ritsner & Gottesman, 2011). The deficit domains are comparable for individuals with schizotypal PD (O'Flynn et al., 2003). More precisely, schizophrenia patients have problems with maintaining vigilance over a period of time (i.e. sustained attention; Continuous Performance Task), as well as problems responding selectively to salient information while ignoring irrelevant information (i.e. selective attention; e.g. Stroop task). Schizophrenia patients have problems remembering events (i.e. bindings items and spatiotemporal context), they show slower learning slopes, impaired recognition memory, notably related to an inefficient encoding. Schizophrenia patients have difficulty in problem solving, notably in adjusting flexibly a classification rule as a function of a feedback (WCST). They also have difficulty with short term storage-retrieval of verbal information (i.e. listening to a list of letters and repeating it immediately after, e.g. letter-number span), in particular when it involves a brief delay, large set sites or an additional cognitive operation (e.g. reordering letters and numbers while retrieving them).

At a more fundamental cognitive processing level, schizophrenia patients have abnormal early information processing, notably related to positive and/or negative symptoms. Positive symptoms (e.g. perceptual aberrations, hallucinations, distraction) were associated with dysfunctions in early sensory gating and information processing (e.g. prepulse inhibition deficit, O'Flynn et al., 2003). Also, positive symptoms such as visual hallucinations or perceptual aberrations could be caused by abnormal visual processing, for instance an impairment of the fast dorsal magnocellular visual pathway (striate, prestriate, superior temporal sulcus). This visual pathway is responsible for processing coarse visual information (i.e. low spatial frequencies, contrasts, global features), but also motion (e.g. eyes motions perception). Other deficits include

oculomotor deficits (e.g. antisaccade task, deficits in smooth-pursuit eye movement task). These faulty processing may be responsible for aberrant sensory experiences, later fueling delusional beliefs (Goldberg et al., 2003).

Interestingly, these core deficits were replicated in unaffected relatives of schizophrenia patients, schizotypal PD patients, and healthy controls with schizotypic traits, with positive and/or negative schizotypic traits (O'Flynn et al., 2003; see also Ettinger et al., 2014). Positive traits, as well as social anhedonia associated with working memory deficits. Both positive and negative traits associated with deficit in sustained attention, as measured by the Continuous Performance Task. Positive rather than negative (anhedonia) traits associated with magnocellular deficits in motion sensitivity and backward masking. More recently, a study pointed out that deficits in visual backward masking associated with cognitive disorganization, but neither with positive nor with negative schizotypic traits (Cappe et al., 2012). Since these fundamental deficits can be found in schizophrenia patients as well as in unaffected relatives and schizotypes, they are putative intermediate phenotypes or endophenotype candidates (Sitskoorn, Aleman, Ebisch, Appels and Kahn, 2004; Snitz et al., 2006).

Clearly, the classical candidate cognitive endophenotypes of SSD do not relate directly with social cognition. Sitskoorn et al. (2004) performed a meta-analysis on 37 studies comparing relatives of schizophrenia patients and controls, in search of the most reliable endophenotypes. Some of the task with the largest effect sizes measured, by decreasing order of effect sizes: verbal memory (immediate and delayed free recall), visuo-motor tracking, attention/verbal memory span , and language/executive function (verbal fluency). Likewise, Snitz et al. (2006) retained endophenotype candidates for different function categories: attention/working memory, verbal memory, visual memory, executive function, spatial ability, motor function, language function and general intelligence. Among these, those with largest effect sizes pertained to different sustained attention deficits. Yet, none of the endophenotype reviewed pertained to social cognition, such as ToM or social cues processing.

Although social cognition and social brains abnormalities are not traditionally present among abovementioned endophenotype candidates, they have been discussed, and start to appear in the debate. Based on an impressive review of genetic, phylogenetic, functional anatomical cognitive and clinical research, Burns (2004) claimed that schizophrenia was a disorder of social brain and social cognition. For superior cognitive functions, Brüne (2005) reviewed ToM impairments in schizophrenia. For more basic cognitive functions, recent empirical results also support face and gaze processing abnormalities in schizophrenia (Hooker & Park, 2005; Langdon et al., 2006), with face memory as an endophenotype candidate (Calkins, Gur, Ragland, & Gur, 2005). Ritsner and

Gottesman (2011) summarized endophenotype candidates of functional psychoses (i.e. schizophrenia but also bipolar and major depression disorder)[12]. Among the psychological or neurocognitive endophenotypes, few pertain to social cognition. We noted, for schizophrenia: face recognition deficits (Sachs, Steger-Wuchse, Kryspin-Exner, Gur, & Katschnig, 2004; Calkins et al., 2005; Leppänen et al., 2008), facial emotion recognition (Erol, Mete, Sonmez, & Unal, 2010), declarative facial memory (Glahn et al., 2010), and for bipolar and/ major depression disorders: cognitive impairment and an isolated facial emotion processing deficit (Summers, Papadopoulou, Burno, Cipolotti, & Ron, 2006), nonspecific deficits in face-emotion recognition (Brotman et al., 2008), and in declarative facial memory (Glahn et al., 2010).

Importantly, we observe that the above-mentioned tasks do not *explicitly and directly* tap social cognition, but social perception, more precisely face processing (Ritsner & Gottesman, 2011; see also Chen, 2011). The relationships between ToM impairments and lower-level social cues processing is often hinted, yet remains poorly understood. Indeed, social cognition deficits are just beginning to be considered in schizophrenia/psychosis endophenotype research. Even though one could reduce a priori any face processing deficit to a more fundamental non-social processing deficit (e.g. working memory, attention etc.), evidence point to a certain face specificity of certain of these deficits (Chen, Norton, McBain, Ongur, & Heckers, 2009), and possible endophenotypes (Calkins et al., 2005). Although it is contentious, SSD deficits or abnormalities in social cues processing could be related to over-mentalizing, and as such may reflect hyper-Mentalistic features, as for instance over-sensitivity to gaze (Hooker & Park, 2005; Langdon et al., 2006).

Below we review face and gaze processing task reported in both ASD and SSD research, which may be useful to distinguish their behavioural phenotypes and may show association with Mentalistic personality traits, on ASp and PSp.

1.5.3 FACE AND GAZE PROCESSING

Joint assessment of personality traits and behaviour permits to test Crespi and Badcock (2008) hypothesis that hyper- and hypo-Mentalistic traits influence behaviour in diametrically

12 Indeed, Gottesman and Ritsner (2011) grouped schizophrenia, schizoaffective disorder, bipolar disorders and major depression disorder under the umbrella term of "functional psychoses". Authors base this wider categorization on the ground these diagnostic categories share clinical presentations, endophenotypes and genes, and also co-occur. Important for the present essay, Gottesman and Ritsner's "functional psychoses" category clearly corresponds to Crespi and Badcock's "psychotic spectrum" conditions category (see also Shevlin, McElroy, Bentall, Reininghaus & Murphy, 2016).

opposite directions. Both autism and psychosis are considered as disorders of the social brain, notably because they feature social cognition deficits (e.g. Burns, 2004; Baron-Cohen & Belmonte, 2005). Yet, it is unclear whether these social cognition deficits stem from similar causes in autism and psychosis or whether different causes lead to comparable deficits. Crespi and Badcock (2008) proposed that both autism and psychosis would be disorders of the social brain, but that both would exhibit diametrical opposition with regard to Mentalism. Instead of testing patients, they proposed to test their theory using healthy individuals characterized by autistic and schizotypic personality traits, that is healthy ends of ASp and PSp. Crespi and Badcock (2008) expected that opposing ASp and PSp personality trait features correspond to opposite cognitive styles, notably expressed in particular gaze and face processing.

Face and gaze processing are ideal targets to attempt to distinguish opposite autistic and schizotypy phenotypes or cognitive styles. We can already mention some important reasons justifying the testing of face and gaze processing.

First, since social cognition *per se* (e.g. ToM, joint attention, social interaction) relies on (i.e. is a necessary but not sufficient condition to) the understanding of social cues, such as those conveyed by face, gaze or mouth (i.e. social perception; Adolphs, 2001), studying social cues processing may provide a more fundamental explanation to social cognition deficits. To put it shortly: *before studying higher level cognitive processes (e.g. ToM), one would rather study the lower level cognitive processes (e.g. gaze perception, face identification) onto which the higher-level cognitive processes rest*.

Second, face and gaze processing were featured among the cognitive processes that Crespi and Badcock (2008) claimed to be opposite between ASp and PSp. Hence, testing them allows an operationalization close from their theory, accurate hypotheses focused on social cognition.

Third, face and gaze processing are simple, relatively low level cognitive processes. Testing lower level cognitive processes may be simpler, result in more reliable testing (better controlled experiments), and might circumvent confounds related to higher level cognitive tasks (e.g. cultural or linguistic variations, metaphor understanding in ToM).

Fourth, face and gaze processing were tested in both ASD and SSD research. These processes are relevant for each ASD and SSD domains independently from the issue of ASp-PSp relationships (see below Chapter 1.5.3.1). Also, as we will review below, face and gaze processing deficits were reported, but not always replicated in both spectra. Sometimes, similar but attenuated deficits were found in unaffected relatives, and sometimes even in healthy participants with heightened autistic and schizotypic traits participants.

Fifth, face and gaze processing are not social cognition *per se* but belong to the associated functions of social cognitions (Kennedy & Adolphs, 2012). In this context, social perception refers to the processing of social cues such as face and gaze, such as GD, face expression, face identity (Adolphs, 2001). Importantly, the brain areas and networks subserving face and gaze processing are abnormal in both ASD and SSD (Burns, 2004; Green, Horan, & Lee, 2015; McAlonan et al., 2005; Pelphrey, Shultz, Hudac, & Van der Wyk, 2011), as well as the white matter tracts connecting these areas to other areas of the social brain (for a review in ASD: Burns, 2004; Hadjikhani, Joseph, Snyder, & Tager-Flusberg, 2006, 2007; for a review in SSD: Jou et al., 2011). Some of these findings can be extended to ASp and PSp, as suggested by studies on unaffected relatives (Calkins et al., 2005; Sucksmith et al., 2011).

In the end, face and gaze processing represent a coherent, convenient, theoretically sound approach to distinguish between autistic and schizotypy cognitive styles, likely based on underlying neurocognitive endophenotypes. The associations of face and gaze processing with "shared" and "diametrical" Mentalistic traits will allow to test the diametrical model. Below, we briefly review findings relatively to (i) face processing, and more precisely face recognition (i.e. face identity and face memory) with reliance on configural processing, (ii) GD processing, and (iii) GC of attention. We will do it briefly for ASp and PSp, and will focus on the available findings for autistic and schizotypic traits.

1.5.3.1 FACE PROCESSING DEFICITS IN ASP AND PSP

Face processing per se was well studied in ASD and SSD (e.g. Butler et al., 2008; Dawson et al., 2002; Shin et al., 2008; Weigelt, Koldewyn, & Kanwisher, 2012), although face emotion processing is even more popular (Harms, Martin, & Wallace, 2010; Kohler, Walker, Martin, Healey, & Moberg, 2009). Studies dealing with face processing and autistic and schizotypic personality traits in healthy populations also exist, but are obviously fewer (e.g. Batty, Francis, Innes-Brown, Joshua, & Rossell, 2014; Halliday, MacDonald, Sherf, & Tanaka et al., 2014; Rhodes, Jeffery, Taylor, Hayward, & Ewing, 2014).

Haxby, Hoffman and Gobbini (2000) proposed that face processing requires a distributed network of brain areas (e.g. inferior occipital gyri, superior temporal sulcus, lateral fusiform gyrus), either belonging or associating with the social brain. For face identity perception, early perception of facial features (notably facial configuration) occurs in inferior occipital gyri. Since in our case we deal with static visual stimuli, information is transmitted to lateral fusiform gyrus that processes

invariant aspect of faces, such as identity. Finally, lateral fusiform gyrus interacts with anterior temporal cortex to retrieve personal identity, name, and associated biographical information. Configural face processing refers to an encoding and retrieval of the arrangement of facial features (e.g. nose, eyes, mouth etc.), whereas featural face processing refers to the encoding of the facial features themselves, and their very peculiarities.

Face processing is typically investigated through several paradigms, manipulating configural and/or featural face processing. For instance, face inversion, Thatcher effect, and part-whole effect have in common that they allow the measurement of two different processes involved in face recognition: featural and configural face processing (Haxby, et al., 2000). In this context, face inversion effect (IE) refers to the fact that a face that was encoded upright is *disproportionately* more difficult to recognize when inverted (i.e. presented upside down), as compared to an object encoded upright and presented inverted (Valentine, 1988; Yin, 1969). The (magnitude of the) IE would account for one's reliance on a configural strategy of face encoding (and recognition). Also, since configural strategy is most prominently used for faces (i.e. disproportionate IE for faces as compared to objects), the magnitude of IE is expected to be larger with faces than objects. Hence, in this context, the magnitude of IE reflects the "faceness" (i.e. resemblance with face) of the stimulus.

In ASp, a broad face processing deficit exist, although not related to any qualitative impairment as previously thought, but a rather quantitative one (Weigelt et al., 2012). Indeed, face processing is qualitatively similar in ASD as in neurotypicals, because of the Thatcher effect, face IE and part-whole effect are preserved. This contradicts previous claims of impaired configural or holistic processing in ASD (Dawson et al., 2005; Gauthier, Klaiman, & Schultz, 2009). There however seems to be a broad deficit relatively to face recognition, in particular when memory demands are increased, that is an important duration between encoding and retrieval (Weigelt et al., 2012). With or without memory demands, a broad face recognition impairment was considered so robust in ASD (Hedley, Brewer, & Young, 2014), and more precisely adaptive coding was proposed to be an ASD endophenotype (Rhodes, Jeffery, Taylor, & Ewing, 2013). Even healthy participants with heightened autistic traits showed deficits in a face memory task, without the need of any increased memory load (Halliday et al., 2014).

In PSp, configural processing deficits were reported, although inconsistently. Most studies reported a normal face IE in schizophrenia patients (Schwartz, Marvel, Drapalski, Rosse, & Deutsch, 2002). Some authors nonetheless reported face configural processing deficit in schizophrenia patients, and proposed them to be responsible for emotion recognition deficits (Bauser et al., 2012; Caharel et al., 2007; Joshua & Rossell, 2009; Shin et al., 2008; Walthers et al., 2009; Watson, 2013). Importantly, the existence of a possible configural processing deficits in

schizophrenia justifies study of simple face processing, notwithstanding the interest of emotion processing for schizophrenia. Attempting to understand the configural processing deficit in schizophrenia, Chambon, Baudouin and Franck (2006) proposed that a configural encoding of face was impaired. Other reports suggest that a broad configural encoding deficit results in an over-reliance on featural information (Caharel et al., 2007; Joshua & Rossel, 2009; Shin et al., 2008). With schizotypic traits, Batty et al., (2014) reported a normal IE, but a deficit in configural face encoding. Authors also associated a configural face encoding deficit with an over-reliance on featural information (what we could aptly call, a local processing bias, well-known in ASp literature Happé, 1999; Happé & Frith, 2006).

1.5.3.1.1 FACE AND IE AND RELIANCE ON CONFIGURAL PROCESSING

Face IE, as such, may be informative and adequate for opposing ASp and PSp cognitive profiles, yet not in the light of a global-local/configural-featural opposition. Crespi and Badcock (2008) assumed that ASp and PSp would be opposite regarding social cognition and global vs. local processing biases. Yet, the lack of configural processing deficit in ASD (Weigelt et al., 2012) questions previous claims (Behrmann et al., 2006). Also, the existence of a configural deficit in schizophrenia (Butler et al., 2008), and local processing biases (Silverstein & Keane, 2011), question the existence of a global processing bias in SSD (Bellgrove et al., 2003), or more generally psychosis as interpreted by Crespi and Badcock (2008). As a result, global-local opposition may not be adequate to distinguish ASp and PSp phenotypes. Merely transposing a shaky global-local opposition to face processing, i.e. configural-featural opposition may not be adequate either. So, we propose an alternative strategy, still based on the face specificity of IE.

1.5.3.1.2 PAREIDOLIA-PRONENESS AND ENCODING STYLES

Our alternative strategy consists in measuring the liability of participants to process ambiguous FLOs stimuli (i.e. objects looking like faces) like faces rather than objects. Obviously, a tendency to process ambiguous stimuli as faces or as objects may account for hyper- and hypo-Mentalistic cognitive styles, respectively. We explain below the relevance of this approach for ASp and PSp.

Schizophrenia and schizotypy are both characterized by a tendency to apophenia and its visual version, pareidolia, whereas no such reports exist for ASp. Indeed, apophenia consists in

attributing a meaning to unrelated stimuli or events, such as seeing a meaningful connection between reading the word "thunder" in the Bible, and a simultaneous crack of thunder outside (Brugger, 2001). Likewise, pareidolia specifies a visual form of apophenia, i.e. the tendency to see a meaningful visual pattern in meaningless noise (Belayachi, Laloyaux, Larøi, & Van der Linden, 2014), such as seeing Jesus on a toast, or the Virgin Mary in a tortilla (Liu et al., 2014). Apophenia was reported in healthy schizotypy (Brugger, 2001; Fyfe, Williams, Mason, & Pickup, 2008) and in SSD (Vercammen, de Haan, & Aleman, 2008). Face pareidolia was also reported in paranormal and religious believers (Riekki, Lindeman, Aleneff, Halm, & Mortimo, 2012). Belayachi et al. (2014) proposed that a hasty, internal liberal encoding style, as defined by Lewicki (2005), might underlie apophenia, and pareidolia (i.e. false-alarm-proneness). Interestingly, Fyfe et al. (2008) claimed that over-mentalizing would be associated with positive schizotypy, and Brosnan et al. (2013) found an association between empathizing and "jumping to conclusion bias", usually associated with schizotypy. Hence, a common hyper-associative/apophenia-prone/liberal/internal cognitive style may underlie both "jumping to conclusion bias" and over-mentalizing in schizotypy. In contrast, there is no evidence of pareidolia-proneness as a function of ASD or autistic traits. Yet, Brosnan et al. (2014) showed opposite "circumstantial reasoning bias" in ASD, suggesting ASD individuals tend to accumulate information before deciding or responding (i.e. miss-proneness). For this reason, ASD individuals may be less pareidolia-prone, and we predict an *increased* apophenia proneness as a function of *decreased* autistic traits.

Although face pareidolia is a subjective experience, behavioural performance and neural correlates can indirectly quantify, and thus evidence, face pareidolia, notably as an automatic phenomenon, not arising from a late interpretation. Face pareidolia was studied using FLOs from various categories (Hadjikhani, Kveraga, Naik, & Ahlfors, 2009; Robert & Robert, 2000), or from one category such as cars' fronts pictures (Windhager et al., 2008). For instance, FLOs elicited EEG components comparable to those of faces, although weaker (Caharel et al., 2007; Churches, Baron-Cohen, & Ring, 2009). Heterogeneous face-like objects and cars' fronts activated face-specific brain regions (Hadjikhani et al., 2009; Kühn, Brick, Müller, & Gallinat, 2014). For instance, Hadjikhani et al. (2009) observed that FLO elicited a brain correlate roughly at the same time and place as compared to those of faces. The early brain response to FLO could not result from a late interpretation. Behavioural studies showed that orientation detection was faster for upright faces as compared to upright FLOs, while accuracy did not differ (Caharel et al., 2007). Also, Windhager et al. (2010) showed that cars' fronts elicited a face-like visual exploration mostly driven by headlights, as eyes do with real faces. Yet, none of these studies proposed a way to quantify the pareidolia effect strictly solely with participants' behavioural responses.

We think it is possible to measure behaviourally a pareidolia effect using a recognition (old-new) paradigm with stimuli inversion. Concretely, we expect the face pareidolia-proneness to be manifested by a FLO IE tending to be comparable to Face IE. Since IE is *disproportionately* greater for face stimuli as compared to objects (Valentine, 1988; Yin, 1969), and FLOs elicited brain and behavioural correlates partially similar to those of faces (e.g. Caharel et al., 2007; Hadjikhani et al., 2009; Kühn, Brick, Müller, & Gallinat, 2014), FLO IE magnitude should lay in-between those of faces and objects, more or less shifted towards the former or the latter. We proposed to quantify the pareidolia effect with a pareidolia index resulting from the difference between FACE inversion and FLO IEs (FACE-IE – FLO-IE). Hence, the smaller this difference (i.e. the pareidolia index), the larger the pareidolia effect. The larger this difference, the smaller the pareidolia effect.

As such, face pareidolia is particularly interesting for contrasting hypo- and hyper-Mentalism. Indeed, face pareidolia is by definition a visual *facial, hence social*, type of apophenia. Hence, one's pareidolia effect may account for one's hyper- or hypo-Mentalism. Concretely, one's *larger* face pareidolia effect (i.e. *smaller* pareidolia index, *smaller* FACE-IE – FLO-IE) should be associated with increased *hyper*-Mentalism (i.e. positive schizotypy), whereas *smaller* face pareidolia effect (i.e. *larger* pareidolia index, *larger* FACE-IE – FLO-IE) should be associated with increased *hypo*-Mentalism (i.e. autistic mentalizing deficits).

Obviously, relating pareidolia-proneness and Mentalism is somehow indirect and disputable. In this respect, gaze processing may represent an even more direct and fundamental approach to investigate hypo- vs. hyper-Mentalistic tendencies. Indeed, gaze conveys many different pieces of information that are key to interpretation of others' mental states (i.e. ToM, mindreading). We present this below.

1.5.4 GAZE PROCESSING DEFICITS IN ASP AND PSP

1.5.4.1 GAZE DIRECTION PROCESSING

Gaze processing, and in particular GD processing, is another potential function distinguishing ASp and PSp, along a Mentalism continuum. Crespi and Badcock (2008) contrasted gaze processing in autism and psychosis claiming the former would be characterized by under-sensitivity to gaze, whereas the latter would be characterized by over-sensitivity to gaze, notably in lower ends of ASp and PSp. In this context, under-sensitivity to gaze may be associated to hypo-Mentalism, and over-sensitivity may be associated to hyper-Mentalism. Even though gaze processing is not social cognition or mentalizing *per se*, it does nonetheless contribute to face perception, emotion perception, attention shifting, and mentalizing (Emery, 2000; Itier & Batty,

2009). Indeed, a crucial brain area for gaze processing is superior temporal sulcus (STS; Haxby et al., 2000). STS codes for biological motion, such as that of hands, body and gaze (Frischen et al., 2007). More precisely, STS anterior part specifically codes for static GDs (Carlin, Calder, Kriegeskorte, Nili, & Rowe, 2011). Interestingly, STS is a part of social brain "mentalizing" network (Kennedy & Adolphs, 2012). Additionally, STS is connected with face processing network that contributes to recognition of face identity (mostly, fusiform face area; Haxby et al., 2000), amygdala for emotion monitoring, and parietal cortex for attention shifting (more precisely, intraparietal sulcus), which explains joint attention (Frischen et al., 2007). As a result, gaze processing is a necessary but not sufficient condition to mentalizing, and more concretely for joint attention. Yet, as far as mentalizing is affected in ASp and PSp, can it be attributed to abnormal gaze processing?

1.5.4.1.1 GAZE DIRECTION PROCESSING IN ASP

In ASD, eye contact has long been reported to be impaired (Kanner, 1943; Asperger, [1944] 1991). Also, the *DSM-IV-TR* (APA, 2000) includes eye-to-eye gaze impairment as one of social interaction and communication impairment present in ASD. Buitelaar (1995) reviewed findings about gaze avoidance and showed absence of visual reciprocity and qualitative impairments were present in ASD individuals, whereas gaze avoidance was not universal. Individuals with ASD report that eye-to-eye gaze is unpleasant (Robison, 2007, cited by Tanaka & Sung, 2013). From the functional and anatomical point of view, GD processing involves STS, and most precisely posterior STS in the case of static gaze stimuli (Haxby et al., 2000). As reviewed by Senju and Johnson (2009), evidence favours the fact ASD individuals passively omit but do not actively avoid eye contact. These authors think that eye contact behaviour in ASD is based on an atypical specialization of the social brain network (i.e. "fast-track modulator model"). More precisely, fast subcortical detection of face and gaze are impaired. A competing hypothesis, however, claims that "eye avoidance" exists in ASD, and is a compensatory strategy to actively avoid unpleasant over-arousal caused by eye-contact (Tanaka & Sung, 2013). As a consequence, eye avoidance strategy would impair ability for encoding and discrimination of facial information, notably GD. Nevertheless, abnormal gaze processing in ASD is a fact, irrespectively of the tasks and the populations considered, suggesting it might also apply to healthy end of ASp.

Several studies related purely to GD perception showed abnormal behaviour and neural responses to gaze, with or without emotional stimuli, in both ASD adults and children, and unaffected relatives (BAP, hence ASp), as compared to controls, as well as in healthy individuals high as compared to low in autistic traits. Indeed, in ASD as compared to healthy controls, neutral

faces elicited increased amygdala signal, and less saccades to eye region, as a function of perceived threat (Tottenham et al., 2014). ASD adults showed altered GD perception, in particular in centre gaze stimuli (Wallace, Coleman, Pascalis, & Bailey, 2006; Wallace et al., 2010). In healthy men but not women, the higher the autistic traits, the smaller the gaze angle had to be for the participants to consider it as directed onto them (Matsuyoshi et al., 2014; see also Sucksmith et al., 2011). To put it differently, the higher men's' autistic traits, the more frontal had to be a gaze (a gaze angle closer to 0°) for them to judge it directed towards them. Moreover, healthy male infant relatives of children with ASD showed electrophysiological abnormalities similar to ASD adults (i.e. late attentional modulation and referential processing of eye gaze) during centre gaze processing (Elsabbagh et al., 2009). These results are in line with recent claims of gaze processing abnormalities as an endophenotype of ASD (Matsuyoshi et al., 2014). Importantly, such early gaze processing deficits may impair the development of shared and joint attention, and higher-level mentalizing functions, ToM skills, ultimately causing social deficits (Itier & Batty, 2009 for a review; Jellema et al., 2009, Kuhn et al., 2010; Tanaka & Sung, 2013). These results suggest that GD processing is a feature common to ASp.

1.5.4.1.2 GAZE DIRECTION PROCESSING IN PSP

In PSp, empirical data also show gaze processing abnormalities in schizophrenia patients, although differently than in ASp. Self-referential information, such as a gaze directed at the patient, may be over-interpreted and feed paranoia and delusions (Couture, Penn, & Roberts, 2006). Rosse, Kendrick, Wyatt, Isaac and Deutsch (1994), and Hooker and Park (2005) showed that schizophrenia patients tended to over-attribute gaze as directed at them, as compared to healthy controls. Interestingly, Tso, Mui, Taylor and Deldin (2012) replicated these findings, and showed gaze over-attribution in schizophrenia patients mostly occurred when GD was ambiguous. Additionally, they showed that this over-attribution was a function of patients' socio-emotional functioning deficits, and negative symptoms (i.e. avolition/amotivation). According to the authors, their findings echo deficits in social brain areas. Interestingly, social brain areas subserve at the same time self-referential processing, and a common factor underlying negative symptoms and ToM (Amodio & Frith, 2006). To our knowledge, there are no studies about GD perception as a function of healthy schizotypy, or in patients' relatives, that is the healthy end of PSp. Yet, some evidence support possibly opposite phenotypes between ASp and PSp.

Studies comparing GD processing as a function of ASD and SSD diagnostic or corresponding autistic and schizotypic traits are scarce, yet some evidence supports opposite

patterns, as advocated by Crespi and Badcock (2008). Interestingly, Matsuyoshi et al.'s (2014) results on healthy individuals with heightened autistic traits contrast with those of Hooker and Park (2005) and Tso et al. (2012). Notwithstanding the different methodologies, these findings lend impression that ASD features a conservative or underinclusive bias, perceiving gaze as directed onto them only at smaller gaze angles. On the contrary, SSD features an opposite liberal/overinclusive bias, perceiving gaze as directed onto them also at greater gaze angles (Hooker & Park, 2005; Lewicki, 2005; Tso et al., 2012). Differently, Sasson et al. (2007) contrasted ASD and SSD gaze processing using passive viewing. Both ASD and SSD patients spent less time fixating faces than healthy control. However, when a face was presented, SSD patients showed a similar but delayed orienting response towards face parts, as compared to controls. In contrast to both groups, ASD patients did not show a difference in orienting to face parts as a function of presence or absence of face. As for ASD and SSD, ASp and PSp might be opposite with respect to gaze processing.

1.5.4.2 GAZE CUEING OF ATTENTION

Other studies went further and assessed the redirection of attention through gaze, that is "gaze cueing" of attention, which is an experimental operationalization of joint attention (Driver et al., 1999; Frischen et al., 2007). GC effect refers to the automatic redirection of an observer's attention following observation of a conspecific's averted gaze. Obviously, analogue redirection of attention through gaze renders possible joint attention (Emery, 2000), such that GC is an experimental way to assess a basic automatic redirection of attention that renders possible joint attention. From the functional viewpoint, redirection of attention does not only involve STS for GD processing, but also the intraparietal sulcus subserving spatially directed attention (Haxby et al., 2000). As could be expected, several studies reported abnormal gaze processing, notably GC, in ASD and SSD (Emery, 2000; Frischen et al., 2007 for reviews).

1.5.4.2.1 GAZE CUEING OF ATTENTION IN ASP

In ASp, deficits were reported sometimes in children and more consistently in adults, mostly in clinical popualtions. Interestingly, autistic traits also proved to be associated with GC magnitude. Senju, Tojo, Dairoku and Hasegawa (2004) showed that ASD children show the expected GC effect. However, neurotypicals show an increased cueing effect with gaze as compared to arrow stimuli. This difference is interpreted as a normal advantage for processing social stimuli. Yet, ASD

children do not show such an advantage. As reviewed by Frischen et al. (2007), studies paradoxically showed more reliable GC effect deficits in adults than in children. Instead, we should expect that gaze deficits in high-functioning ASD or Asperger individuals would be more severe in childhood and improve towards adulthood, along with social functioning. Kylliäininen and Hietanen (2004) and Senju et al. (2004) did not always find impairments in children. In adults with ASD, Ristic et al. (2005), and Vlamings, Stauder, van Son and Mottron (2005) reported GC deficits. Other studies investigated GC effect in healthy individuals with high as compared to low autistic traits, that is at the healthy end of ASp. Bayliss and Tipper (2005) reported that autistic traits correlated with a smaller GC effect. Another study replicated these findings, additionally showing that this association between increased autistic traits and decreased GC effect was especially true in men rather than women (Bayliss, Di Pellegrino, & Tipper, 2005). More interesting, Alwall, Johansson and Hansen (2010) showed that an increased GC effect associated with increased Empathizing scores in healthy individuals (EQ; Wakabayashi, Wheelwright, & Baron-Cohen, 2006b).

1.5.4.2.2 GAZE CUEING IN PSP

In PSp, there is some evidence of gaze over-sensitivity, manifested by a facilitated GC, yet studies are scarce and absent for healthy schizotypy. Langdon et al. (2006) reported an over-sensitivity to gaze in schizophrenia patients. In their first experiment, patients were more sensitive to the displayed gaze. Comparing young schizophrenics to healthy controls, Magnée, Kahn, Cahn and Kemner (2011) showed a preserved GC effect in the former group, but accompanied of a prolonged brain activity following GC. These authors interpreted these findings as reflecting a prolonged evaluation of displayed gaze. Ultimately, this could be interpreted as a similar over-sensitivity to gaze. To our knowledge, similar studies were not conducted with healthy individuals with heightened schizotypic traits.

Taken together, gaze processing in ASp and PSp shows some evidence of opposite under- and over-sensitivity, although this may be clearer for ASp than PSp. Although corresponding evidence is scarce for healthy autistic traits and absent for healthy schizotypic ones, we believe that sensitivity to gaze and GC liability are suitable task to attempt contrast hypo- and hyper-Mentalistic phenotypes of ASp and PSp, respectively.

1.6 STUDIES AND PREDICTIONS

The following chapters (from 2nd till 5th) will present 4 different questions stemming from aforementioned psychometric and behavioural issues. We briefly review these questions and explain their interrelationships.

The second chapter presents the psychometric validation of the French version of the schizotypy questionnaire sO-LIFE (Mason et al., 2005). We translated and back-translated the original English version of the sO-LIFE, and collected *n*=1'048 responses in a French speaking undergraduate student population. We assessed the factor structure of the French sO-LIFE version using CFAs, comparing 3- and 4- factor models. We compared it with an English speaking reference sample of *n*=439 responders using multi-group CFAs, again comparing 3- and 4-factor models. Also, we reported correlation of sO-LIFE dimension scores with those of a broad personality instrument, the EPQR-A. We expected that the sO-LIFE would feature acceptable psychometric properties as well as the 4-factor structure. Finally, we discussed the sO-LIFE factor structure, its reliability, provided its descriptives, discussed its comparability in terms of psychometric features (e.g. reliability, dimensions) with the English version, and its relationships with a broad personality model. This work is published as Sierro, Rossier, Mason and Mohr (2016).

The third chapter presents the psychometric validation of the French version of the autistic traits questionnaire AQ, and an analysis of the relationships of autistic traits, with schizotypic traits (sO-LIFE) and broad personality dimensions (EPQR-A). We used a published French translation of the AQ, though without full psychometric evaluation (Sonié et al., 2011). We collected data on all three instruments in the same French speaking undergraduate students population (*n*=921). We used CFA to assess different AQ factor structures reported in the literature and identified an appropriate one for our French-speaking population. We expected that our results support an alternative factor structure of AQ with acceptable psychometric properties. Using this appropriate AQ factor structure, we reported on descriptive and psychometric features of AQ dimensions (e.g. reliability, sex differences). Relying on our previous validation of sO-LIFE, we examined the relationships between autistic and schizotypic traits, using correlations, and PCA. In particular, we examined overlap and opposition between autistic and schizotypic traits, with a particular emphasis on diametrical traits. As for autistic-schizotypic traits relationships we expected the existence of (i) overlapping/shared traits, but also (ii) diametrically opposite traits. Finally, we discussed these overlapping and opposing personality traits in light of theories of ASp-PSp relationships. This work is published as Sierro, Rossier and Mohr (2016).

A fourth chapter presents an investigation of the recognition of facial, object and ambiguous face-like object stimuli (FLO), as a function of autistic hypo-Mentalistic and positive schizotypy hyper-Mentalistic traits. We used an old-new recognition paradigm with inversion. In a set of 3 studies, we tested each time *n*=48 undergraduate students using this protocol and personality questionnaires. In particular, we investigated the relationship between hyper-Mentalism and pareidolia-proneness. We based our measure of hyper- and hypo-Mentalistic personality traits on the results of the previous psychometric chapters (in particular Chapter 3). We measured pareidolia-proneness by comparing IEs of face and FLOs, assuming that IE would represent the extent to which a FLO is processed like a face. We used regressions to test whether personality traits predicted behavioural performances. We expected that increased hyper-Mentalistic traits would relate to increased pareidolia-proneness, while increased hypo-Mentalistic ones would relate to decreased pareidolia-proneness. Also, we examined the basic performance in face/FLO/object recognition as a function of autistic and schizotypic traits. Finally, we discussed our results in light of previous literature, ASp-PSp relationships theories, and studies' limitations.

A fifth chapter presents an investigation of gaze processing as a function of autistic hypo-Mentalistic and positive schizotypy hyper-Mentalistic traits. We used two protocols measuring GD processing, and GC liability, in order to establish the relative sensitivity to gaze showed by participants. In the same experiment, *n*=68 participants responded to autistic and schizotypy questionnaires and completed both tasks. Again, we derived our measurement of hypo- and hyper-Mentalistic personality traits from the two first psychometric chapters. For GD, we contrasted the social and non-social, as well as centre and averted conditions, plus the centre gaze performance, to establish the relative sensitivity to gaze of participants. For GC, we contrasted the performance in conditions where gaze predicted cue location (congruent) with those where gaze did not predict cue location (incongruent). We computed a GC index, that represented one's liability to have one's attention automatically redirected by a displayed gaze. We used regressions to relate personality features and behavioural performances. We expected that increased hyper-Mentalistic traits would relate to increased gaze sensitivity in GC and GD, while increased hypo-Mentalistic traits would relate to a decreased gaze sensitivity. Finally, we discussed the relationships between hypo- and hyper-Mentalism of gaze processing, and the implication of their relationships for endophenotype research, and theories about ASp-PSp relationships, and study's limitations.

2 FRENCH VALIDATION OF THE O-LIFE SHORT QUESTIONNAIRE[13]

Abstract

The original 104 items Oxford-Liverpool Inventory of Feelings and Experiences (O-LIFE) questionnaire is a validated schizotypy questionnaire distinguishing three schizotypic dimensions (Unusual experiences, Introvertive anhedonia, and Cognitive disorganisation). It also includes items on Impulsive nonconformity assessing traits sensitive to borderline and antisocial personality. Recently, Mason et al. (2005) published a shortened 43-items version including all sub-dimensions. The aim of this study was to validate a French version of this short form and to study the relationships between its French and English-speaking versions. O-LIFE Short data was obtained from 1,048 students from two higher education institutions in the French speaking part of Switzerland. Results were compared with those from an English normative sample (n = 439). A series of CFAs showed acceptable configural and metric invariance across the two language versions. Moreover, results from the French data support the use of both 4 and 3 dimensional models of schizotypy and show expected correlations with other relevant self-report instruments. This French version of the O-LIFE Short form is an appropriate tool to use in French-speaking environments.

Keywords: Schizotypy, self-report, French translation, psychometric validation

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2.1 INTRODUCTION

Schizotypy describes a schizophrenic phenotype, i.e. individuals with a genetic liability to schizophrenia (Meehl, 1990). Meehl argued that schizophrenia risk entails both genetic liability and social-environment risk factors (e.g. schizotypal personality and resultant behaviour). Subsequent research followed largely two separate traditions (Mason & Claridge, 2006). In the dimensional tradition, schizotypy represents a healthy personality expression that may become clinically relevant when its expression is severe (see also Nelson et al., 2013). In the categorical tradition, schizotypy is taxonomic, carriers of the putative schizotaxia gene carry the genetic liability for schizophrenia (Lenzenweger & Korfine, 1992).

Both traditions predominantly use self-report questionnaires to assess schizotypy in healthy (Schizotypal Personality Questionnaire (SPQ): Raine, 1991; Chapman/Wisconsin Schizotypy scales (WSS): Chapman, Chapman, & Kwapil, 1994; Oxford Liverpool Inventory of Feelings and Experiences (O-LIFE): Mason et al., 1995) and sometimes in clinical populations (e.g. Cochrane et al., 2010). Importantly, analyses of schizotypy items and scales, when sufficiently inclusive, commonly reflect the three symptom dimensions reported in patients (Liddle, 1987; Arndt et al., 1991): positive symptoms (e.g. magical ideation, aberrant perceptual experiences), negative symptoms (e.g. anhedonia, social withdrawal) and cognitive disorganisation (mainly disordered thought and language).

Self-report questionnaires should be reliable and valid, possessing discriminant validity when selecting high and low "risk" individuals from healthy populations (e.g. Cappe et al., 2012), and in identifying clinical (e.g. Alvarez-Moya, Barrantes-Vidal, Navarro, Subira, & Obiols, 2007) populations. The original questionnaires in this field tend to be lengthy, consisting of over 70 items (SPQ = 74, O-LIFE = 104, WSS = 166). Yet, questionnaire length can raise response burden, potentially reducing both response rate and data quality (Galesic & Bosnjak, 2009). Also, shorter questionnaires are easier to use when assessing large populations. Hence, questionnaires have to be long enough to provide relevant information and short enough to optimize data quality. In addition, their psychometric properties should be assessed. So far, several shorter schizotypy questionnaires have been validated: the 22-items SPQ-B (Raine & Benishay, 1995), the 60-items WSS (Winterstein, Silvia, et al., 2011), and the shortened 43-items O-LIFE questionnaire (sO-LIFE) (Mason et al., 2005). With the present study, we continue this psychometric work by validating the sO-LIFE questionnaire in French. By inference, subsequent commentaries focus on the original O-LIFE and sO-LIFE.

The original 104-item O-LIFE was created using factor analyses on a large number of items from a wide range of sources. The 4-factor solution extracted showed an acceptable internal consistency (Mason et al., 1995), and was further supported by CFA (Mason, 1995). The 4 factors consist of three schizotypy subscales (positive schizotypy: Unusual Experiences (UnEx); negative schizotypy: Introvertive Anhedonia (IntAn); Cognitive Disorganisation (CogDis), and Impulsive Nonconformity (ImpNon), a construct close to Eysenck's Psychoticism dimension (Mason et al., 1995). Later, Mason et al. (2005) presented descriptive values and acceptable internal reliability for the sO-LIFE. Cella et al. (2013) confirmed the 4-factor solution (4F-solution) of the sO-LIFE. Lin et al. (2013), on the other hand, favoured a 3-factor solution (3F-solution) based on CFA results from an Australian young adult ultra high-risk sample, excluding ImpNon for lack of robustness.

To (i) enable sO-LIFE's use in additional languages and (ii) test whether the 3F- or 4F-solutions is preferable, we validated the French sO-LIFE in a Swiss French-speaking sample, and compared results to a UK English-speaking sample (English sO-LIFE). We conducted CFAs for each language (assessing its structure for each language) and a cross-language validation (testing between-language differences). Additionally, we correlated the French sO-LIFE with questionnaires assessing related constructs: the French-version of the Eysenck Personality Questionnaire Revised and Abridged (EPQR-A; Bouvard, et al., 2010) measuring Eysenck's personality dimensions associated with schizotypal traits (Burch et al., 2006) and the French-version of the SPQ-B (Ortuño-Sierra et al., 2013) whose dimensions measure schizotypal traits in a similar way to the O-LIFE (Asai et al., 2011).

2.2 METHOD

2.2.1 PARTICIPANTS

French-speaking university students (n = 1,048; 18-30 years old, 737 women) completed the French sO-LIFE online (see Supplementary Material). They were recruited during psychology courses at two local Universities (UNIL, EPFL). Of these, 514 were psychology students and 530 also completed the EPQR-A and SPQ-B questionnaires. OM collated 439 English-speaking participants (299 women) of corresponding age. They were not exclusively student-based, but were predominantly recruited via London-based Universities. All participants provided written informed consent prior to participation. The study was conducted in accordance with the guidelines of the declaration of Helsinki (World Medical Organization, 2013).

2.2.2 INSTRUMENTS

2.2.2.1 THE SHORT OXFORD-LIVERPOOL INVENTORY OF FEELING AND EXPERIENCES QUESTIONNAIRE (SO-LIFE)

The 43-item English sO-LIFE (Mason et al., 2005) assesses positive schizotypy (12 UnEx items, e.g. "Are your thoughts sometimes so strong that you can almost hear them?"), negative schizotypy (10 IntAn items, e.g. "Do you prefer watching television to going out with people?"), CogDis (11 items, e.g. "Are you easily confused if too much happens at the same time?") and ImpNon (10 items, e.g. "Do you at times have an urge to do something harmful or shocking?"). Participants indicate whether the statement is true or false. The number of positive responses (negative responses are reversely coded) is summed for each subscale so that higher scores indicate higher schizotypy. Normative values can be found in Mason et al. (2005). We translated the English sO-LIFE into French following common translation and back-translation procedures (e.g. Rossier, Quartier, Enescu, & Iselin, 2007). A French speaker of advanced English proficiency (GS) translated the English version into French. Subsequently, a bilingual person (naive to the English version) back-translated the French version. Comparing the two versions revealed minor differences, which were discussed by GS, the back-translater, and the senior researcher (CM) until agreement (see Supplementary Material for the French sO-LIFE and Cappe et al., 2012 for its use in a behavioural study).

2.2.2.2 THE SCHIZOTYPAL PERSONALITY QUESTIONNAIRE BRIEF FORM (SPQ-B)

The 22-item French SPQ-B (Ortuño-Sierra et al., 2013; Raine & Benishay, 1995) assesses positive schizotypy (6 "Cognitive-Perceptive" items (Cog-Per), e.g. "Have you ever had the sense that some person or force is around you, even though you cannot see anyone?"), negative schizotypy (8 "Interpersonal" items (Int), e.g. "People sometimes find me aloof and distant") and 6 "Disorganized" items (Dis) (e.g. "Some people find me a bit vague and elusive during a conversation"). Participants make true-false statements. The number of positive answers is summed for each subscale so that higher scores indicate higher schizotypy. Normative values can be found in Ortuño-Sierra et al. (2013).

2.2.2.3 THE EYSENCK PERSONALITY QUESTIONNAIRE REVISED AND ABRIDGED (EPQR-A)

The French 24-item EPQR-A (Bouvard et al., 2010) assesses Eysenck's personality dimensions with 6 Extraversion items (e.g. “Are you a talkative person?”), 6 Neuroticism items (e.g. “Does your mood often go up and down?”), 6 Psychoticism items (e.g. “Do you prefer to go your own way rather than act by the rules?”), and 6 “Lie” items (e.g. “Have you ever blamed someone for doing something you knew was really your fault?”) measuring social desirability (Shevlin, Bailey, & Adamson, 2002). Participants make true-false statements. The number of positive answers (negative responses are reversely coded) is summed so that higher scores indicate higher personality traits (or a higher likelihood of lying). Normative values can be found in Bouvard and Cosma (2008).

2.2.3 DATA ANALYSIS

To test for item reliability, we performed separate ordinal Cronbach alphas (Gadermann, Guhn, Zumbo, & Columbia, 2012; see also Lin et al., 2013). Cronbach alpha values above .70 were considered acceptable (Nunnally & Bernstein, 1994). Before performing CFAs, we submitted the raw data to a parcellation procedure using single-factor approach (see Landis, Beal, & Tesluk, 2000). Because the assumption of multinormality was not always met (Mardia test statistics), robust estimates were performed and reported throughout. Since our English data sample size was relatively small and data moderately skewed, we followed the recommendations by Lei (2009). For all CFAs, we chose Maximum Likelihood estimator with Satorra-Bentler scaled statistics, instead of weighted least squares (WLSMV), as used by Lin et al (2013). To confirm the stability of the latent structure of the French sO-LIFE, we performed CFAs on the French and English samples, separately. We computed the following fit indices: χ^2 per degrees of freedom (χ^2/df) (acceptable when < 3.00); Tucker Lewis index (TLI) and Comparative Fit Index (CFI) (both acceptable ≥.90, good ≥.95); badness of fit indices Root Mean Squares Error of Approximation (RMSEA) (close fit ≤.05; reasonable fit ≤.08); and a comparative fit index, Bayesian information criterion (BIC) (when the fit of two models is compared, the model with the lower BIC value is considered superior) (Brown, 2006). To compare the invariance of the latent structure of the sO-LIFE across languages, we performed CFAs on the English and French data. We tested for the 4F-solution (all 43 items) and 3F-solution (33 items, ImpNon items excluded). We report fit indices for each invariance level (i.e. configural, metric, and scalar). All analyses were performed using R software (R Core Team, 2014) with “lavaan” package (Rosseel, 2012).

2.3 RESULTS

2.3.1 PARTICIPANTS

A two-way ANOVA on age with sex and language group as between-subject factors showed a significant main effect of language group ($F(1,1483) = 488.41$; $p < .001$; $\eta^2_p = .25$. The mean (± SD) age (always in years) was higher in the English (24.9 ± 3.0) than French (21.3 ± 2.29) sample. There was no main effect of sex, $F(1,1483) = 3.34$, $p = .07$, $\eta^2_p = .002$ (women: 22.2 ± 3.04; men: 22.8 ± 2.92), but a significant interaction between sex and language group ($F(1,1483) = 12.66$; $p < .001$; $\eta^2_p = 0.01$). Scheffé post-hoc tests indicated that all comparisons were significant (all *p's* < .05), apart from the one showing that English men (24.7 ± 3.13) were comparable in age to English women (25.0 ± 2.94, $p > .05$). For the French sample, men (21.9 ± 2.34) were significantly older than women (21.1 ± 2.23, $p < .05$). Across both sexes, the English sample was older than the French sample.

2.3.2 SELF-REPORT QUESTIONNAIRES

Means, standard deviations, ordinal Cronbach alphas, Cohen's *d* effect size, skewness and kurtosis for each subscale and sample are given in Table 1. Table 1 also gives independent *t*-test results for sex differences. The latter showed that French women scored significantly higher in UnEx and CogDis but lower in IntAn than French men. English women scored higher in UnEx and CogDis than English men. Ordinal Cronbach alphas showed acceptable internal consistency for each subscale, apart from the Cronbach alphas just below acceptable values for IntAn in English women (.65), of marginal value for ImpNon in both English (.68) and French (.69) men.

Table 1. Normative and descriptive data for the French and English samples with standardized ordinal Cronbach's alphas (α), for women and men separately, as well as for the whole sample.

		Women		Men							
Language	Scales	α	*M* (±SD)	α	*M* (±SD)	α	*t*	*p*	*d*	*S*	*K*
French			*n* = 737		*n* = 311			*df* = 1047			
	Global	.88	14.52±6.25	.88	14.28±6.34	.88	0.55	.580	0.04	0.37	-0.08
	UnEx	.82	3.85±2.59	.84	3.43±2.57	.83	2.39	.017	0.16	0.47	-0.42
	CogDis	.83	5.7±2.77	.86	5.13±2.91	.84	3.04	.002	0.21	-0.04	-0.82
	IntAn	.73	1.99±1.69	.70	2.51±1.87	.72	-4.22	<.001	-0.29	0.98	0.91
	ImpNon	.76	2.97±2.05	.69	3.22±2.01	.74	-1.77	.077	-0.12	0.67	-0.05
English			*n* = 299		*n* = 140			*df* = 437			
	Global	.90	15.37±7.02	.88	13.69±6.63	.90	2.38	.018	0.24	0.23	-0.37
	UnEx	.89	3.90±3.04	.86	3.18±2.70	.88	2.40	.017	0.25	0.68	-0.30
	CogDis	.85	5.55±2.93	.82	4.60±2.72	.85	3.25	<.001	0.33	0.00	-0.88
	IntAn	.65	1.96±1.65	.75	2.11±1.87	.69	-0.86	.392	-0.09	0.85	0.24
	ImpNon	.70	3.96±2.07	.69	3.80±2.17	.68	0.73	.468	0.07	0.17	-0.59

Global: total sO-LIFE score; UnEx: Unusual Experiences; CogDis: Cognitive Disorganization; IntAn: Introvertive Anhedonia; ImpNon: Impulsive Nonconformity

2.3.1 CFAS

2.3.1.1 STRUCTURE EXAMINATION OF THE FRENCH SO-LIFE

The CFAs on the French-speaking sample compared a 1-factor solution to (i) a 4F-solution and (ii) 3F-solution. The CFA showed a better fit for the 4F-solution than the 1-factor solution, as indicated by a lower BIC value for the former (see Table 2). RMSEA and χ^2/df values indicated an acceptable fit for the 4F-solution. The TLI and CFI were, however, just below .900 and were, as such, not acceptable. When considering the 3F-solution, all indices (χ^2/df; RMSEA; TLI, CFI) indicated a better fit as compared to the 1-factor solution. All indices indicated an acceptable fit. When comparing the 4F- and the 3F-solution, fit indices were only within acceptable range for the 3F-solution. Removal of the ImpNon items raised TLI and CFI values slightly to an acceptable level and minimized the BIC value (Table 2).

Table 2. CFA results comparing the 4F-solution and 3F-solution for the English (n = 439) and French (n=1'048) sample, separately.

Language		Factors	χ^2	df	χ^2/df	TLI	CFI	RMSEA	BIC
French	4F-solution	1	1188.31*	209	5.68	.665	.697	.067	44419.61
		4	542.75*	203	2.67	.880	.895	.040	43777.51
	3F-solution	1	802.95*	119	6.75	.678	.718	.074	34458.04
		3	285.44*	116	2.46	.918	.930	.037	33928.16
English	4F-solution	1	674.445*	209	3.23	.698	.727	.071	18905.76
		4	341.24*	203	1.68	.908	.919	.039	18297.42
	3F-solution	1	460.25*	119	3.87	.710	.746	.081	14529.94
		3	202.55*	116	1.75	.924	.936	.041	14270.80

Note. * $p < .001$

4F: four factors; 3F: 3 factors; χ^2: chi-square; df: degrees of freedom; TLI: Tucker-Lewis Index; CFI: Comparative Fit Index; RMSEA: Root Mean Squares Error of Approximation; BIC: Bayesian Information Criteria

2.3.1.2 STRUCTURE EXAMINATION OF THE ENGLISH SO-LIFE

The analogue procedure for the English sO-LIFE (Table 2) showed a better fit for the 4F-solution than the 1-factor solution. The 4F-solution yielded acceptable fit according to all indices (χ^2/df; RMSEA; TLI, CFI). For the 3F-solution, this was better than the 1-factor solution. All fit indices indicating a close or acceptable fit. When comparing the 4F- and 3F-solution, the 3F-solution was superior having higher TLI and CFI indices as well as a lower BIC value.

Table 3. Multi-group CFA results comparing the 4F-solution and 3F-solution, respectively, between English (n = 439) and French (n = 1'048) sample.

Subscales	Invariance	χ^2	df	χ^2/df	TLI	CFI	RMSEA	BIC
4F (43 items)	Configural	881.73*	406	2.17	.890	.910	.040	62482.04
	Metric	1003.19*	424	2.37	.872	.883	.043	62470.19
	Scalar	1537.41*	442	3.48	.768	.780	.058	62824.99
3F (33 items)	Configural	486.21*	232	2.10	.921	.933	.038	48283.73
	Metric	604.61*	246	2.46	.895	.905	.044	48300.27
	Scalar	1022.08*	260	3.93	.789	.798	.063	48585.31

Note. * $p < .001$

4F: four factors; 3F: 3 factors; χ^2: chi-square; df: degrees of freedom; TLI: Tucker-Lewis Index; CFI: Comparative Fit Index; RMSEA: Root Mean Squares Error of Approximation; BIC: Bayesian Information Criteria

2.3.1.3 CROSS-LANGUAGE ASSESSMENT

The cross-language fit for the 4F- and 3F-solutions was assessed using multi-group CFAs on all data with "language" as between-subject factor. Results supported the assumption of invariance of the questionnaire structure across languages for both solutions, with all fit indices indicating acceptable fit (Table 3). The CFA showed that the TLI and the CFI are slightly better for the 3F- than 4F-solution. The result for metric invariance is, however, mixed. While some fit indices (χ^2/df; RMSEA) indicated acceptable value and the BIC value was at the lowest, TLI and CFI were barely acceptable for the 3F-solution, and not for the 4F-solution. At scalar invariance level, all fit indices worsened and were not acceptable anymore, neither for the 4F- nor the 3F-solution. As such, there was evidence for configural invariance in both solutions, but metric invariance was only acceptable for the 3F-solution. Scalar invariance, however, was not reached for either solution, making the direct comparison of English and French scores impossible.

2.3.2 CORRELATIONS WITH THE EPQR-A AND THE SPQ-B

We considered effects above .10 to be small, above .30 to be moderate and above .50 to be large (Cohen, 1988). The Spearman correlations showed the expected significance pattern (Table

4); conceptually related subscale scores of the sO-LIFE and the SPQ-B correlated positively (UnEx and Cog-Per, IntAn and Int, CogDis and Dis) with correlations ranging from moderate to high (Cohen, 1988). Significant (though moderate) positive correlations were also found between UnEx and Dis, CogDis and Cog-Per, and CogDis and Int. All sO-LIFE subscales scores correlated positively with Neuroticism scores, IntAn and CogDis correlated negatively with Extraversion, and ImpNon correlated positively with Psychoticism. Higher sO-LIFE subscale scores (apart from no relationship with IntAn scores) correlated negatively with Lie scores.

Table 4. Spearman correlations between sO-LIFE scores and SPQ-B scores and EPQR-A scores, respectively (n = 530).

		sO-LIFE			
		UnEx	CogDis	IntAn	ImpNon
SPQ-B	Cog-Per	.62***	.35 ***	.11*	.33***
	Dis	.43***	.46***	.23 ***	.37***
	Int	.21***	.35***	.43***	.15**
EPQR-A	Extraversion	$.07^{ns}$	-.20***	-.36***	.12**
	Neuroticism	.32***	.53***	.21***	.34***
	Psychoticism	.12**	$.06^{ns}$	$.04^{ns}$	.27***
	Lie	-.24***	-.12**	$.00^{ns}$	-.38**

Note. ^{ns} > .05; * < .05; ** < .01; *** < .001; *p*-values are corrected for type I error rate using Holm's corrections (Holm, 1979).
UnEx: Unusual Experiences; CogDis: Cognitive Disorganization; IntAn: Introvertive Anhedonia; ImpNon: Impulsive Nonconformity
Cog-Per: Cognitive-Perceptual; Dis: Disorganization; Int: Introversion

2.4 DISCUSSION

Schizotypy is relevant to personality (Mason & Claridge, 2006) and clinical high-risk (e.g. Alvarez-Moya et al., 2007) research. For this reason, it is important that schizotypy questionnaires are of appropriate length and show satisfactory psychometric properties (e.g. Cella et al., 2013). We provide normative values and the factor structure of the French sO-LIFE based on data from 1'048 Swiss French-speaking students (age range 18 to 30 years). Results showed that (i) the 4F-solution (with ImpNon items) and 3F-solution (without ImpNon items) of the French sO-LIFE have satisfactory psychometric properties; (ii) the French and English version have comparable structures; (iii) for the French sO-LIFE the internal consistency ranged from marginal to acceptable

(discrete values were lower than those reported in Mason et al., 2005; Lin et al., 2013), and did largely so for sex and language, separately; (iv) the French sO-LIFE scores correlated with related questionnaires in predictable ways; and (v) we largely replicated previous reports of significant sex differences with women scoring higher than men in UnEx and CogDis, and men scoring higher than women in IntAn (Mason et al., 1995; Cella et al., 2013).

The CFAs on French sO-LIFE data validated both the 4F-solution (Cella et al., 2013) and 3F-solution (Lin et al., 2013). Hence, the French sO-LIFE is psychometrically sound both when excluding and including ImpNon items. Some fit indices of the 4F-solution were nevertheless not acceptable indicating that the 3F-solution is preferable. When comparing the French and English version directly, we observed that configural, metric, but not scalar, invariances were respected, regardless of whether ImpNon items were included or not. These results indicate that the French sO-LIFE retained the psychometric properties of the original 4F- and 3F-solution and that English and French scores are not directly comparable. For these language differences as well as the largely replicated sex differences (e.g. Mason et al., 1995; Cella et al., 2013), we consider those to emerge from "differential item functioning" (i.e. respondents interpret and answer items differently because they belong to a given group: Winterstein, Ackerman et al., 2011; Shevlin et al., 2002) or from defensive responding (Mohr & Leonards, 2005). Minor language and sex differences should thus not be central when validating schizotypy measures.

Regarding the importance of ImpNon to the schizotypy concept, some authors favour its inclusion (Ettinger, Corr, Mofidi, Williams, & Kumari, 2013), while others consider it sensitive to impulsive, borderline and antisocial features (Bouvard & Cosma, 2008) rather than to schizophrenia (Cochrane et al., 2010). Mason et al. (2006) adopt a "unitary view of psychosis" arguing that ImpNon would provide a broader perspective on risk, with relevance for example to bipolar disorder. Our CFAs showed that both factor solutions of the sO-LIFE are psychometrically acceptable. This finding was observed for both language versions. Thus, the controversial debate about the inclusion of ImpNon may continue (e.g. Cella et al., 2013; Lin et al., 2013), but does not seem to hinder schizotypy research. The decision of whether or not to include ImpNon should rely on theoretical grounds (i.e. definition of schizotypy) and research goals (e.g. when also studying drug use or other personality dimensions).

The French sO-LIFE scores correlated in predictable ways with associated (EPQR-A) and similar (SPQ-B) constructs. Our significant correlations between sO-LIFE and EPQR-A scores replicated previous reports (for a summary see Burch et al., 2006), i.e. all sO-LIFE scores correlated positively with Neuroticism scores (e.g. Barrantes-Vidal, Ros-Morente, & Kwapil, 2009; Ross et al., 2002), CogDis and IntAn scores correlated negatively with Extraversion scores (e.g. Mason, et

al., 1995; Ross et al., 2002) and ImpNon scores correlated positively with Psychoticism scores (e.g. Burch et al., 2006; see also Bouvard & Cosma, 2008). Thus, schizotypy is associated with general personality traits (see also Asai et al., 2011). The significant relationships between sO-LIFE and Neuroticism scores yield psychopathological potential, since Neuroticism scores associate with psychopathological vulnerability (Ormel, Rosmalen, & Farmer, 2004), particularly with schizophrenia (e.g. van Os & Jones, 2001; Cochrane et al., 2010). The sO-LIFE scores correlated positively with conceptually corresponding SPQ-B scores (i.e., Cog-Per with UnEx, Dis with CogDis, IntAn with Int). Also, CogDis correlated positively with Int and ImpNon correlated positively with both Cog-Per and Dis. These correlations replicate findings by Asai et al. (2011). Taken together, the correlation results confirm that the sO-LIFE validly measures schizotypy in a fully dimensional perspective (Mason et al., 2006; Nelson et al., 2013).

Study limitations were a reduced representativeness of our sample, i.e. our French-speaking sample data were collected in higher education environments, and only a third were male. Secondly, we performed confirmatory CFAs analyses, whereas other tools (e.g. items response theory) and discussion of potential "differential items functioning" might be more appropriate to further the improvement of current schizotypy scales (Winterstein, Ackerman, et al., 2011). Thirdly, our study validated the French sO-LIFE in a healthy student population, but evidence of validity in clinical population will have to be provided (e.g. Lin et al., 2013; Cochrane et al., 2010).

2.5 CONCLUSION

We report on a psychometric study validating a French sO-LIFE. Our results showed good reliability and validity. For both the 4F-solution (with ImpNon items) and the 3F-solution (without ImpNon items), validity of the French sO-LIFE was supported by (i) preserved configural and metric invariances by comparing the French and English version, (ii) an acceptable to good fit in CFA and (iii) correlations with instruments measuring close or similar constructs. The inclusion of the ImpNon items neither impaired nor improved sO-LIFE's psychometric properties and use of the scale should be decided on grounds of theory and research question. We propose the French sO-LIFE is an adequate tool to be used in French-speaking environments.

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3 VALIDATION OF THE FRENCH AUTISM SPECTRUM QUOTIENT SCALE AND ITS RELATIONSHIPS WITH SCHIZOTYPY AND EYSENCKIAN PERSONALITY TRAITS[14]

Abstract

Background: Autism and schizophrenia spectra were long considered distinct entities. Yet, recent studies emphasized overlapping clinical and personality features suggesting common mechanisms and liabilities. Independent notions, however, highlight that the two spectra oppose each other socially (positive schizotypal hyper-Mentalism versus autistic hypo-Mentalism).

Methods: To clarify these relationships, we used data from 921 French-speaking Swiss undergraduates to firstly validate the French Autism Spectrum Questionnaire (AQ) identifying an optimal factor structure. Secondly, we assessed relationships between this AQ structure and schizotypic personality traits.

Results: Results from correlational and principal component analyses replicated both overlapping and opposing relationships.

Conclusions: We conjecture that autistic traits opposing positive schizotypy represent autistic mentalizing deficits. We discuss implications of our findings relative to theories of autism and schizophrenia spectrum relationships.

Keywords: psychometry; validation; questionnaire; autism; schizotypy; mentalizing

[14] Sierro, G., Rossier, J., & Mohr, C. (2016). Validation of the french Autism Spectrum Quotient scale and its relationships with schizotypy and Eysenckian personality traits. *Comprehensive Psychiatry*, *68*:147–155. doi: http://dx.doi.org/10.1016/j.comppsych.2016.03.011

3.1 INTRODUCTION

Psychiatric conditions are not the exclusive sufferings of acutely ill patients. The liability persists between acute psychiatric episodes in patients and presents in non-affected family members. The systematic communication of these observations dates back to the first half of the last century (Asperger, 1944 [1991]; Bleuler, 1911), leading to the formulation of dimensional models such as for schizophrenia (Claridge, 1997; Meehl, 1990), and more recently autism (Constantino & Todd, 2003). Dimensional models assume that psychopathology can be placed along a continuum with the most severe clinical expression being found at one end (e.g. schizophrenia), milder forms being present in relatives of patients, and the least severe expression being present in individuals from the general population (e.g. schizotypic traits: Claridge, 1997; Kwapil & Barrantes-Vidal, 2015).

In schizophrenia, researchers long assumed that our understanding of the aetiological underpinnings and behavioural correlates at the healthy end of the continuum (schizotypy) can inform on mechanisms relevant to the most severe clinical expression (Claridge, 1997; Meehl, 1990). Schizotypy is commonly assessed using self-report questionnaires (Mason, 2015 for an overview) describing subclinical symptom dimensions known from patients, i.e. "positive" symptoms, "negative" symptoms and disorganized symptoms (Arndt, Alliger, & Andreasen, 1991; Liddle, 1987). In the clinical realm, elevated schizotypic scores associated with worse mental health (Goulding & Ödéhn, 2009) and higher schizophrenia symptoms in at-risk and patient populations (Cochrane et al., 2010; Debbané et al., 2015). In the general population, elevated schizotypy associated with behavioral, cognitive or neural markers that have also been observed in clinical populations along the schizophrenia spectrum (SSp[15]; e.g. Ettinger et al., 2015 for a recent review).

Similarly, autism is part of a spectrum extending from clinical disorders into the general population. Autism spectrum disorders (ASD) is the umbrella term for neurodevelopmental conditions of varying severity (e.g. Kanner's autism, Asperger's syndrome) comprising enduring symptoms affecting early childhood development: (i) impairment in social interaction and communication, and (ii) abnormal and repetitive behavior, interests and activities (American Psychiatric Association, 2013). Although ASD do not cover per se autistic personality manifestations in the general population, recent notions suggest that autistic traits should be included in the ASD (Lai et al., 2013), because of their presence in family members (constituting the broader autism phenotype [BAP], Piven et al., 1997), and the general population (Constantino &

[15] In the present paper, we used SSp instead of PSp, to focus more precisely on schizophrenia spectrum, as accounted by schizotypic traits. However, in the most parts of this thesis manuscript, we extended the broader psychotic spectrum, and used psychotic spectrum (PSp). Given our very definition of schizophrenia spectrum in the Chapter 1 (p. 53, footnote 12), both may be seen as quasi equivalent.

Todd, 2003). Henceforward, we use autism spectrum (ASp) to refer to ASD comprising autistic personality traits in the general population.

As for schizotypy, autistic traits in the general population are assessed using self-report questionnaires. The AQ is the questionnaire of major use (Baron-Cohen et al., 2001), although other instruments exist (e.g. Social Responsiveness Scale; Constantino, Davis et al., 2003). The AQ has acceptable reliability and validity as well as a good sensitivity to detect people diagnosed with ASD (Broadbent, Galic, & Stokes, 2013). Also, the AQ measures the same traits in ASD patients and individuals from the general population (Murray et al., 2014). Higher as compared to lower AQ scores in the general population go along with genetic (Lundström et al., 2012), cognitive, brain anatomical and functional peculiarities comparable to those reported from patients with autism (Sucksmith et al., 2011).

Most of the time, SSp and ASp are considered independent illness spectra. Recent research, however, stresses potential clinical (Konstantareas & Hewitt, 2001) and genetic associations (Crespi & Crofts, 2012). Crespi and Badcock (2008) argued that psychosis and autism are "diametrically opposite" to each other with regard to social cognition (thus, social brain functioning), in particular the two superimposed dimensions of Mentalism and Mechanism. Mentalism refers to a cognitive style characterized by enhanced abilities in perspective taking, mentalizing (i.e. theory of mind, mindreading), and sensitivity to gaze (allowing efficient interpersonal interactions). Mechanism refers to a cognitive style characterized by enhanced abilities in perception, memory and attention to detail (allowing efficient interaction with the physical world). Accordingly, ASp would be characterized by diminished mentalizing (hypo-Mentalism) and increased interactions with the physical world (hyper-Mechanism). SSp, on the other hand, would be characterized by increased positive symptoms (hyper-Mentalism) and diminished interactions with the physical world (hypo-Mechanism).

Independent reports emphasized that ASp and SSp symptoms *overlap* suggesting common mechanisms and liabilities (Konstantareas & Hewitt, 2001), here, (i) autistic social skills/interest deficits associated with elevated negative schizotypy, and (ii) autistic attention to detail associated with positive schizotypy (Hurst, Nelson-Gray et al., 2007; Russell-Smith et al., 2011). These psychometric observations emphasize *shared* rather than *diametrically opposite* features for ASp and SSp, although some studies found these opposite features (e.g. Hurst, Nelson-Gray et al., 2007; Russell-Smith et al., 2011). Most recently, Dinsdale, et al. (2013) and Del Giudice, Klimczuk, Traficonte and Maestripieri (2014) replicated both *overlapping* and diametrically *opposing* deficits between autistic and schizotypic traits. In particular, Dinsdale et al. (2013) reported that elevated positive schizotypy associated with reduced autistic expressions of some autistic dimensions (Social

Skill and Imagination deficits). These relationships are further supported by cross-cultural data, because Dinsdale et al. (2013) reported comparable results from a Canadian and Japanese sample. The controversial debate on overlapping versus opposing deficits in ASp and SSp is ongoing, and its universality needs to be tested. To do so, we need validated self-report questionnaires in different languages.

In our French-speaking environment, we can assess schizotypy using the validated French sO-LIFE (Sierro, Rossier, Mason et al., 2016). Regarding autistic traits, the AQ (Baron-Cohen et al., 2001) seems the scale of choice, because of its wide use and French translation (Sonié et al., 2011). The French AQ translation needs to be validated using conventional confirmatory factor analysis (CFA). Accordingly, our study had two major goals. Firstly, we validated the French AQ (Sonié et al., 2011) using standard CFAs. The original 5-factor structure by Baron-Cohen et al. (2001) has been questioned (e.g. Hurst, Mitchell, et al., 2007; Lau, Kelly et al., 2013). Thus, we investigated which previously published 5-factor structure best fits our French-speaking data (Baron-Cohen et al., 2001; Hoekstra et al., 2011; Kloosterman et al., 2011; Lau, Kelly et al., 2013). Secondly, we used correlations and principal component analysis (PCA) to replicate previously reported overlapping and opposing relationships between autistic and schizotypic traits (Del Giudice et al., 2014; Dinsdale et al., 2013). For further validation purposes, we correlated our AQ and sO-LIFE scores with scores from a broader personality model (Abridged and Revised Eysenck Personality Questionnaire, EPQR-A ; Bouvard et al, 2010).

3.2 METHODS

3.2.1 PARTICIPANTS

Our population of 921 students (665 females) had a mean (± SD) age (in years) of 22.2 (± 2.46, range 18 to 30). Most students were undergraduate psychology students taking part for course credit.

3.2.2 SELF-REPORT INSTRUMENTS

3.2.2.1 THE AUTISM SPECTRUM QUOTIENT (AQ; BARON-COHEN ET AL., 2001)

The original 50-item AQ (24 reverse formulated items) assesses autistic traits along 5 dimensions. Ten items each belong to the 5 dimensions of Social Skill deficits (SocSki, α=.77, e.g.

“I find it hard to make new friends.”), Communication deficits (Comm, α=.65, e.g. “I am often the last to understand the point of a joke.” when someone is talking to me.”), Attention Switching deficits (AttSwi, α=.67, e.g. “I prefer to do things the same way over and over again.”), Imagination deficits (Ima, α=.65, e.g. “I don’t particularly enjoy reading fiction.”), and Attention to Details (AttDet, α=.63, e.g. “I tend to notice details that others do not.”). Respondents indicate their agreement with each item's description using a 4-point Likert scale (“definitely agree”, “agree”, “don't agree”, “definitely disagree”). Dimension scores consist in summing up the responses to all items of a given dimension, so that higher scores reflect higher autistic traits. In line with most recent studies (e.g. Lau, Kelly et al., 2013), we did not use the traditional yes-no response dichotomization (Baron-Cohen et al., 2001). We computed our dimension scores using the 4-point Likert scale, so that “definitely agree”, “agree”, “don't agree” or “definitely disagree” response would respectively weigh 4, 3, 2 or 1 point(s) (the opposite for reverse formulated items, 1, 2, 3 or 4 points). The French translation and descriptive values can be found in Sonié et al. (2011).

3.2.2.2 THE SHORT OXFORD-LIVERPOOL INVENTORY OF FEELINGS AND EXPERIENCES (SO-LIFE; MASON ET AL., 2005)

The 43-item sO-LIFE assesses schizotypy along 4 dimensions comprising positive schizotypy (Unusual Experiences [UnEx], α=.80, e.g. “Are your thoughts sometimes so strong that you can almost hear them?”), negative schizotypy (Introvertive Anhedonia [IntAn], α=.62, e.g. “Do you prefer watching television to going out with people?”), Cognitive Disorganisation (CogDis, α=.77, e.g. “Are you easily confused if too much happens at the same time?”) and Impulsive Nonconformity (ImpNon, α=63, e.g. “Do you at times have an urge to do something harmful or shocking?”). The latter is not a classical schizotypic dimension, and measures impulsive, anti-social, and eccentric behaviours. Respondents indicate their agreement with each item's description responding “yes” or “no”. Positive answers (for reverse coded items, negative answers) weigh one point. For each dimension, scores are computed by summing these points, so that higher scores denote higher schizotypy. The validation of the French sO-LIFE and descriptive values can be found in Sierro, Rossier, Mason et al. (2016).

3.2.2.3 THE ABRIDGED AND REVISED EYSENCK PERSONALITY QUESTIONNAIRE (EPQR-A; FRANCIS ET AL., 1992)

The 24-item EPQR-A (10 reverse formulated items) assesses Eysenck's personality dimensions along 4 dimensions comprising 6 Extraversion items (E, α=.74-.84, e.g. “Are you a talkative person?”), 6 Neuroticism items (N, α=.70-.77, e.g. “Does your mood often go up and

down?"), 6 Psychoticism items (P, α=.33-.52, e.g. "Do you prefer to go your own way rather than act by the rules?"), and 6 Lie items (Lie, α=.59-.65, e.g. "Have you ever blamed someone for doing something you knew was really your fault?") measuring social desirability. Participants make true-false statements. The number of positive answers (negative responses are reversely coded) is summed so that higher scores indicate higher personality traits (or a higher likelihood of lying). Bouvard et al. (2010) validated the French version (normative values: Bouvard & Cosma, 2008).

3.2.3 PROCEDURE

Participants completed questionnaires online. Of the 921 students completing the AQ and sO-LIFE, 768 (575 women) also completed the EPQR-A. Informed consent was obtained from all participants. Before filling in their demographic information, followed by the personality questionnaires, participants had indicated on the webpage that they agree to participate in the study, that they accept that their data is used anonymously, for non-commercial research purposes. It was stressed that participation was not compulsory and that consent and participation could be withhold at any time, without negative consequences. These and additional questionnaires (not reported here) took about 45 minutes to complete. The study was conducted in accordance with the guidelines of the declaration of Helsinki (World Medical Organization, 2013).

3.2.4 STATISTICAL ANALYSES

To investigate the factor structure of the AQ, we performed CFAs on four published 5-dimension AQ models (Baron-Cohen et al., 2001; Hoekstra et al. 2011; Kloosterman et al., 2011; Lau, Kelly et al. 2013), because this number of dimensions has been established by parallel analyses (e.g. Kloosterman et al., 2011) and CFAs (e.g. Lau, Kelly et al., 2013). These models group items into partially different latent dimensions, with some items being completely omitted, and others exchanged between dimensions.

Before performing these CFAs, assumptions were tested and criteria were applied (Brown, 2006). Results were acceptable for Bartlett's test of sphericity ($p < .0001$; acceptable if significant), good for Keyser-Meyer-Olkin (KMO = .80; acceptable >.70) and acceptable for determinent ($>10^{-5}$; values are acceptable above 10^{-5}). The multinormality assumption was not met (skew $p < .001$; kurtosis $p < .001$), so that robust estimates were chosen and reported for all CFAs (weighted least squares, WLSMV estimator). For each model, we computed fit indices measuring the extent to

which a given theoretical model explained the observed data. The absolute fit of each model was assessed by the ratio between χ^2 and degrees of freedom (χ^2/df), acceptable when < 3, the RMSEA (Root Mean Squares Error of Approximation) indicating a close fit when ≤ 0.05, and a reasonable one when ≤ 0.08, and the SRMR (Standardized Root Mean Residual) indicating a good fit when ≤ 0.08. To compare the different models, comparative fit indices were assessed using the Comparative Fit Index (CFI) as well as the Tucker Lewis Index (TLI). Both indices show a close fit and acceptable comparative fit when ≥ 0.90, and a good comparative fit when ≥ 0.95. For all CFAs, error terms were allowed to covariate following the recommendations of Saris, Satorra and van der Veld (2009). This procedure resulted in adjusted models (whenever modification indices were > 50), among which a maximum of 5 error terms were allowed to covariate. Table 5 shows the results of CFAs.

Descriptive analyses and gender differences are reported and for the best fitting model (Kloosterman et al., 2011). We termed this model AQ-K. We compared means between sexes using two-tailed independent *t*-tests (reporting Cohen's *d* effect size; AQ-K: Table 6). We assumed the data to be normally distributed when skew and kurtosis were comprised between -1 and 1 (Kline, 2005). Cronbach's α internal consistency measures were considered minimally acceptable when > .65, acceptable when > .70, and optimal when > .80 (Nunnally & Bernstein, 1994).

To test the interrelationships between questionnaire scores, we computed zero-order Pearson correlations between AQ dimensions, sO-LIFE dimensions and EPQR-A dimensions (Table 7). We considered correlation coefficients small when > .10, moderate when > .30 and large when > .50 (Cohen, 1988).

In line with Dinsdale et al. (2013), we performed PCAs on AQ-K and sO-LIFE dimensions (Table 8). We aimed to replicate their PC structure, which consisted in two principal components (PCs). Following Brown (2006), we present our PCAs with promax rotations. The PCA results were easy to interpret. We found a first PC labelling it “Shared Features” (PCSF-K). Here, “negative” autistic traits and negative schizotypy load in the same direction. We found a second PC representing “Diametrical Features” (PCDF-K). Here, specific autistic features load in the opposite direction to positive schizotypy. The PCDF is theoretically most relevant to our study.

To account for multiple testing, we set the α-level at .05 for *t*-tests and correlations, but interpret significant results only when *p*'s<.001 (see also Russell-Smith et al., 2011). We used R software (R Core Team, 2014) with the package “lavaan” (Rosseel, 2012) for CFAs models.

3.3 RESULTS

3.3.1 PARTICIPANTS

Women (21.89±2.38) were younger than men (22.88±2.53) ($t(919)=-5.54$, $p < .001$; $d=-0.41$). Because the difference is only about 1 year, we do not consider this difference further.

3.3.2 ASSESSING THE FACTOR STRUCTURE OF THE FRENCH AQ USING CFA

The CFAs on the published 5-factor AQ models (Baron-Cohen et al., 2001; Hoekstra et al. 2011; Kloosterman et al., 2011; Lau, Kelly et al. 2013) showed that the AQ-K (Kloosterman et al., 2011) best fitted our French data (Table 5). This model had the highest CFI (>.800) as compared to the other models, together with acceptable χ^2/df, RMSEA and SRMR. The original AQ model (Baron-Cohen et al., 2001) showed insufficient fit. Lau, Kelly et al's (2013) model showed acceptable χ^2/df, RMSEA and SRMR, but unacceptable CFI. The TLI too was below acceptable threshold and lower as compared to those obtained with the AQ-K. Thus, AQ-K best explained our data. We focus subsequent analyses on AQ-K scores.

3.3.3 DESCRIPTIVE VALUES FOR THE AQ-K

The AQ-K consists of 28 items belonging to 5 dimensions: Social Skills deficits (SocSki) Communication/Mindreading deficits (ComMind), Attention to Details (AttDet), Imagination deficits (Ima), and Restricted/Repetitive Behaviour (RRBeh). In Table 6, we report descriptive values, statistics and internal consistency computations. Apart from the AQ-K Total score, skew and kurtosis values were below 1, indicating normal distributions. Cronbach's α values ranged from acceptable to unacceptable. Men scored significantly higher than women in AttDet and Ima, but were comparable to women for the other dimensions (Table 6).

Table 5. Summary of the Confirmatory Factor Analyses goodness of fit statistics for previously published AQ models. Results are based on data from 921 participants.

N Factors	Study	Items	S-B χ^2	df	χ^2/df	RMSEA	SRMR	CFI	TLI
5	Baron-Cohen et al. (2001)	50	4254.485*	1165	3.65	.054	.078	.460	.430
5	Lau, Kelly et al. 2013	34	1765.047*	687	2.57	.041	.057	.773	.755
5	Kloostermann et al., 2011	28	948.259*	338	2.81	.044	.053	.807	.784
5	Hoekstra et al. 2011	28	1277.937*	345	3.70	.054	.065	.687	.657
1	none	50	5244.856*	1175	4.46	.061	.089	.286	.255

* $p < .001$
S-B χ^2: Satorra-Bentler chi-square; df: degrees of freedom; RMSEA: Root Mean Squares Error of Approximation; SRMR: Standardized Root Mean Residual; CFI: Comparative Fit Index; TLI: Tucker-Lewis Index

3.3.4 RELATIONSHIPS BETWEEN AQ-K, SO-LIFE AND EPQR-A

For correlations between sO-LIFE and AQ-K (Table 7), we found significant positive correlations between SocSki and IntAn (large), AttDet and UnEx (moderate), and AttDet and ImpNon (small). Importantly, we found small *negative* correlations between UnEx and both ComMind and Ima, and between ComMind and ImpNon. Correlations between the EPQR-A and AQ-K (Table 7) showed small positive correlations of N with SocSki, RRBeh and AttDet, respectively. E showed a large negative correlation with SocSki, small negative correlations with ComMind and Ima, respectively, and a small positive correlation with RRBeh. P showed a small positive correlation with AttDet and SocSki, and a small negative correlation with ComMind.

Table 6. Descriptive values for the AQ-K model (Kloosterman et al., 2011). Results are based on data from 921 participants.

		Women			Men			Total							
Nb items	Scores	α	Mean	Sd	α	Mean	Sd	α	Mean	Sd	*t*	*p*	*d*	*S*	*K*
			n = 665			*n* = 256			*n* = 921			*df* = 919			
28	TOTAL	.66	56.98	6.96	.68	59.55	7.31	.67	57.69	7.15	-4.95	<.001	-0.36	0.34	0.77
8	SocSki	.79	15.98	3.83	.80	16.53	4.10	.79	16.13	3.91	-1.93	.054	-0.14	0.64	0.65
5	ComMind	.66	10.06	2.35	.68	10.2	2.54	.67	10.10	2.40	-0.77	.439	-0.06	-0.08	-0.23
5	AttDet	.66	11.18	3.06	.63	12.05	2.96	.66	11.43	3.06	-3.88	<.001	-0.29	0.23	-0.33
5	Ima	.50	8.65	2.34	.42	9.63	2.28	.49	8.92	2.36	-5.75	<.001	-0.42	0.41	-0.09
5	RRBeh	.37	11.11	2.14	.30	11.14	2.12	.35	11.12	2.14	-0.18	.857	-0.01	0.26	0.23

α: Cronbach's Alpha; *t*: Student's *t* value; *p*: Student's t-test p-value; *d*: Cohen's *d*; *S*: skew; *K*: kurtosis

Total: AQ.K total score; SocSki: Social Skills deficits; ComMind: Communication/Mindreading deficits; AttDet: Attention to Details; Ima: Imagination deficits; RRBeh: Routines/Repetitive Behaviours

Table 7. Zero-order Pearson correlations between the subscale scores of the (A) sO-LIFE, (B) Kloosterman et al. (2011)'s AQ-K, and the (C) EPQR-A. Results are based on data from 921 participants.

	Dimension	A1	A2	A3	A4	A5	B1	B2	B3	B4	B5	B6	C1	C2	C3
A1	Total	–													
A2	UnEx	.761**	–												
A3	CogDis	.794**	.426**	–											
A4	IntAn	.437**	.078*	.221**	–										
A5	ImpNon	.693**	.455**	.365**	.109**	–									
B1	Total	.419**	.199**	.355**	.479**	.158**	–								
B2	SocSki	.304**	.074*	.247**	.556**	.041*	.723**	–							
B3	ComMind	-.073*	-.214**	.103**	.038	-.133**	.384**	.170**	–						
B4	Ima	-.008	-.131**	.026	.159**	-.029	.475**	.132**	.244**	–					
B5	RRBeh	.333**	.278**	.258**	.144**	.210**	.431**	.045	.002	.065*	–				
B6	AttDet	.421**	.446**	.233**	.154**	.297**	.443**	.143**	-.295**	-.070*	.198**	–			
C1	N†	.566**	.325**	.560**	.250**	.359**	.264**	.247**	-.013	.025	.206**	.154**	–		
C2	E†	-.159**	.055	-.234**	-.390**	.099	-.401**	-.598**	-.163**	-.138**	.125**	-.035	-.168**	–	
C3	P†	.203**	.156**	.060	.056	.308**	.089*	.088*	-.125**	-.003	-.008	.203**	.007	.020	–
C4	Lie†	-.243**	-.205**	-.110*	-.005	-.346**	-.009	.097*	.017	.009	-.118*	-.082*	-.047	-.144**	-.118*

* $p < .05$; ** $p < .001$; † $n = 769$

Total: sO-LIFE Total score; UnEx: Unusual Experiences; CogDis: Cognitive Disorganization; IntAn: Introvertive Anhedonia; ImpNon: Impulsive Nonconformity
Total: AQ.K total score; SocSki: Social Skills deficits; ComMind: Communication/Mindreading deficits; AttDet: Attention to Details; Ima: Imagination deficits; RRBeh: Routines/Repetitive Behaviours

3.3.5 PCA FOR THE AQ-K AND sO-LIFE SCORES

The PCA on AQ-K (Table 8) showed the two PCs, the PCDF-K explained 27% of the variance and the PCSF-K explained 20% of the variance. The factor loadings for PCSF-K were all positive (apart from negligible ones for UnEx and AttDet). The factors loadings were highest for IntAn and SocSki (>.70), intermediate for ComMind and Ima (>.50), and lowest for CogDis (>.30). The factor loadings for PCDF-K were mostly positive, with the notable exceptions of ComMind and Ima. Positive factor loadings were highest for UnEx and AttDet (>.70), intermediate for ImpNon and CogDis (>.50), and lowest for RRBeh (>.30). Crucially, the factor loadings were negative for ComMind (-.41) and Ima (-.23). Thus, they loaded in opposite directions to UnEx, AttDet, ImpNon, CogDis and RRBeh.

Table 8. Principal Component Analysis outcome accounting for the subscale scores of the sO-LIFE and AQ-K. The PCA was conducted using promax rotations. Results are based on data from 921 participants.

	Promax Rotation	
	PCDF-K	PCSF-K
Explained Variance	27%	20%
UnEx	**.81**	-.07
CogDis	**.57**	.36
IntAn	.19	**.71**
ImpNon	**.69**	.00
AttDet	**.70**	-.04
ComMind	**-.41**	**.54**
Ima	-.23	**.52**
RRBeh	**.45**	.18
SocSki	.12	**.76**

Loadings less than -.40 and more than .40 are in boldface.
UnEx: Unusual Experiences; CogDis: Cognitive Disorganization; IntAn: Introvertive Anhedonia; ImpNon: Impulsive Nonconformity
SocSki: Social Skills deficits; AttDet: Attention to Details; ComMind: Communication/Mindreading deficits; Ima: Imagination deficits; RRBeh: Routines/Repetitive Behaviours

3.4 DISCUSSION

Dimensional models of psychopathology have received increasing interest over the last decades, for both the SSp (e.g. Kwapil & Barrantes-Vidal, 2015) and the ASp (e.g. Lai et al., 2013). For research in the general population, the personality traits relevant to the pathological conditions are commonly assessed using self-report questionnaires (e.g. Russell-Smith et al., 2011). Given their wide use, it is key that these self-report questionnaires are reliable and valid, irrespectively of culture and language (Lau, Gau et al., 2013). Therefore, we present psychometric properties of the French AQ (Sonié et al., 2011), and investigate its relationships with the validated French sO-LIFE (Sierro, Rossier, Mason et al., 2016). We collected data on the French versions of AQ (Sonié et al., 2011), sO-LIFE (Sierro, Rossier, Mason et al., 2016) and EPQR-A (Bouvard et al., 2010) at a French-speaking Swiss university. We firstly validated the French AQ using common psychometric test procedures (e.g. CFA). Among the four 5-factor models we compared, the best model was the 28-item AQ-K model (Kloosterman et al., 2011). Hence, we focused subsequent analyses on the AQ-K.

The correlations of AQ-K scores with sO-LIFE and EPQR-A, respectively, corresponded to previous reports (e.g. Kanai et al., 2011; Russell-Smith et al., 2011). Crucially, we replicated significant negative correlations between positive schizotypy and autistic traits using AQ-K's Ima and ComMind (Dinsdale et al., 2013; Hurst, Nelson-Gray et al., 2007; Russell-Smith et al., 2011). Finally, PCAs between AQ scores and sO-LIFE scores replicated previous results (Del Giudice et al., 2014; Dinsdale et al., 2013), showing (i) a PC characterized by mostly positive loadings for AQ dimensions and schizotypic dimensions (PCSF-K), and (ii) another PC characterized by positive loadings for positive schizotypy and negative ones for Ima and ComMind (PCDF-K). We discuss these findings in turn.

3.4.1 VALIDATION OF THE FRENCH AQ

CFAs on our French AQ data showed that the AQ-K model (Kloosterman et al., 2011) best fitted our data. This observation is not surprising, because suboptimal fit indices have been reported by previous AQ validations, and alternative factor structures (Dutch: Kloosterman et al., 2011; Hoekstra et al., 2011). A recent factor solution on AQ data showed adequate goodness of fit results (English: Lau, Kelly et al., 2013). In our study, the factor solution by Lau, Kelly et al. (2013) had the second best fit. This solution is close to the AQ-K (yet, it does not include Ima). Similarly, internal consistencies have been low for most AQ dimensions, apart from good internal

consistencies for SocSki and ComMind (e.g Hurst Mitchell et al., 2007; Kloosterman et al., 2011; Lau, Kelly et al., 2013). We too found internal consistencies that ranged from acceptable (SocSki) to very low (RRBeh and Ima), and in-between, minimally acceptable for ComMind and AttDet. Disappointing internal consistencies might result from small item numbers (Cortina, 1993), such as for AQ-K Ima (5 items).

Correlations between Eysenck's personality dimensions (E, N, P) and AQ-K dimensions were largely comparable to those reported in a previous study (Kanai et al., 2011[16]). For instance, our study showed that higher N went along with globally higher autistic traits (AttDet, RRBeh, and SocSki). Also, higher E went along with lower autistic social deficits (ComMind, Ima, and SocSki). Finally, higher P went along with higher AttDet. Importantly, AQ relationships with Eysenck's personality dimensions are very similar to those between sO-LIFE Eysenck personality dimensions (see also e.g. Burch et al., 2006; Sierro, Rossier, Mason et al., 2016). This observation suggests that autistic and schizotypic profiles may not be distinguishable using short instruments representing a broader personality.

3.4.2 RELATIONSHIPS BETWEEN AQ-K AND sO-LIFE

The results from the second part of our analysis showed that the relationships between AQ and sO-LIFE resemble results from previous reports (e.g. Hurst, Nelson-Gray et al., 2007), irrespectively of questionnaires and factor solutions. We replicated findings that showed shared and diametrically opposite features (e.g. Del Giudice et al., 2014; Dinsdale et al., 2013).

With regard to shared features, enhanced negative schizotypic features correlated with enhanced autistic social deficits (IntAn–SocSki; see also USA sample: Hurst, Nelson-Gray et al., 2007; Australian sample: Russell-Smith et al., 2011). Descriptively, these correlations and shared traits correspond to a schizoid phenotype in the general population (Ford & Crewther, 2014). The underlying causes of this overlap remain unclear. This overlap may stem from vague item formulations and confounded phenotypes (Del Giudice, Angeleri, Brizio, & Elena, 2010). In contrast, this overlap may reflect common psychopathological and genetic mechanisms (Craddock & Owen, 2011; Rausch & Johnson, 2008). Similarly, we replicated relationships between positive schizotypy (UnEx; including ImpNon) and AttDet (UnEx–AttDet, ImpNon–AttDet; Hurst, Nelson-Gray et al., 2007; Russell-Smith et al., 2011). Again, this overlap may reflect a genuine shared

[16] Austin (2005) and Wakabayashi et al. (2006a) also reported correlations between Five Factor Model's NEO-PI-R and AQ autistic traits.

phenotype (e.g. bias for unusual information / interests), or confounded phenotypes. As for the latter solution, both AttDet and UnEx may capture *apparently similar* interest in unusual features resulting from *opposite* hyper-Mechanistic or hyper-Mentalistic cognitive styles (e.g. respectively, a *mathematical* versus a *numerological* interest for numbers; see also discussion further below). Our data cannot prove that "positive" and "negative" features apply to both ASp and SSp (are interchangeable). Recent evidence, however, stresses the transdiagnostic presence of "negative" features in ASp, SSp and other disorders (Malaspina et al., 2014), as it might be the case for "positive" ones such as delusions (Bebbington & Freeman, 2017).

With regard to diametrically opposite features, we replicated that enhanced positive schizotypy correlated with AQ-K's dimensions (ComMind–UnEx; ComMind–ImpNon). Because these negative correlations are relatively weak, various authors have given their existence little or no weight. Some argued against the idea of Crespi and Badcock (2008) that ASp and SSp would lie on opposite ends of a Mentalism continuum (Russell-Smith et al., 2011). Dinsdale et al. (2013, p. 8), on the other hand, argued for an "autism-schizotypy axis [reflecting] a diametrical pattern between autistic features and positive aspects of schizotypy". The consistency with which these negative relationships occur across studies (including ours) suggests to us that they reflect diametrically opposite features between ASp and SSp.

We found that enhanced positive schizotypy features (UnEx) correlated with low AQ-K ComMind. The positive schizotypic features likely reflect hyper-Mentalism tendencies (e.g. paranoid ideations; Brosnan et al., 2010; Fyfe et al., 2008). The autistic dimensions likely reflect hypo-Mentalistic tendencies. ComMind would reflect impaired mindreading, i.e. inability to draw inferences about other persons' thoughts (Baron-Cohen, 2002). Ima would reflect deficits in representing social situations, such as story/character comprehension, and pretend play (Ten Eycke & Müller, 2015). Bishop et al. (2004) have already proposed that an "empathizing" construct was underlying AQ social deficit dimensions (SocSki, Comm, Ima) characterizing ASD *and* the BAP. Thus, positive schizotypy (including ImpNon) and autistic mentalizing deficits seem ideal candidates to describe diametrically opposite traits between SSp and ASp (Dinsdale et al., 2013).

In sum, we suggest that our and previous oppositions between positive schizotypy and autistic mentalizing deficits represent the Mentalism continuum that distinguishes SSp from ASp (Crespi & Badcock, 2008). Thus, autistic mentalizing deficits (ComMind, Ima) represent the autistic *hypo-Mentalistic* end of the Mentalism continuum, whereas positive schizotypy (UnEx, ImpNon) represents its *hyper-Mentalistic* end (Badcock, 2004). Our and independent PCA results (Dinsdale et al., 2013) support the existence of both shared (PCSF) and diametrical (PCDF scores) dimensions. Hence, current models of autism-psychosis relationships are insufficient and alternative

ones should integrate both shared and diametrical features (Chisholm et al., 2015 for a review). Also, the distinction between hypo- and hyper-Mentalistic tendencies may help searching for *specific* ASp and SSp endophenotypes (Ford & Crewther, 2014; Hasler et al., 2006).

3.4.3 STUDY LIMITATIONS AND CHALLENGES

As most previous studies, we validated the AQ using student populations, although validation in the wider population is preferable. As noted above, the psychometric properties of the current AQ-K and most previous AQ validations are not entirely satisfactory with regard to validity and reliability. Future studies should thus continue improving the psychometric properties (and likely also item content) of the current AQ (e.g. Lau, Kelly, et al., 2013) including this French version.

As for schizotypic traits, most previous studies used the Schizotypal Personality Questionnaire (SPQ; Raine, 1991) rather than the sO-LIFE used here. In general, we do not think that the type of questionnaire undermines our conclusions. In previous studies, the SPQ (or its brief version) and O-LIFE (or its brief version) were shown to measure similar constructs (Japanese population: Asai et al., 2011; French-speaking Swiss population: Sierro, Rossier, Mason et al., 2016). Also, several studies reported both overlapping and diametrical relationships between autistic and schizotypic traits irrespectively of the schizotypy questionnaire used (e.g. Brosnan et al., 2010; Dinsdale et al., 2013; the present study).

We observed additional results worth further discussion (e.g. the role of ImpNon, and relationships that cannot be explained by the Mentalism argument). The inclusion of ImpNon to schizotypy is questioned (Cochrane et al., 2010). Yet, ImpNon associated negatively with ComMind in our data, as it did with Ima in a previous study (Russell-Smith et al., 2011). Instead of representing a schizotypic dimension, ImpNon might be sensitive to hypomania, because both share the same liability (Claridge & Blakey, 2009). Mania, indeed, associated with over-mentalizing (Brosnan et al., 2010), potentially explaining our negative ImpNon–ComMind relationship. Likewise, Del Giudice et al. (2014) showed that impulsivity and sensation seeking associated with the schizotypy-autism continuum. These previous results demonstrate that not only schizophrenia-related positive schizotypy is relevant when considering ASp–SSp relationships, but also broader psychosis-related traits such as impulsivity or sensation seeking.

Importantly, we would like to note that both we and Dinsdale et al. (2013) found results supporting the model by Crespi and Badcock (2008), but only partially. So far, findings favor a model in which ASp–SSp can be placed on opposite ends of a Mentalism continuum, but not on opposite ends of a Mechanism continuum (but see Brosnan et al., 2010). We give a pertinent

example on AttDet. AttDet can be assumed to reflect hyper-mechanistic and systemizing tendencies (Baron-Cohen, 2002). In this case, AttDet should correlate negatively with hyper-mentalistic tendencies (e.g. UnEx; see also Brosnan et al., 2010; Crespi & Badcock, 2008), but not positively with UnEx (current study; Dinsdale et al., 2013; Russell-Smith et al., 2011). AttDet's loose item formulation (Del Giudice et al., 2010) might confound local biases in autistic tendencies (Happé & Frith, 2006) and/or deviant sensory experiences (Leekam, Nieto, Libby, Wing, & Gould, 2007) with perceptual and unusual experiences known from positive schizotypy (Mohr & Claridge, 2015). Indeed, Garnett, Attwood, Peterson and Kelly (2013) distinguished autistic dimensions accounting for (i) a "fact-oriented" cognitive style, conceptually close from hyper-Mechanism, and (ii) a more or less marked "sensory sensitivity" to details from various perceptual modalities. Improvements to the AQ should account for such possible confounds, indicating whether AttDet and RRBeh measure hyper-Mentalism, hyper-Mechanism, both or none.

Finally, the psychometric measurement of the diametrical model remains a challenging task. We make some suggestions for its improvement: (i) systematic validation of the instruments used (e.g. with CFA, item response theory), (ii) cross-cultural and cross-vocational assessments (e.g. engineering vs humanities), (iii) adding personality items accounting for hyper-Mentalism (e.g. mentalizing-related positive schizotypic traits: Fyfe et al., 2008; manic, impulsive and sensation seeking traits: Brosnan et al., 2010, Del Giudice et al. 2014; paranormal beliefs: Lindeman, Svedholm-Häkkinen, & Lipsanen, 2015), (iv) adding items accounting for hypo-Mentalism (e.g. lack of cognitive empathy or Empathizing Quotient: Brosnan et al., 2010; lack of social imagination and fantasy), (v) adding items accounting for hypo- and hyper-Mechanism (e.g. Systemizing: Brosnan et al., 2010), and (vi) joint use of instruments measuring the related construct of cognitive styles, such as Systemizing and Empathizing: Brosnan et al., 2010; Lindeman et al., 2015). Likely then, future studies can distinguish ASp and SSp on a Mechanism continuum too (Crespi & Badcock, 2008).

3.5 CONCLUSION

Personality questionnaires such as the AQ and sO-LIFE are promising instruments to better understand ASp and SSp. We validated the French AQ and assessed its relationships with the French sO-LIFE. Our data best fitted the AQ-K, the 28-item 5-factor structure by Kloosterman et al. (2011). We replicated (i) the overlapping relationships between autistic and schizotypic "positive" and "negative" traits, but also (ii) the opposing relationships between "positive" schizotypic and autistic traits AQ-K's Ima and ComMind (Del Giudice et al., 2014; Dinsdale et al., 2013). This result

suggested that “diametrically opposite” autistic traits represent autistic mentalizing deficits. We consider that these opposing relationships support the notion by Crespi and Badcock (2008) that ASp and SSp represent diametrical ends of a Mentalism continuum, such that *“social cognition [would be] underdeveloped in autistic-spectrum conditions and hyper-developed on the psychotic spectrum”* (Crespi & Badcock, 2008, p. 241). This suggestion implies that research on ASp–SSp would gain taking into account not only overlapping but also opposing features, in that Mentalism might differentiate both spectra.

4 PAREIDOLIA-PRONENESS AND MENTALISM[17]

Abstract:

Autism (ASD) and Schizophrenia (SSD) Spectrum Disorders are separate diagnostic entities. Yet, they share clinical and behavioural features (e.g. social withdrawal). Recently, psychometric studies confirmed both shared and opposing relationships between autistic and schizotypic traits [Dinsdale et al., 2013]. These studies offered some support for an opposition between ASD *hypo-functioning* social cognition (e.g. ToM deficits) and SSD *hyper-functioning* one (e.g. thoughts of reference, paranoia) [Crespi & Badcock, 2008]. Yet, are these opposite psychometric relationships mirrored by behavioural ones in a social cognition task? In three subsequent studies, we investigated whether shared and opposing psychometric relationships would associate with behavioural liability to perceive illusory faces in ambiguous stimuli (i.e. face pareidolia). We predicted that pareidolia-proneness would associate with *increased* positive schizotypy and *decreased* autistic mentalizing deficits. Pareidolia-proneness was assessed in an old-new recognition paradigm of upright and inverted faces, FLOs, and object stimuli. The performance advantage of upright over inverted stimuli indicated the extent to which FLOs were processed like faces, i.e. the pareidolia effect. Our results did not show the expected relationships with personality traits, and cast doubt on our behavioural measurement of pareidolia-proneness. However, positive schizotypy associated with impaired configural processing for stimuli with a face-like configuration (faces, FLOs), while autistic mentalizing deficits associated with improved object processing. Instead of an opposition relative to pareidolia-proneness, our results suggest distinct phenotypes pertaining to basic face and object processing. We discuss underlying implications for ASD-SSD relationship theories, cognitive mechanisms, limitations, and future perspectives.

Keywords: autism; schizotypy; personality; face processing; pareidolia

[17] Sierro, G., Fioravera, A., Ouali, M., & Mohr, C. Impaired configural performance in undergraduate students with schizotypy but not autistic personality traits: Face-like objects reveal distinct performances. *In preparation.*

4.1 INTRODUCTION

Schizophrenia Spectrum Disorders (SSD) and Autism Spectrum Disorders (ASD) both comprise deficits in social interactions and skills (*DSM-5*, APA, 2013). The negative symptoms in SSD (e.g. anhedonia, social withdrawal) actually resemble social interaction and communication deficits in ASD, to the point of overlap and confusions (Hallerbäck et al., 2012; Nylander & Gillberg, 2001; Perlman, 2000). If one considers that ASD and SSD share a common history, these difficulties are hardly surprising. In his seminal description of ASD, Kanner (1943) used the term "autism" to characterize the condition of children who were unable to relate to others, by analogy to schizophrenic "autism" described by Bleuler (1911). Until the 70^{ies}, infantile autism was considered to represent a subset of psychosis (*DSM-III*, APA, 1980). It was only subsequently that ASD and SSD were considered separate diagnostic entities, mainly because of their different ages of onset (Kolvin, 1971; Rutter, 1972). Somehow reversing this separation, current debates emphasize phenomenological similarities between ASD and SSD as possibly reflecting common mechanisms if not vulnerabilities (King & Lord, 2011; Rausch & Johnson, 2008). Independent current debates consider, on the other hand, that ASD and SSD lie on opposite ends of a shared social function deficit continuum (Crespi & Badcock, 2008). These social function deficits originate at one end from hypo-active mentalizing tendencies (hypo-Mentalism; e.g. ToM deficits) in ASD and at the other end from hyper-active mentalizing tendencies (hyper-Mentalism; e.g. paranoia, delusions) seen in positive symptoms in SSD (Abu-Akel & Bailey, 2000; Badcock, 2004).

Crespi and Badcock (2008) proposed to test opposite social function deficits in ASD and SSD using the healthy ends of the spectra, namely autistic as well as schizotypic personality traits. Thus, these authors adopted a dimensional perspective connecting psychopathology and personality (Claridge, 1997). Dimensional models in both ASD and SSD are based on the assumption that disorders are extreme expressions of healthy variants of phenotypes. Symptoms present in the clinical population are qualitatively similar though quantitatively milder in the healthy population. In the case of SSD, schizotypy might actually reflect SSD vulnerability in the general population (e.g. Meehl, 1990). Similarly, autistic personality traits would reflect ASD vulnerability in unaffected relatives (i.e. BAP), and by extension also in the general population (e.g. Bailey et al., 1998; Constantino & Todd, 2003; Lai et al., 2013). Thus, the dimensional perspective assumes that we can learn about the clinical expression from the functioning and dysfunctioning in the healthy expression. Meanwhile, we profit from the opportunity to test populations not suffering from secondary side-effects (e.g. chronic medication) potentially with paradigms (e.g. lengthy testing sessions) and under testing conditions inappropriate for patients (e.g. psychopharmacological studies; Ettinger et al., 2014).

Autistic and schizotypic traits are commonly assessed using spectrum-specific self-report questionnaires. For schizotypy, a common questionnaire is the Short Oxford-Liverpool Inventory of Feelings and Experiences (sO-LIFE; Mason et al., 2005) or the Schizotypal Personality Questionnaire (Raine, 1991). For autistic traits, the most common questionnaire is the Autism Spectrum Quotient (AQ; Baron-Cohen et al., 2001) with others being the Social Responsiveness Scale (Constantino & Todd, 2003) or the Broad Autism Phenotype Questionnaire (Hurley et al., 2007). These questionnaires are economic and convenient tools to assess illness-related traits in small and large samples. They make it possible to search for behavioural, biological, or endophenotypic markers (Ettinger et al., 2015; Sucksmith et al., 2011) in randomly selected or preselected healthy populations (e.g. Cappe et al., 2012; Rhodes et al., 2013). As a common observation from such studies, elevated autistic and schizotypic traits, respectively, associated with deficits previously observed from the corresponding clinical disorder. Of major interest to our study, there has been hardly any study that assessed both autistic and schizotypic personality traits in relationships with the same behavioural markers (e.g. Russell-Smith et al., 2010). Yet, in our view, such an endeavour would be critical to test experimentally Crespi and Badcock (2008)'s proposition of opposite social functioning in ASD and SSD using the healthy ends of the spectra. In other words, social cognitive functions should relate in predictable ways with hypo-Mentalism (autistic traits) and hyper-Mentalism (positive schizotypy), while the shared traits should be of low relevance.

In the current context, one recent study is critical (Dinsdale et al., 2013). These authors distinguished, among the autistic and the schizotypic traits, those that were shared and those that were opposite, hence possibly spectrum-specific. The authors observed that autistic and schizotypic traits share *negative* features (i.e. social deficits and anhedonia) and *positive* features, i.e. positive schizotypy and autistic attention to detail (see also Hurst, Nelson-Gray, Mitchell, & Kwapil, 2007; Russell-Smith et al., 2011), as has been noted for ASD and SSD (Konstantareas & Hewitt, 2001). Dinsdale et al. (2013) showed statistically that autistic social deficit traits opposed positive schizotypic traits. While previous studies reported this opposition too (i.e. negative correlations), these previous authors did not discuss them (Hurst, Nelson-Gray et al., 2007; Russell-Smith et al., 2011). Following Dinsdale et al. (2013), Sierro, Rossier and Mohr (2016) replicated their findings, and interpreted them as an opposition between positive schizotypic traits and autistic *mentalizing deficit* traits (i.e. deficits in mindreading and social imagination; see also Bishop et al., 2004), reflecting hyper- and hypo-Mentalism respectively. These psychometric results support Crespi and Badcock's (2008) diametrical model at the trait level. Further studies should show corresponding opposing hypo- and hyper-Mentalistic expressions at the behavioural level. Crespi and Badcock

(2008) have, indeed, proposed that opposite phenotypes or cognitive styles would express, notably, in opposite face and gaze processing.

We suggest that face processing of ambiguous stimuli may help accounting for behavioural expressions of hyper- and hypo-Mentalism. Face processing is highly developed in human beings (Haxby et al., 2000). Faces inform us on another person's identity, intentions, emotions, and otherwise communicative messages (e.g. information in the environment by following another person's gaze). Processing information in faces is impaired, though differently, in both ASD (Dawson et al., 2005; Weigelt et al., 2012) and SSD (Caharel et al., 2007; Joshua & Rossell, 2009; Shin et al., 2008). These studies showed that ASD expresses preserved configural processing but a broader face memory deficit, whereas SSD expresses an impaired configural encoding of face. Moreover, hypo- and hyper-Mentalism in face processing shows in an undersensitivity to gaze in ASD and an oversensitivity to gaze in SSD (Hooker & Park, 2005; Wallace et al., 2006). We suggest here that these opposite processing styles for faces should express in opposite processing biases for FLOs. Hyper-Mentalism in SSD should result in a tendency to see a face in FLOs and hypo-Mentalism in ASD should result in the converse tendency not to see a face. As we will argue below, these opposite biases for FLO can be assessed behaviourally as a stronger versus weaker face IE (Yin, 1969).

Individual differences in the tendency to see faces in FLO have been studied before. Here, researchers study apophenia, or more precisely face pareidolia. Apophenia refers to attributing meaning to unrelated stimuli or events (e.g. seeing a connection between reading the word "thunder" and hearing a simultaneous crack of thunder outside; Brugger, 2001). Pareidolia is the visual form of apophenia, i.e. the tendency to see a meaningful pattern in ambiguous visual stimuli, such as seeing a face in clouds or seeing Jesus' face on a toast (Belayachi et al., 2014; Liu et al., 2014). Supporting our reasoning so far, face pareidolia is enhanced in individuals high in positive schizotypy and in patients with schizophrenia when compared to respective controls (Belayachi et al., 2014; Brugger, 2001; see also: Riekki et al., 2013). Thus, apophenia (hyper-associative) cognitive style is a common mechanism underlying positive schizotypy and over-Mentalism (Fyfe et al., 2008). ASD children identified fewer FLO than neurotypicals (Ryan, Stafford & King, 2016), and showed abnormal orientation towards FLO (Guillon et al., 2016). Despite the lack of published study using autistic traits in healthy populations, we expect autistic mentalizing deficit traits to associate with a smaller pareidolia-proneness.

While previous studies assessed face pareidolia explicitly (asking participants if and where they see a face; Churches et al., 2009), we assessed face pareidolia for FLO more implicitly by

assessing participants' IE. To do so, we need to be certain that FLO are processed like faces. An eye movement study showed that FLO (car fronts) and faces elicit a similar visual exploration pattern (Windhager et al., 2010). Electrophysiologically, FLOs as compared to faces elicited comparable though weaker EEG components (Caharel et al., 2007; Churches et al., 2009). Neuroimaging studies indicated that FLO activated face-specific brain regions (Hadjikhani et al., 2009; Kühn, Brick, Müller, & Gallinat, 2014). The configural processing style typically applied when processing faces can be disrupted, though, by presenting inverted faces (Haxby et al., 2000). For upright faces, healthy people are experts in the processing and discrimination of faces and their identities, i.e. the analysis of the configural relationships of face parts in faces. For inverted faces, the processing of the configural information becomes disrupted. Thus, for face IEs, individuals are commonly better to recognize upright as compared to inverted faces, and in the case of face pareidolia, we should also observe a FLO IE.

In three subsequent studies, we tested IE for objects, FLOs and faces in undergraduate students differing in their scores on the sO-LIFE (Mason et al., 2005) and the AQ (Baron-Cohen et al., 2001). Across studies, we increasingly improved the formerly published visual material (Hadjikhani et al., 2009), because homogenizing the perceptual features within-category is likely to facilitate IE (Ashworth, Vuong, Rossion, & Tarr, 2008; Thierry, Martin, Downing, & Pegna, 2007). The material used in Hadjikhani et al. (used in our first study) is visually more homogenous for faces than for objects and FLOs. Thus, we homogenized visually the items in the object category (chairs; studies 2 and 3) and the FLO category (car fronts; study 3). We expected that enhanced Mentalism tendencies (high positive schizotypy; low autistic mentalizing deficits) would relate to a stronger pareidolia bias (FLO IE) and lower Mentalism tendencies (low positive schizotypy; high autistic mentalizing deficits) would relate to a weaker pareidolia bias (FLO IE). These opposite tendencies might also emerge for face IE, but not for object IE. We did not expect that these IE differences relate to shared autistic and schizotypic traits (Dinsdale et al., 2013).

4.2 STUDY 1

4.2.1 MATERIALS AND METHODS

4.2.1.1 PARTICIPANTS

Our sample consisted of 48 undergraduate students (22 women; age: mean±sd: 25.29±10.51). All were recruited via personal contact around the local university. Participation was voluntary. Some students took part for course credits. Before study commencement, all participants signed an informed consent form. Participants agreed that their grouped, anonymized data can be used for research purposes. All were informed that their participation is voluntary, and that they

could withdraw from participation at any time, without negative consequences. All aspects of the study were conducted in accordance with the guidelines of the declaration of Helsinki (World Medical Organization, 2013).

4.2.1.2 SELF-REPORT QUESTIONNAIRES

4.2.1.2.1 AUTISTIC TRAIT QUESTIONNAIRE

We assessed autistic traits using the French version of the Autism Spectrum Quotient (AQ; Baron-Cohen et al., 2001), recently translated (Sonié et al., 2011) and validated (Sierro, Rossier, & Mohr, 2016). The AQ comprises 50 items (24 reverse formulated items). Respondents indicate their agreement with each item on a 4-point Likert scale (i.e. "Definitely agree", "Agree", "Don't agree", and "Definitely disagree"). The AQ consists of 5 dimensions (10 items per dimension): Social Skill deficits (SocSki, e.g. “I find it hard to make new friends.”), Communication deficits (Comm, e.g. “I am often the last to understand the point of a joke.”), Attention Switching deficits (AttSwi, e.g. “I prefer to do things the same way over and over again.”), Imagination deficits (Ima, e.g. “I don’t particularly enjoy reading fiction.”), and Attention to Details (AttDet, e.g. “I tend to notice details that others do not.”). In line with original scoring method, we dichotomized responses (Baron-Cohen et al., 2001), giving 1 point for "Definitely agree" and "Agree", and 0 for "Don't agree", and "Definitely disagree" responses (inverted for reverse formulated items). Dimension scores were computed by summing the scores of the respective items. Thus, dimension scores range from 0 to 10.

4.2.1.2.2 SCHIZOTYPIC TRAIT QUESTIONNAIRE

We assessed schizotypic traits using the French version (Sierro, Rossier, Mason et al., 2016) of the sO-LIFE (Mason et al., 2005). The sO-LIFE comprises 43 items (8 reverse formulated items) consisting in assertions about participants’ feelings and experiences. Respondents indicate whether or not each item's assertion accurately describe them by choosing either *yes* or *no*. sO-LIFE comprises 4 dimensions: positive schizotypy (Unusual Experiences [UnEx], e.g. “Are your thoughts sometimes so strong that you can almost hear them?”, 12 items), negative schizotypy (Introvertive Anhedonia [IntAn], e.g. “Do you prefer watching television to going out with people?”, 10 items), cognitive disorganization (CogDis, e.g. “Are you easily confused if too much happens at the same time?”, 11 items), and impulsive nonconformity (ImpNon, e.g. “Do you at times have an urge to do something harmful or shocking?”, 10 items). Each "yes" answer scores one point whereas each "no"

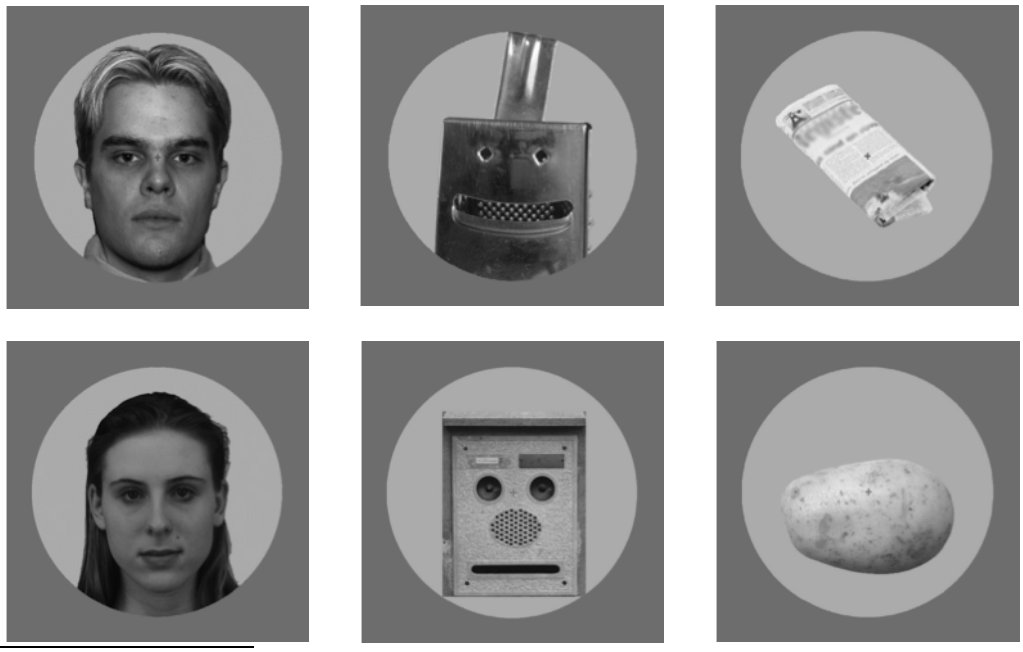
answer scores zero points (inverted for reverse formulated items). We summed the scores of all items belonging to a given subdimension.

4.2.1.3 VISUAL RECOGNITION TASK

4.2.1.3.1 STIMULI

We used the face (FACE) stimuli, FLO stimuli, and object [OBJ] stimuli used before by Hadjikhani et al. (2009) and Churches et al. (2009). In these previous studies, the authors had used a total of 120 stimuli (40 per category). The FACE stimuli consisted of a homogenous sample of FACES from the NimStim Emotional Face Stimuli database[18]. The FACE stimuli consisted of an equal amount of front-viewing male or female neutral faces (examples, see Figure 1). The FLOs were taken from the *FACES* book by Robert and Robert (2000) by Hadjikhani et al. (2009). The FLO stimuli consisted of a heterogeneous sample of pictures depicting non-living objects, which were rated as looking like a face (examples, see Figure 1). The OBJ stimuli consisted of a heterogeneous sample of pictures depicting non-living objects (examples, see Figure 1; see also Hadjikhani et al., 2009).

Figure 1. We show two stimuli per category, left column for faces (FACE), central column for face-like objects (heterogeneous FLO) and right column for objects (heterogeneous OBJ).



[18] The NimStim Emotional database website: http: //www.macbrain.org/faces/index.htm#faces

We standardized all stimuli; they were transformed to gray-scale, resized, and centered using The GIMP 2.6.12. (http: //www.gimp.org) and Adobe Photoshop Elements 8 (Adobe Systems, San Jose, California). Each OBJ stimuli was shown in the centre of a 300 pixel large circle (diameter of 11.4 cm on the screen). This circle was shown in the centre of a 480 × 480 pixel large dark grey square (13.98 × 13.98 cm on the screen) (see Figure 1). The FACE stimuli were centred according to the nasion, the FLO and OBJ according to the centre of the image itself.

4.2.1.3.2 PROTOCOL

Half of the stimuli of each category were randomly allocated to an *old* stimuli group and the other half to a *new* stimuli group. For FACE, FLOs, and OBJ, separately, participants were first exposed to a learning phase (seeing the old stimuli in an upright position), followed by a recognition phase. In the learning phase, participants were passively and sequentially exposed to the old stimuli (exposure duration of 3000 ms), interspersed by an interstimulus interval of 500 ms (seeing a central fixation cross). In the learning phase, the order of stimuli was pseudo-randomized (i.e. the same randomized stimuli order was displayed sequentially for all participants). After the learning phase, participants took a short break (about <1 min) while reading instructions for the recognition phase. In the recognition phase, participants saw sequentially the previously presented old stimuli (targets) and randomly intermixed the new stimuli (distractors). The order of stimuli was also pseudo-randomized. Half of the stimuli were presented upright (UPR) and the other half presented inverted (INV). The pictures remained on the screen until a response was provided. If participants decided that it was an old item, they had to press the u-button on the keyboard. If participants decided that it was a new item, they had to press the p-button on the keyboard. Between picture presentations, participants saw a central fixation cross for 1’000 ms.

Each participant performed the learning and recognition phase for FACE, FLOs and OBJ. We counterbalanced between participants the order in which the three categories were presented. The experiment was programmed using E-Prime 2.0 Pro on Windows OS. We recorded participants' response keys and latencies. The task took about 10 to 15 min to perform.

4.2.1.4 GENERAL PROCEDURE

With study enrolment, participants first filled in the AQ and sO-LIFE questionnaires. Subsequently, they were explained the computer task and installed in front of a computer (distance

from eye-screen ~57cm). The experimenter ensured they understood the computerized instructions of the task. Subsequent to the computer task, participants were fully debriefed and thanked for their participation.

4.2.1.5 DATA ANALYSIS

We initially tested 48 participants for which we obtained complete behavioural and psychometric data. To account for the relationships between autistic and schizotypic traits to face pareidolia, we took two different approaches, once we calculated two scores according to previous PCAs (Chapter 3; Sierro, Rossier & Mohr, 2016; see Dinsdale et al., 2013), and once we kept hypothesis-relevant individual dimension scores. With regard to the PCA, the two scores either represented shared or opposite autistic and schizotypic traits. The two scores were determined considering first the results of previous PCAs on AQ and sO-LIFE scores collected from large populations (Dinsdale et al., 2013; Sierro, Rossier & Mohr, 2016). In these former studies, authors reported on one PC representing the shared autistic and schizotypy features (PCSF) and another PC representing the opposing autistic Mentalistic deficit and positive schizotypy (PCDF). In the present study, we computed our PCSF and PCDF scores by using the PC analysis results from a larger but comparable population (Sierro, Rossier & Mohr, 2016). Thus, to calculate our respective PCSF and PCDF scores, we multiplied each individual subscale score with the respective PC loadings for this scale (see Dinsdale et al., 2013). Subsequently, we calculated the mean of these individual scores obtaining a separate PCSF and PCDF for each individual. These PC scores were based on the AQ with the traditional dichotomic scoring (Baron-Cohen et al., 2001), hence we named them PCSF-BC, and PCDF-BC (Appendices Table 27).

For our second approach, we retained specific schizotypic and autistic traits dimensions, UnEx and Ima scores, because these dimensions are the most likely to reflect hyper-Mentalism, and hypo-Mentalism, respectively (Sierro, Rossier & Mohr, 2016). We reported questionnaires scores and PC scores descriptives, and compared these with previous data from a normative population (Sierro, Rossier & Mohr, 2016).

For the recognition task, we computed individuals' percentage of correct responses and reaction times (RTs) per stimulus category and stimulus orientation. Beforehand, we removed RTs below 500 ms and above 3 sec, and then we computed mean percent correct (Accuracy) and mean RT, based on correct responses, for each participant and for each condition. Overall, mean Accuracy was 90%±15%, and mean RT was 1128.48±580.47 ms.

To measure pareidolia effect, we adopted two complementary strategies: once we computed pareidolia indices representing liability to experience pareidolia, once we used IE indices and raw behavioural variables in Accuracy and RT. Based on the behavioural variables, we computed category-specific IE indices representing the performance costs induced by stimulus inversion. For IE in Accuracy, we computed the difference between INV and UPR performances (percent correct responses UPR condition – percent correct responses INV condition), and for IEs in RT (RT INV condition – RT UPR condition). Thus, for both Accuracy and RT, the larger the IE index, the larger is participants' performance decrease due to inversion, i.e. the larger their reliance on a configural processing strategy. Our first way to account for pareidolia effect was to compute a pareidolia index by subtracting FLO IE from FACE IE, for Accuracy and RT (FACE IE – FLO IE; e.g. Butler et al., 2008). Thus, we assumed that smaller pareidolia indices represent a larger pareidolia effect, i.e. a comparable configural processing style for FACE and FLO stimuli, while larger pareidolia indices represent a smaller pareidolia effect, i.e. a stronger configural processing for FACE as compared to FLO stimuli. Our second way to account for pareidolia index was to consider IE indices and raw behavioural variables in Accuracy and RT. More precisely, we assumed a genuine pareidolia effect would be represented by the magnitude of IE for FLO, and more precisely by performances in FLO:UPR, both reflecting face-like configural processing.

Overall, most of our questionnaires and behavioural variables were normally distributed, as their skew was comprised within the +1/-1 range. In particular, pareidolia indices for both Accuracy and RT (also IE indices for FACE, FLO and OBJ for Accuracy, and Accuracy for FACE:INV and FACE:UPR) were in acceptable range. In contrast, some raw behavioural variables were skewed and showed a floor effect (e.g. RT OBJ).

To test whether we obtained the expected differences in IE between categories, we performed two repeated measures ANOVAs, one on the accuracy IE indices and one on the RT IE indices with Category as within-subject independent variable, following recommendations by Field, Miles and Field (2012). Throughout, we report effect size statistics using generalized eta-squared (η_G^2; Olejnik & Algina, 2003). To test specific predictions, we applied orthogonal planned contrasts (Brauer & McClelland, 2005). Post-hoc tests correcting for multiple comparisons were applied (Bonferroni: sphericity assumptions violated; Tukey: sphericity respected; Field et al., 2012). We report corrected *p*-values in case of significant results. To test for Category-specific IE, we performed unilateral one sample *t*-tests against $\mu=0$, for each Category and index type (accuracy, RT) separately. To test for a pareidolia effect, we also performed unilateral one sample *t*-tests against $\mu=0$ for pareidolia indices of each measure (accuracy, RT). We postulate that IEs and

pareidolia indices would be positive, and significantly higher to zero ($\mu>0$). For these tests, we report Cohen's *d* effect sizes.

To assess the relationships between our personality scores of interest and pareidolia effect, we performed multiple linear regressions, once with the PC scores and once with the original dimensions scores (UnEx, Ima). In a first approach, we created regression models with pareidolia index scores (Accuracy, RT) as the respective outcome measures, once with PC scores (PCSF-BC, and PCDF-BC) as predictor variables, once with original dimension scores (UnEx, Ima) as predictor variables. For each regression, we report standardized slopes (β), *t*, *p*-values and standard errors in Tables, and R^2, *F*, *df* and *p*-values in text. Significant and trend relationships are represented using scatter plots. In a second approach, we created regression models with IE index scores and the other behavioural variables (Accuracy, RT) as outcome measures, with the original dimension scores (UnEx, Ima) as predictor variables. Here, we did not perform regression analyses on PC scores, because we perform analyses that are targeted at pre-defined theory-based predictors (UnEx, Ima). For each regression, we report R^2 and β of each predictor in Tables, and R^2, *F*, *df* and *p*-values of significant models in the text.

4.2.2 RESULTS

4.2.2.1 PARTICIPANTS

We tested a comparable number of men (n=26) and women (n=22; χ^2=0.33, p=.564). The mean (±SD) age of men (24.29±10.54) did not differ from the one of women (26.43±10.61; Wilcoxon rank-sum test: W=297, p=.306, z=-0.14).

4.2.2.2 SELF-REPORT QUESTIONNAIRES

According to student's *t*-tests (Table 9), our AQ and sO-LIFE subscale scores were largely comparable to those reported in comparable French-speaking undergraduate samples (for sO-LIFE: Sierro, Rossier, Mason et al., 2016; for AQ and PC scores: Sierro, Rossier & Mohr, 2016). Our CogDis scores and PCDF scores were significantly smaller than those of the normative samples.

Table 9. Descriptives for Study 1 for dimension scores of O-LIFE Short and AQ. Significant differences between Study 1 and Normative sample scores are in bold.

		Study 1			Normative Sample					
Instrument	Dimension	mean	sd	α	mean	sd	*t*	*df*	*p*	*d*
O-LIFE Short	Global	11.52	6.91	.88†	14.45†	6.28	**-3.15**	**1'094**	**.002**	**-0.46**
	UnEx	3.27	2.34	.83†	3.73†	2.59	-1.21	1'094	.226	-0.18
	CogDis	3.54	3.13	.84†	5.53†	2.82	**-4.77**	**1'094**	**<.001**	**-0.70**
	IntAn	1.94	1.92	.72†	2.14†	1.76	-0.78	1'094	.438	-0.12
	ImpNon	2.79	1.89	.74†	3.04†	2.04	-0.83	1'094	.405	-0.12
AQ-BC	Total	15.1	7.35	.75††	15.56††	5.58	-0.55	967	.584	-0.08
	SocSki	2.35	2.18	.70††	2.15††	1.95	0.69	967	.491	0.10
	AttSwi	3.69	2.27	.56††	4.12††	2.06	-1.40	967	.161	-0.21
	Comm	1.81	1.88	.46††	2.01††	1.57	-0.85	967	.395	-0.13
	Ima	2.98	1.96	.53††	2.58††	1.69	1.59	967	.113	0.24
	AttDet	4.31	2.56	.69††	4.70††	2.16	-1.21	967	.227	-0.18
PC score	PCSF-BC	8.99	5.88	–	9.72††	4.65	-1.05	967	.296	-0.16
PC score	PCDF-BC	9.03	4.76	–	10.80††	4.83	**-2.48**	**967**	**.013**	**-0.37**

mean=mean; sd=standard deviation; *t*=Student's *t*-test statistic; *p*= Student's *t*-test p-value; *d*= Cohen's *d* effect size

† normative sample based on *n*=1'048 respondents (Sierro, Rossier, Mason et al., 2016)

†† normative sample based on *n*=921 respondents (Sierro, Rossier, & Mohr, 2016)

PCSF-BC: PC score of Shared Features with AQ-BC; PCDF-BC: PC score of Diametrical Features with AQ-BC

Global: total sO-LIFE score; UnEx: Unusual Experiences; CogDis: Cognitive Disorganization; IntAn: Introvertive Anhedonia; ImpNon: Impulsive Nonconformity

Total: Total AQ-BC score; SocSki: Social Skills deficits; AttSwi: Attention Switching deficits; Comm: Communication deficits; Ima: Imagination deficits; AttDet: Attention to Details

4.2.2.3 VISUAL RECOGNITION TASK PERFORMANCE

The descriptives for IE indices for Accuracy and RTs are reported in Table 10. The ANOVA on the accuracy IE indices showed a significant main effect of Category (ε_{HF}=.79, F(1.58, 74.46)=16.35, p<.001, η_G^2=.17) (see Figure 2a). Planned orthogonal contrasts showed a significant linear decrease in the IE from FACE to FLOs and to OBJ (t(94)=5.60, p<.001, r=.50), whereas the orthogonal contrast was not significant (t(94)=-1.17, p=.244, r=.12). Post-hoc comparisons showed that the IE was significantly larger for FACE (0.15±0.22) than for FLO (0.04±0.15) and OBJ (-0.1±0.07) (p's <.05), but did not differ between FLO and OBJ (p >.05). The one-sample t-tests against μ = 0 were significant for FACE (t(47)=4.95, p<.001, d=0.72) and FLO (t(47)=1.82, p=.037, d=0.27), but not for OBJ (t(47)=-128, p=.896, d=-0.19). In the first two cases, the IE is positive, thus, participants showed an advantage for UPR as compared to INV faces as well as FLO. The pareidolia index for Accuracy was significantly larger than 0 (t(47)=3.25, p=.001, d=0.47), suggesting larger reliance on a configural processing for FACE as compared to FLO.

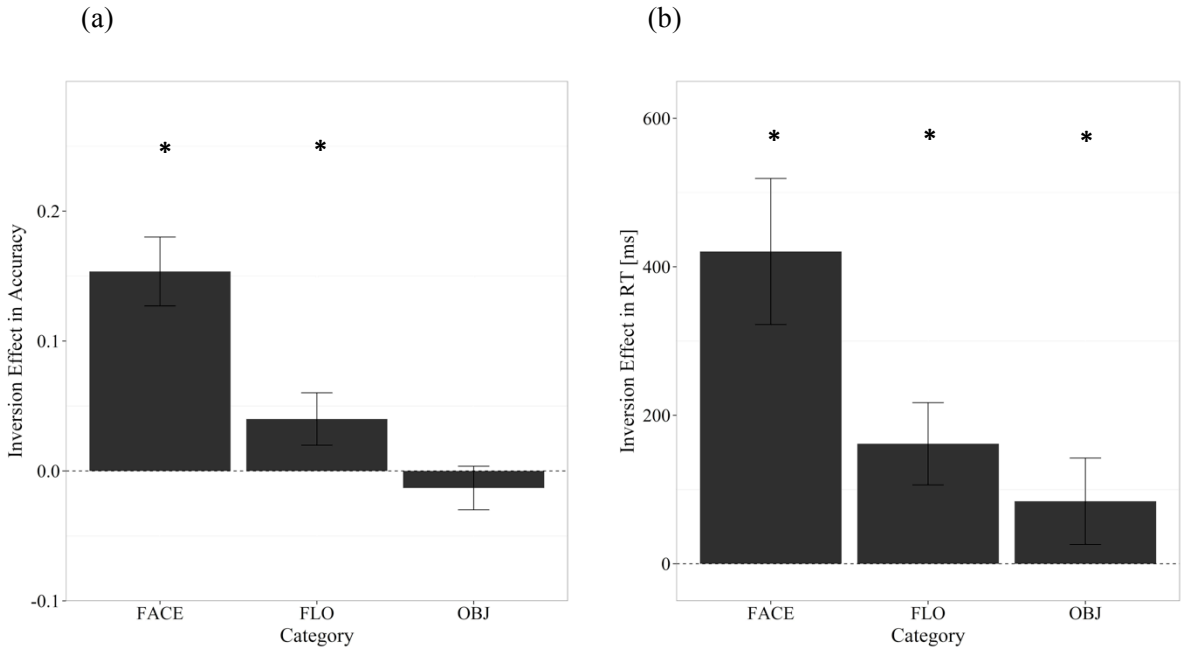
The second ANOVA on the RT IE indices showed a significant main effect of Category (ε_{HF}=.81, F(1.62,76.14)=11.48, p<.001, η_G^2=0.12; see Figure 2b). Planned orthogonal contrasts showed a significant linear decrease in the IE from FACE to FLOs and to OBJ (t(94)=4.58, p<.001, r=.43), whereas the orthogonal contrast was not significant (t(94)=-1.40, p=.166, r=.14). Post-hoc comparisons showed that the IE was significantly larger for FACE (412±538) as compared to FLO (161±347) and OBJ (84±170; p's <.05), whereas IEs for FLO and OBJ did not significantly differ from each other (p >.05). The one-sample t-tests against μ=0 were significant for FACE (t(47)=3.52, p<.001, d=0.51), FLO (t(47)=3.22, p=.001, d=0.47), and OBJ categories (t(47)=3.43, p<.001, d=0.50). In all cases, the IE is positive, thus participants showed an advantage for UPR as compared to INV stimuli. The pareidolia index for RT was significantly larger than 0 (t(47)=2.15, p=.018, d=0.31), suggesting larger reliance on a configural processing for FACE as compared to FLO.

Table 10. Descriptives for Study 1 for Accuracy and RT performances, as a function of Category and Orientation.

	Accuracy		RT	
Measures	mean	sd	mean	sd
FACE:INV	0.72	0.2	1597.8	882.2
FACE:UPR	0.88	0.13	1177.21	459.19
FLO:INV	0.91	0.14	1245.83	611.82
FLO:UPR	0.95	0.09	1084.17	406.1
OBJ:INV	0.99	0.05	875.03	264.89
OBJ:UPR	0.98	0.06	790.82	183.04
IE:FACE	0.15	0.22	420.59	827.86
IE:FLO	0.04	0.15	161.66	347.5
IE:OBJ	-0.01	0.07	84.21	170.28
IE:FACE-FLO	0.11	0.24	258.93	832.49

mean=mean; sd=standard deviation
FACE: face stimuli; FLO: face-like object stimuli; OBJ: object stimuli
INV: inverted orientation; UPR: upright orientation; IE: Inversion effect
IE:FACE-FLO: Pareidolia Index

Figure 2. Mean IE indices for (a) Accuracy and (b) RT for the three stimulus categories separately. Vertical bars represent standard errors (corrected for repeated measures). Stars indicate IEs that are significantly different from 0 at one-sample *t*-tests (* *p*'s<.050).



4.2.2.4 RELATIONSHIPS BETWEEN PERSONALITY SCORES AND VISUAL RECOGNITION PERFORMANCE

The regression models in Table 11 were non-significant (all *p's* >.05), with the exception of one model showing a trend. For Accuracy, the model predicting a pareidolia index in Accuracy with PC scores showed a trend towards significance (R^2=.12, F(2,45)=2.95, p=.063). PCDF-BC significantly predicted a larger pareidolia effect in Accuracy (i.e. smaller IE:FACE-FLO), as represented by Figure 3. Also, PCSF-BC scores tended to associate with a smaller pareidolia effect in Accuracy (i.e. larger IE:FACE-FLO). These findings could not be explained by variance in UnEx or Ima, since these models were not significant (R^2=.07, F(2.45)=1.76, p=.184). For RTs, none of the models were significant, i.e. predicting pareidolia effect with PC scores (R^2=.05, F(2,45)=1.20, p=.310), or UnEx and Ima scores (R^2=.02, F(2,45)=0.34, p=.711).

Table 11. Regression analysis results of pareidolia indices for Accuracy and RT as outcome measures, and PCDF-BC, PCSF-BC, UnEx and Ima scores as Predictor variables. Significant results are indicated in bold

	Outcome	Predictors	R^2	β	*t*(46)	*P*	IC 2.5 %	IC 97.5 %
Accuracy	IE:FACE-FLO		*0.11°*					
		PCSF-BC		0.282	1.710	.094	-0.002	0.025
		PCDF-BC		**-0.389**	**-2.36**	**.025**	**-0.037**	**-0.003**
	IE:FACE-FLO		.07					
		UnEx		-0.254	-1.657	.105	-0.058	0.006
		Ima		0.215	1.407	.166	-0.011	0.065
RT	IE:FACE-FLO		.05					
		PCSF-BC		.264	1.544	.130	-11.361	85.998
		PCDF-BC		-.118	-0.691	.493	-80.818	39.526
	IE:FACE-FLO		.02					
		UnEx		-0.040	-0.254	.801	-127.552	99.031
		Ima		0.131	0.828	.412	-79.574	190.687

° p<.10; * p<.05; ** p<.001
PCSF-BC: PC score of Shared Features with AQ-BC; PCDF-BC: PC score of Diametrical Features with AQ-BC
UnEx: Unusual Experiences; Ima: Imagination deficits
IE:FACE-FLO: Pareidolia Index

We performed regression analyses with IE variables and raw behavioural variables as outcome variables and UnEx and Ima as predictor variables (Table 12). For Accuracy, all regression models were non-significant (p's >.05). Trends were observed for IE:OBJ (R^2=.11, F(2.45)=.074), and FLO:UPR models (R^2=.114, F(2,45)=2.891, p=.066). Larger UnEx scores tended to associate with improved Accuracy for FLO:UPR, whereas larger Ima scores associated with significantly smaller Accuracy for FLO:UPR. Moreover, larger Ima scores associated with significantly smaller IE for OBJ. For RTs, all regression models were non-significant (p's>.05) except the one for FACE:UPR (R^2=.15 , F(2,45)=3.87, p=.028). Larger UnEx scores significantly associated with longer RTs for FACE:UPR (see Table 12).

Figure 3. Scatterplot demonstrating the statistical trend relationship between pareidolia index scores for Accuracy (IE:FACE-FLO) as a function of "diametrical" Mentalistic traits (PCDF-BC). The smaller the index, the more FACE and FLO are treated in a similar fashion.

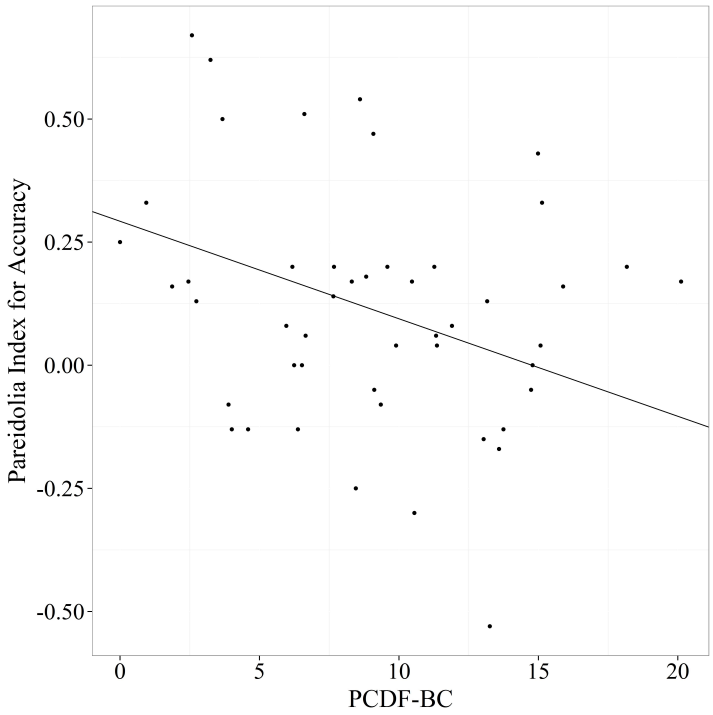


Table 12. Regression analysis results with IE indices and raw behavioural variables as outcome measures and UnEx and Ima as predictor variables. Significant results are indicated in bold

	R^2	β's for Accuracy		R^2	β's for RTs	
		UnEx	Ima		UnEx	Ima
IE:FACE	.04	-.219	.113	.02	-.089	.144
IE:FLO	.03	.094	-.184	.01	-.117	.030
IE:OBJ	*.11°*	.051	**-.345***	.05	-.147	.236
FACE:UPR	.03	-.183	.015	**.15***	**.406***	-.093
FACE:INV	.02	.122	-.113	.03	.127	.087
FLO:UPR	*.11°*	*.291°*	**-.303***	.01	-.022	-.070
FLO:INV	.01	.096	-.005	.01	-.081	-.029
OBJ:UPR	.06	.024	-.244	.00	.019	.005
OBJ:INV	.03	-.040	.183	.02	-.081	.155

° p<.10; * p<.05; ** p<.001

UnEx: Unusual Experiences; Ima: Imagination deficits; FACE: face stimuli; FLO: face-like object stimuli; OBJ: object stimuli; INV: inverted orientation; UPR: upright orientation; IE: Inversion effect

4.2.3 SHORT DISCUSSION

In this first study, we observed significant IEs, notably for FLO. Also, we confirmed relationships of pareidolia-proneness with "diametrical" Mentalism traits, but not "shared" ones. Yet, we did not find opposition in behaviour for hyper- versus hypo-Mentalism traits. First, we observed significant IEs in Accuracy and RT, in particular for FACE and FLO. Regressions showed that PCDF-BC predicted a larger pareidolia effect in Accuracy (smaller pareodolia index), while shared negative traits did not (PCSF-BC). Yet, pareidolia effect in RT did not show comparable results. Finally, we found no opposition between hyper- and hypo-Mentalistic traits, because UnEx and Ima did not show any significant relationships with pareidolia indices, neither in Accuracy nor in RTs. Subsequent detailed regressions showed no rise of IE:FLO as a function of personality traits, as should be expected from a pareidolia effect. Hence, instead of finding a link with the pareidolia measures, UnEx and Ima associated with distinct Face and FLO processing performances. Larger UnEx scores associated with slower FACE:UPR processing, and a trend to better FLO:UPR recognition, while larger Ima scores associated with worse FLO:UPR recognition.

With our behavioural paradigm, we demonstrated IE for FACE *and* FLO, suggesting that FLO elicit face-like processing and are thus appropriate to investigate pareidolia-proneness. IE were demonstrated for FACE as well as FLO in Accuracy and RTs. For OBJ, the IE was present only in RTs. More precisely, for both Accuracy and RTs, planned contrasts and post-hoc comparisons showed that IE was stronger as a function of stimuli's putative faceness, i.e. strongest for FACE, weaker for FLO, and weakest for OBJ. These results confirm that FLOs, as an ambiguous intermediary category, are also sensitive to IE, in line with reports of face-like processing for heterogeneous FLOs (Churches et al., 2009; Hadjikhani et al., 2009). We, however, note that Caharel et al. (2013) did not find a behavioural IE:FLO using Arcimboldo paintings. These overall behavioural results indicate that we can use IE:Accuracy and IE:RTs variables as indices sensitive to configural processing, and by inference for further statistical comparisons on links with personality traits. In particular, observing IE:FLO is promising when aiming to investigate configural processing in participants with ASD or autistic traits, because FLO are less likely to trigger avoidance behaviour towards human faces in these populations (as proposed by Hadjikhani et al., 2009).

In line with our prediction, we found that larger Mentalism associated with a larger pareidolia-proneness, although not confirmed by all our measures. Larger Mentalism scores (PCDF-BC) showed a significant association with a larger pareidolia effect for Accuracy (i.e. smaller IE:FACE-FLO). However, this effect could neither be explained by positive schizotypy (UnEx), nor

by Ima because they did not associate with differences in pareidolia effect for Accuracy (i.e. smaller IE:FACE-FLO). Similarly, larger UnEx scores did not associate with a larger IE:FLO for Accuracy, but only showed a trend association with better FLO:UPR performance for Accuracy. Still, a genuine pareidolia effect (i.e. smaller IE:FACE-FLO) should be reflected by a more face-like FLO processing (i.e. *larger* IE:FLO and FLO:UPR, in Accuracy and/or RT). Hence, our data do not permit to interpret the association between distinct traits (PCDF-BC) and pareidolia index for Accuracy (IE:FACE-FLO) as *undoubtedly* accounting for pareidolia-proneness.

Instead of pareidolia-proneness, positive schizotypy (UnEx) and autistic mentalizing deficits (Ima) might reflect opposite basic FACE and FLO processing. Increased positive schizotypy associated with slower FACE:UPR performance in RTs and tended to associate with a better Accuracy for FLO:UPR, whereas increased Ima associated with a lower Accuracy for FLO:UPR. Worse performance on FACE as a function of UnEx are reminiscent of configural processing deficits reported from schizophrenia patients (Schwartz et al., 2002), in particular when positive symptoms are enhanced (Chambon et al., 2006). They are also reminiscent of similar deficits reported from unaffected relatives of patients with schizophrenia and individuals with schizotypic traits (Batty et al., 2014; Kim et al., 2010; Larøi, D'Argembeau, Brédart, & Van der Linden, 2007). The opposite relationships of UnEx and Ima regarding Accuracy for FLO:UPR might be due to a higher fantasy-proneness that goes along with elevated positive schizotypy (Merckelbach, Rassin, & Muris, 2000) and less social imagination that goes along with Ima (Crespi, Leach, Dinsdale, Mokkonen, & Hurd, 2016; Sierro, Rossier & Mohr, 2016). In particular fantasy-proneness and high social imagination (high UnEx, low Ima) may be beneficial to encode and recognize the previously seen FLO. Though one would prefer more compelling statistical outcomes, the current results might support a diametrical opposition of ASD and SSD (Crespi & Badcock, 2008), yet pertaining to basic face processing rather than pareidolia-proneness .

4.2.4 LIMITATIONS

The study questions have been straight forward. The overall results are promising, but far from clear-cut. Given the numerous comparisons, the significant results were few, though in the expected direction. One could now conclude that the results are chance observations, or one could question the questionnaires as well as the behavioural paradigm. Moreover, some questionnaire scores (IntAn, SocSki and Comm) and behavioural variables were not normally distributed (raw Accuracy variables FLO:INV, FLO:UPR, OBJ:INV, OBJ:UPR; all raw RT variables, all IE:RT indices).

Self-report questionnaires measuring schizotypic and autistic traits generally suffer from validity and reliability issues that would need to be fixed (e.g. Sierro, Rossier, Mason et al., 2016; Sierro, Rossier & Mohr, 2016). For instance, Hurst, Nelson-Gray et al. (2007) criticized the lack of reliability of certain dimensions, notably Ima, of the original AQ, English version (Baron-Cohen et al., 2001). Also, most studies revised Baron-Cohen et al. (2001)'s original AQ factor structure and proposed alternative ones for English version (e.g. Hurst, Nelson-Gray et al., 2007; Lau, Kelly et al., 2013). A recent validation study of French AQ supported these claims and suggested to use Kloostermann et al.'s (2011) alternative factor structure (Sierro, Rossier & Mohr, 2016). Further assessment and improvement of sO-LIFE and AQ will be necessary, irrespectively of linguistic versions (Fonseca-Pedrero et al., 2015; Sierro, Rossier, Mason et al., 2016; Sierro, Rossier & Mohr, 2016). Ultimately, improved factor structures as well as reliability may improve the detection of behaviour-personality relationships.

With regard to the behavioural paradigm, our results suggest that our visual stimuli were adapted for investigating pareidolia, although more stimuli and a better standardization of OBJ might further improve the paradigm. The significant IE for both FACE and FLO suggested both these stimuli sets succeeded in eliciting a face-like processing. In contrast, we only partially managed to do so for OBJ. Possibly, the lack of standardization and homogeneity of OBJ category decreased IE for OBJ. Indeed, OBJ category consists of heterogeneous objects, contrary to FACE that consists of homogeneous stimuli. Interstimulus perceptual variance influences face and object processing, notably the N170 component (Ashworth et al., 2008; Thierry et al., 2007). To target categorical differences, a subsequent study was performed to standardize the visual properties within categories. Moreover, the general mean Accuracy of the task was high, suggesting the task was too easy. Adding more stimuli would adjust the task's difficulty, allow more improved measurement of performance, and detection of relationships with personality traits.

In sum, the current study certainly suffers from some limitations. Before concluding that the current results are chance observations, we rather argue that potentially stronger results can be gathered if the stimuli are improved (standardization of the OBJ category) and questionnaire data are used based on validated French versions (Sierro, Rossier & Mohr, 2016). Hence, we applied two modifications to our protocol in Study 2. First, we used improved AQ dimensions based on an analysis of AQ validity and reliability in a French-speaking population, similar to the one of our sample (Sierro, Rossier, & Mohr, 2016). Second, we tested whether more standardized stimuli would improve IE magnitude and relationships of paradelia proneness with personality.

4.3 STUDY 2

In Study 2, we tested IE for objects, FLOs and faces in undergraduate students differing in their scores on the sO-LIFE (Mason et al., 2005) and the AQ (Baron-Cohen et al., 2001). We expected that enhanced Mentalism tendencies (high positive schizotypy; low autistic mentalizing deficits) would relate to a stronger pareidolia bias (FLO IE) and lower Mentalism tendencies (low positive schizotypy; high autistic mentalizing deficits) would relate to a weaker pareidolia bias (FLO IE). These opposite tendencies might also emerge for face IE, but not for object IE. We did not expect that these IE differences relate to shared autistic and schizotypic traits (Dinsdale et al., 2013). Study 2 was similar to Study 1 apart from the following changes. We homogenized the perceptual features for the OBJ category to facilitate IE (Ashworth, Vuong, Rossion, & Tarr, 2008; Thierry, Martin, Downing, & Pegna, 2007). To do so, we used objects from one category (i.e. chairs). We aimed at having a better OBJ category to compare with FLO. Also, we used an improved factor structure for the French AQ (Sierro, Rossier & Mohr, 2016). We aimed at improving the reliability and validity of certain scales (e.g. Ima), hence the detection of possible diametrically opposite behavioural correlates between autistic and schizotypic traits. We detail these changes below.

4.3.1 MATERIAL AND METHODS

4.3.1.1 PARTICIPANTS

Our sample consisted of a new sample of 48 undergraduate students (36 women; age: mean±sd: 23.79±4.70). Recruitment was performed in the same way as we recruited the population for Study 1.

4.3.1.2 SELF-REPORT QUESTIONNAIRES

All details are similar to Study 1 (see Study 1, Materials & Methods, Self-report questionnaires), apart from the use of a 4-point Likert scale scoring and the adoption of an alternative factor structure to the AQ. In line with most recent studies (e.g. Lau, Kelly et al., 2013; Sierro, Rossier & Mohr, 2016), we did not dichotomize responses (Baron-Cohen et al., 2001), but allocated points according to the whole Likert scale. Concretely, "Definitely agree", "Agree", "Don't agree", and "Definitely disagree" responses respectively weigh 4, 3, 2 or 1 point(s) for regular items, and 1, 2, 3 or 4 point(s) for reversely formulated items. Moreover, we did not use the original Baron-Cohen et al. (2001) factor structure, but the one by Kloosterman et al. (2011; AQ-K),

because the latter best fitted a comparable French-speaking population (Sierro, Rossier & Mohr, 2016). Kloosterman et al. (2011)'s factor structure consists of fewer AQ items. Moreover, the 5 dimensions resemble independent factor structures (e.g. Lau, Kelly et al., 2013): Social Skills deficits (SocSki), Communication/Mindreading deficits (ComMind), Attention to Details (AttDet), Imagination deficits (Ima), and Routines/Repetitive Behaviour (RRBeh).

For the remainder of this chapter, we use AQ-K, PCDF-K and PCSF-K scores instead of AQ-BC, PCDF-BC and PCSF-BC scores, because the former scores (-K standing for Kloosterman et al., 2011 factor structure) were validated against the latter ones (-BC standing for Baron-Cohen et al., 2001 original factor structure) in a comparable population (Sierro, Rossier, & Mohr, 2016). Data from French-speaking undergraduates (n=921; Sierro, Rossier, & Mohr, 2016) showed AQ-BC scores and corresponding AQ-K scores measured comparable constructs, with the exception of the Comm-ComMind pair. Indeed, PCSF and PCDF scores computed from AQ-K and AQ-BC dimensions showed large positive correlations (Table 13). Similarly, four AQ-K dimensions (i.e. SocSki, AttDet, Ima, RRBeh) showed large positive correlations with the four corresponding AQ-BC ones (i.e. SocSki, AttDet, Ima, AttSwi) (Table 14). The small positive positive correlation between Comm and ComMind suggest that both dimensions may not account for a similar construct. Far from being a problem, using AQ-K's ComMind instead of AQ-BC's Comm seems an opportunity to better assess the Mentalism continuum posited by the diametrical model (Crespi & Badcock, 2006). AQ-K's ComMind, together with Ima, may best account for "empathizing" deficits or hypo-Mentalism, while positive schizotypy's UnEx accounts for hyper-Mentalism (Bishop et al., 2004; Sierro, Rossier, & Mohr, 2016).

Table 13. Pearson correlations between PCSF and PCDF scores stemming from dichotomized AQ-BC scoring, and 4-point Likert scale AQ-K.

	PCSF-BC	PCDF-BC
PCSF-K	**.891***	.199*
PCDF-K	.352*	**.940***

* p<.001
Large correlations (r's>.500) are boldfaced.
PCSF-BC: PC score of Shared Features with AQ-BC; PCDF-BC: PC score of Diametrical Features with AQ-BC
PCSF-K: PC score of Shared Features with AQ-K; PCDF-K: PC score of Diametrical Features with AQ-K

Table 14. Pearson correlations between dichotomized AQ-BC scores and 4-point Likert scale AQ-K scores.

		AQ-BC					
		Total	SocSki	Comm	Ima	AttSwi	AttDet
AQ-K	Total	**.834****	**.687****	**.601****	.447**	.490**	.281**
	SocSki	**.643****	**.786****	**.546****	.197**	.336**	.071**
	ComMind	.300**	.394**	.371**	.221**	.237**	-.264**
	Ima	.304**	.125**	.147**	**.670****	.128**	-.078**
	RRBeh	.410**	.181**	.311**	.056*	**.545****	.116**
	AttDet	.405**	.088**	.106**	.087**	.071**	**.776****

* $p<.05$; ** $p<.001$
Large correlations (r's>.500) are boldfaced.
Total score: Total AQ-BC/K score; SocSki: Social Skills deficits; Comm: Communication deficits; ComMind: Communication/Mindreading deficits; Ima: Imagination deficits; AttDet: Attention to Details; AttSwi:Attention Switching deficits; RRBeh: Routines/Repetitive Behaviours

4.3.1.3 VISUAL RECOGNITION TASK

4.3.1.3.1 STIMULI

Stimuli features were similar to those presented in Study 1 (Study 1, Material & Methods: Face Recognition Paradigm, Stimuli), apart from the increase in stimuli number and perceptual standardization of OBJ by using chairs. We particularly aimed to increase the number of stimuli from 40 to 80 stimuli per Category (240 in total) and to homogenize and standardize the OBJ Category (e.g. Kim et al., 2010; Shin et al., 2008). Thus, we could again present half of the stimuli as "old" items and the remaining half as the "new" items (40 per Category). For the OBJ Category, we decided to use chairs, because they have a regular, and predictable shape, which would unlikely elicit face pareidolia. For the FACE catergory, we selected face images from the Radboud data base, which we could access subsequent to an official request (Langner et al., 2010). For the FLOs Category, we googled the internet for images entering keywords like "face-like", "FLOs", "face pareidolia". For the OBJ Category too, we googled the internet for images entering keywords like "chair". We chose chairs presented from the front, depicted in a rightward orientation (Figure 4), so that their geometric features were well visible and and comparable between images.

Figure 4. Examples of chair stimuli used for the new OBJ Category.

4.3.1.3.2 PROTOCOL

The recognition paradigm used in Study 2 was similar to the one used in Study 1 (Study 1, Protocol), apart from being longer (more pictures were shown). As a result, the Study duration was longer (20 min).

4.3.1.4 MEASURES AND STATISTICS

Analyses were similar to those performed in Study 1 (Study 1, Materials & Methods, Data Analysis), apart from using AQ-K, PCSF-K and PCDF-K scores instead of AQ-BC, PCSF-BC, and PCDF-BC scores. Overall, mean accuracy was 69%±15%, and mean RTs were 1116.68±276.02 ms. For regression analyses and correlations, we used AQ-K (ComMind, Ima) scores instead of AQ-BC (Ima) scores (Sierro, Rossier & Mohr, 2016), as well as PCSF-K, and PCDF-K, instead of PCSF-BC, and PCDF-BC. In terms of normality, all variables' skew values were between +1/-1 range. Among questionnaires and behavioural variables, only IE:FACE-FLO for RTs was marginally skewed.

4.3.2 RESULTS

4.3.2.1 PARTICIPANTS

There were significantly more women (n=36) than men (n=12; χ^2=12, p=.001). Since assumptions of normality and homoscedasticity were not met, we performed age comparisons between Sexes using Wilcoxon rank-sum test. The mean age did not significantly differ between men (25.67±7.02) and women (23.17±3.55; W=150, p=0.113, z=0.14).

4.3.2.2 SELF-REPORT QUESTIONNAIRES

Questionnaires dimension scores (Table 15) were comparable to those reported from a larger French-speaking undergraduate samples for sO-LIFE scores (Sierro, Rossier, Mason et al., 2016), and AQ and PC scores (Sierro, Rossier, & Mohr, 2016). These scores did not differ from normative values, with the exception of lower sO-LIFE Global, CogDis, and ImpNon scores in our sample.

4.3.2.3 VISUAL RECOGNITION TASK PERFORMANCE

Descriptives for IE indices for Accuracy and RT are reported in Table 16. To investigate IE, we first conducted a one-way repeated measures ANOVA on IE:Accuracy with Category as within subject variable. There was a main effect of Category ($F(2,94)=27.57$, $p<.001$, $\eta_G^2=.30$) (see Figure 5a). Planned contrasts (1,0,-1; -1,2,-1) were used to test whether IE would linearly decrease as a function of decreasing faceness of stimuli (from FACE to OBJ). There was a significant linear decrease of IE across categories, but in an inverted fashion ($t(94)=-4.32$, $p<.001$, $r=.41$). The orthogonal contrast was also significant ($t(94)=6.55$, $p<.001$, $r=.56$). Post-hoc contrasts testing IE that would be maximal for faces and minimal for other categories (2,-1,-1) was not significant ($t(94)=0.47$, $p=.642$, $r=.05$). The contrast testing a maximal IE for FACE and minimal for FLO was significant (1,-1,0; $t(94)=3.51$, $p<.001$, $r=.36$). Tukey post hoc tests showed that IE:FACE (0.21±0.15) was significantly larger than both IE:FLO (-0.03±0.16) and IE:OBJ (0.08±0.12) (*p's* <.001). Surprisingly, IE:FLO was significantly *smaller* than IE:OBJ (0.08±0.12) ($p<.05$). To ascertain the existence of a significant IE for Accuracy, we conducted planned unilateral one-sample *t*-tests against $\mu=0$ for each Category. There were significant IEs for FACE ($t(47)=9.50$, $p<.001$, $d=1.39$) and OBJ ($t(47)=4.27$, $p<.001$, $d=0.62$), but not for FLO ($t(47)=-1.12$, $p=.865$, $d=-0.16$). The pareidolia index for Accuracy was significantly larger than 0 ($t(47)=7.12$, $p<.001$, $d=1.03$), suggesting larger reliance on a configural processing for FACE as compared to FLO.

Table 15. Descriptives for dimension scores of O-LIFE Short and AQ. Significant differences between Study 2 and Normative sample scores are in bold.

		Study 2			Normative Sample					
Instrument	Dimension	mean	sd	α	mean	sd	*t*	*df*	*p*	*d*
sO-LIFE	Global	11.85	6.95	.88†	14.45†	6.28	**-2.79**	**1094**	**.005**	**-0.41**
	UnEx	3.38	3.18	.83†	3.73†	2.59	-0.91	1094	.365	-0.13
	CogDis	4.10	2.62	.84†	5.53†	2.82	**-3.45**	**1094**	**<.001**	**-0.51**
	IntAn	2.02	1.56	.72†	2.14†	1.76	-0.46	1094	.643	-0.07
	ImpNon	2.35	1.88	.74†	3.04†	2.04	**-2.30**	**1094**	**.022**	**-0.34**
AQ-K	SocSki	15.23	3.35	.79††	16.13††	3.91	-1.56	967	.118	-0.23
	AttDet	11.52	3.31	.66††	11.43††	3.06	0.20	967	.843	0.03
	ComMind	10.08	2.35	.67††	10.10††	2.40	-0.06	967	.955	-0.01
	Ima	8.98	2.33	.49††	8.92††	2.36	0.17	967	.864	0.03
	RRBeh	10.71	1.89	.35††	11.12††	2.14	-1.30	967	.194	-0.19
PC score	PCSF-K	25.69	4.54	–	26.97††	5.02	*-1.73*	*967*	*.084*	*-0.26*
PC score	PCDF-K	15.49	7.10	–	16.90††	6.18	-1.53	967	.127	-0.23

mean=mean; sd=standard deviation; *t*=Student's *t*-test statistic; *p*= Student's *t*-test p-value; *d*= Cohen's *d* effect size
† normative sample based on *n*=1'048 respondents (Sierro, Rossier, Mason et al., 2016)
†† normative sample based on *n*=921 respondents (Sierro, Rossier & Mohr, 2016)
PCSF-K: PC score of Shared Features with AQ-K; PCDF-K: PC score of Diametrical Features with AQ-K
Global: total sO-LIFE score; UnEx: Unusual Experiences; CogDis: Cognitive Disorganization; IntAn: Introvertive Anhedonia; ImpNon: Impulsive Nonconformity
SocSki: Social Skills deficits; AttDet: Attention to Details; ComMind: Communication/Mindreading deficits; Ima: Imagination deficits; RRBeh: Routines/Repetitive Behaviours

Similarly, we conducted a one-way repeated measures ANOVA on IE:RT with Category as within-subject variable. There was a main effect of Category (ε_{HF}=.80, F(1.61,75.54)=15.99, p<.001, η_G^2=.17) (see Figure 5b). Planned contrast (1,0,-1) testing linear decrease of IE across categories was significant (t(94)=4.46, p<.001, r=.42) as was the orthogonal contrast (-1,2,-1) (t(94)=-3.48, p<.001, r=.34). The first significant post-hoc contrast tested that IE is maximal for

faces and minimum for the other categories (2,-1,-1) (t(94)=5.60, p<.001, r=.50). The second non-significant post-hoc contrast tested that the IE is bigger for FLO than for OBJ (t(94)=0.78, p=.436, r=.08). Post-hoc tests were conducted using pairwise using Bonferroni correction, since sphericity assumption was violated. IE:FACE (233±254) was significantly different from IE:FLO (32±116) and IE:OBJ (62±185) (p's <.001), but IE:FLO was not different from IE:OBJ (p>.05).

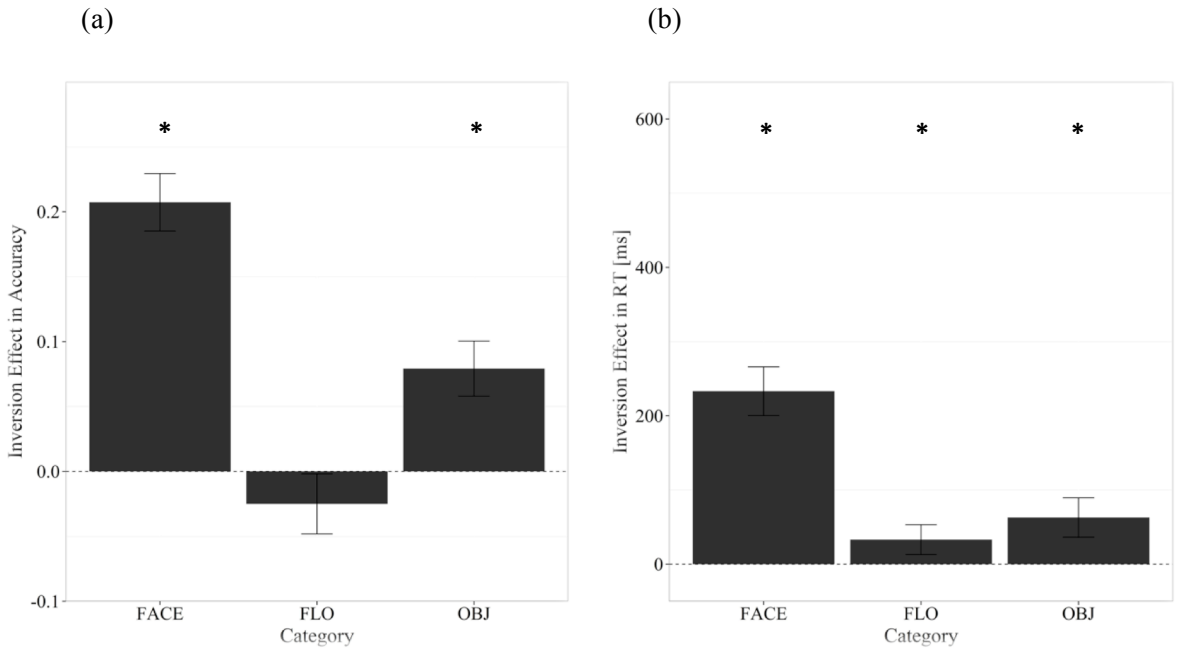
The one-sample t-tests against μ=0 on IEs were significant for FACE (t(47)=6.35, p<.001, d=0.93), FLO (t(47)=1.97, p=.027, d=.28), and OBJ (t(47)=2.35, p=.011, d=.34). The pareidolia index for RT was significantly larger than 0 (t(47)=5.15, p<.001, d=0.74), suggesting larger reliance on a configural processing for FACE as compared to FLO.

Table 16. Descriptives for Study 2 for Accuracy and RT performances, as a function of Category and Orientation.

	Accuracy		RT	
Measures	mean	sd	mean	sd
FACE:INV	0.57	0.14	1298.94	321.46
FACE:UPR	0.77	0.12	1065.85	264.39
FLO:INV	0.75	0.13	1079.79	237.23
FLO:UPR	0.73	0.13	1046.8	227.51
OBJ:INV	0.63	0.14	1135.79	250.13
OBJ:UPR	0.71	0.14	1058.88	233.62
IE:FACE	0.21	0.15	225.48	230.35
IE:FLO	-0.02	0.16	32.99	116.03
IE:OBJ	0.08	0.13	62.86	185.03
IE:FACE-FLO	0.23	0.23	200.10	269.00

FACE: face stimuli; FLO: face-like object stimuli; OBJ: object stimuli
INV: inverted orientation; UPR: upright orientation; IE: Inversion effect
IE:FACE-FLO: Pareidolia Index

Figure 5. Inversion effect for (a) Accuracy and (b) RT. Error bars represent standard errors with correction for repeated measures. Stars indicate IEs that are significantly different from 0 at one-sample *t*-tests (* *p*'s<.050).



4.3.2.4 RELATIONSHIPS BETWEEN PERSONALITY SCORES AND VISUAL RECOGNITION PERFORMANCE

We report regression results for pareidolia indices and personality variables in Table 17. For Accuracy, neither the regression model with PC scores (R^2=.048, F(2,45)=1.13, p=.333), nor the one with UnEx, ComMind and Ima (R^2=.086, F(3,44)=1.38, p=.261) did significantly predict pareidolia effects (IE:FACE-FLO). Yet, larger UnEx scores tended to associate with a larger pareidolia effect in Accuracy (i.e. smaller IE:FACE-FLO), as represented by Figure 6. Likewise, for RTs, neither the regression model with PC scores (R^2=.007, F(2,45)=0.156, p=.856), nor the one with UnEx, ComMind and Ima (R^2=.024, F(3,44)=0.368, p=.777) did significantly predict pareidolia effects in RT (IE:FACE-FLO).

Table 17. Regression of Accuracy and RT indices measuring similarity of IE between FACE and FLO (pareidolia effect), and between FLO and OBJ, as a function of PCDF-K and PCSF-K scores. Significant findings are indicated in bold

	Outcomes	Predictors	R^2	β	*t*(46)	*p*	IC 2.5 %	IC 97.5 %
Accuracy	IE:FACE-FLO		.048					
		PCSF-K		-0.063	-0.427	.671	-0.018	0.012
		PCDF-K		-0.202	-1.381	.174	-0.016	0.003
Accuracy	IE:FACE-FLO		.086					
		UnEx		*-0.311*	*-2.000*	*.052*	*-0.044*	*0.0001*
		ComMind		-0.110	-0.687	.496	-0.042	0.021
		Ima		-0.043	-0.287	.776	-0.033	0.025
RT	IE:FACE-FLO		.007					
		PCSF-K		-0.002	-0.014	.989	-17.948	17.705
		PCDF-K		0.083	0.056	.581	-8.262	14.558
RT	IE:FACE-FLO		.024					
		UnEx		0.001	0.009	.993	-27.275	27.527
		ComMind		-0.019	-0.114	.910	-40.504	36.156
		Ima		-0.15	-0.973	.336	-53.281	18.582

° $p \leq .100$; * $p < .050$;** $p < .001$

PCSF-K: PC score of Shared Features with AQ-K; PCDF-K: PC score of Diametrical Features with AQ-K

UnEx: Unusual Experiences; ComMind: Communication/Mindreading deficits; Ima: Imagination deficits

IE:FACE-FLO: Pareidolia Index

Figure 6. Scatter plot of the trend relationship between pareidolia index for Accuracy (IE:FACE-FLO), as a function of positive schizotypy (UnEx) for Study 2

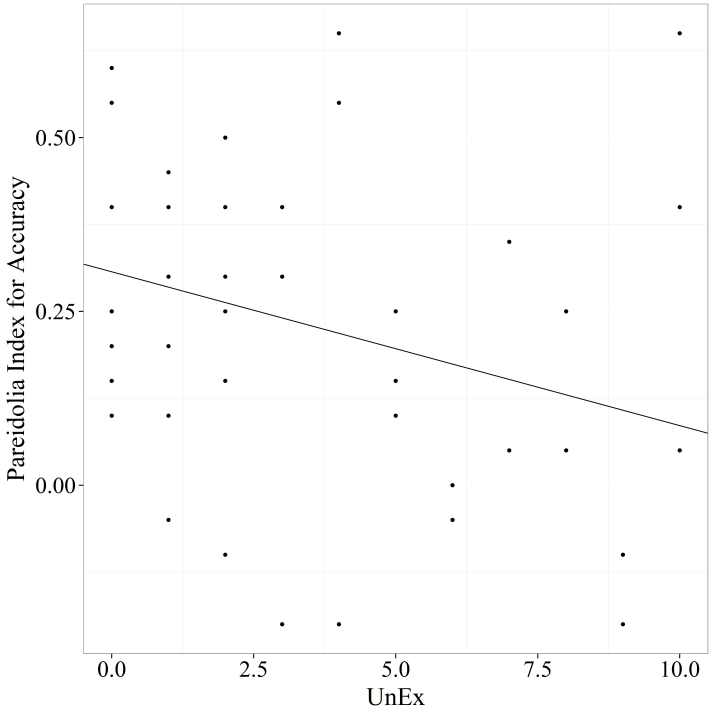


Table 18. Regressions with IE indices and raw behavioural variables as outcome measures and UnEx, ComMind and Ima as predictor variables. Significant findings are indicated in bold.

		β's for Accuracy				β's for RT		
	R^2	UnEx	ComMind	Ima	R^2	UnEx	ComMind	Ima
IE:FACE	.10	**-.325***	-.176	-.052	.04	.003	.075	-.198
IE:FLO	.02	.137	-.010	.012	.04	.003	.209	-.087
IE:OBJ	.06	-.124	.174	-.036	*.15°*	-.188	.185	**-.293***
FACE:UPR	.05	-.012	.062	-.236	*.15°*	.116	.164	**.319***
FACE:INV	.13	**.349***	.246	-.143	.05	.098	.195	.106
FLO:UPR	*.14°*	**.394***	.090	.055	.04	-.019	.193	.014
FLO:INV	.05	.231	.104	.041	.08	-.017	*.287°*	-.029
OBJ:UPR	.07	*.294°*	.096	-.004	.13	.169	.072	**.310***
OBJ:INV	**.20***	**.418***	-.062	.030	.07	.047	.216	.126

° $p \leq .100$; * $p < .050$;** $p < .001$

UnEx: Unusual Experiences; ComMind: Communication/Mindreading deficits; Ima: Imagination deficits

We report the findings from the regression analyses with IE variables and raw behavioural variables as outcome variables and UnEx, ComMind and Ima as predictor variables in Table 18. For Accuracy, all regression models were non-significant (*p's* >.05), with the exception of one model. The model for OBJ:INV was significant (R^2=.20, F(3,44)=3.59, p=.021). Larger UnEx scores significantly associated with larger Accuracy for FLO:UPR, and OBJ:INV, and did so as a trend for OBJ:UPR. Moreover, larger UnEx scores significantly associated with smaller IE for FACE, because of their association with a significantly larger Accuracy for FACE:INV. We note the model for FLO:UPR showed a trend toward significance (R^2=.14, F(3,44)=2.38, p=.082). For RTs, all regression models were non-significant (*p's* >.05). However, larger Ima scores significantly associated with reduced IE for OBJ, largely owing to significantly longer RTs for OBJ:UPR. Likewise, larger Ima scores associated with significantly longer RTs for FACE:UPR, and larger ComMind scores tended to associated with longer RTs for FLO:INV.

4.3.3 SHORT DISCUSSION

In Study 2, we expected significant IEs for all three categories, decreasing from FACE, through FLO to OBJ's standardized chairs. Additionnaly, we expected that pareidolia effect (i.e. IE:FACE-FLO) would be significant and that its magnitude would reflect the diametrical relationships between positive schizotypy hyper- and autistic hypo-Mentalistic traits.

Contrary to our expectations, we did neither observe a IE:FLO Accuracy, nor a decrease of this IE as a function of faceness of stimuli, as shown in Study 1. We replicated the main effects of Category on Accuracy and RT. We found significant (i) IE:Accuracy for FACE and OBJ (Chairs), but not for FLO, and (ii) IE:RT for all three stimulus categories (see also Study 1). In both instances, IE did not decrease as a function of face resemblance. Consistent with the results of Study 1, the IE:FACE (Accuracy and RT) were largest as compared to the other IEs, and IE:RT did not differ between FLO and OBJ. We did not expect that IE:Accuracy would be significantly *larger* for OBJ than FLO. This observation indicates that the standardization of OBJ (chairs of comparable shape and orientation) enhanced the IE for OBJ in Accuracy. Rassuring, the doubling of stimuli number decreased overall Accuracy to levels comparable to those reported in the previous literature (e.g. 70%; Kim et al., 2010).

The current results did not show that "shared", "diametrical" or Mentalistic traits predict pareidolia effect, although some indications occurred for UnEx. Pareidolia effect (i.e. reduced difference between Face and FLO performances) did not associate with Mentalistic (PCDF-K), or negative traits (PCSF-K). Autistic mentalizing deficit traits (ComMind, Ima) did not significantly

associate with any variation of pareidolia effect. Larger UnEx scores, however, tended to associate with larger pareidolia effect in Accuracy (i.e. smaller pareidolia index, IE:FACE-FLO). As we will see in the next paragraph, yet again (see Short discussion of Study 1), the additional regression models conducted on IE and raw behavioural variables questioned the fact that personality traits related to pareidolia-proneness.

Instead of pareidolia-proneness, positive schizotypy might link to particular basic visual processing, independent from Mentalism. Larger UnEx scores did not associate with a larger IE:FLO, as would be expected from a genuine pareidolia effect. Instead, larger UnEx scores associated with smaller IE:FACE in Accuracy, owing to better accuracy for INV standardized stimuli (i.e. FACE, OBJ), but not the heterogeneous FLO. These observations suggest that positive schizotypy may reflect a processing bias for local information (or with regards to the current stimuli, a featural processing strategy). This suggestion would be expressed as an advantage for processing standardized stimuli rather than unstandardized ones (FLO). Interestingly, both smaller IE and over-reliance on featural information or local bias were observed in schizophrenia patients, notably as a function of positive symptoms (Chambon et al., 2006; Joshua & Rossel, 2009).

Autistic traits too might link to particular basic visual processing not relevant to positive schizotypic traits, independent from Mentalism. Ima and ComMind were both unrelated to pareidolia effect, and did not oppose to positive schizotypy. We found that (i) Ima associated with configural processing deficits and (ii) ComMind showed indices of intact configural processing. With regard to the first finding, larger Ima scores associated with a decreased IE for OBJ in RTs, and slower RTs for UPR conditions of standardized stimuli (FACE; OBJ). These associations suggest that enhanced imagination deficits may associate with configural processing deficits expressed in a difficulty using predictable configurations of standardized stimuli (FACE, OBJ:Chairs), as has already been discussed for populations showing high autistic traits (Rhodes et al., 2013). With regard to the second finding, and contrary to results for both UnEx and Ima, higher ComMind deficits associated with a trend for longer RTs for FLO:INV stimuli, suggestive of a reliance on configural processing. This conjecture is compatible with reports of intact configural processing in ASD individuals and those with high autistic traits (Hedley et al., 2014; Stevenson et al., 2016; Weigelt et al., 2012). Contrary to Study 1, we did not notice any opposition between positive schizotypy and autistic mentalizing deficit traits that would bring support to the diametrical opposition in social cognition (Crespi & Badcock, 2008).

4.3.4 LIMITATIONS

We discuss several limitations relative to questionnaires, the population we tested, and the behavioural data including the stimuli used that all may contribute to a lack of associations between personality traits and pareidolia effect. The AQ-K is an improved version to the AQ-BC, at least in our French speaking Swiss population (Sierro, Rossier & Mohr, 2016). According to this recent study, however, the resultant dimensions still suffer from limited reliability and the factor structure needs replication. Also, we here tested a sample in which women were overrepresented, while this was not the case in Study 1. This unequal balance between women and men might have biased overall behavioural performances as well as scores in our self-report questionnaires. Having tested more women than men in the second study could have biased questionnaire scores as well as performance towards overall higher positive schizotypy scores (Sierro, Rossier, Mason et al., 2016) and a hyper-Mentalism cognitive processing style (Crespi & Badcock, 2008), respectively. To get a clearer view of sampling biases, we will later compare psychometric and behavioural variables for all 3 Studies (see Chapter 4.5.).

When considering limitations regarding our behavioural data including the stimuli, we would like to highlight that a lack of within category homogeneity of the stimuli might be partly responsible for the smaller IE:FLO in Study 2. After standardizing and homogenizing individual items within the OBJ Category (using images of chairs of similar shape and orientation), performance for OBJ items resulted in a significant IE on Accuracy, which was even larger than the IE:FLO. In contrast, we observed no significant IE:FLO, although stimuli were identical to Study 1. Instead of a decreasing IE as a function of faceness of stimuli, we might have observed that IE are determined, at least partly, by standardization and within Category homogeneity of the category-specific stimuli (Ashworth et al., 2008; Thierry et al., 2007). Hence, it is possible that standardization of FLO stimuli might also lead to an increase in IE magnitude, as seen for OBJ stimuli when comparing IE between Studies 1 and 2.

To increase FLO's IE in Accuracy and enhance the likelihood of measuring pareidolia effects, we decided to do the same for stimuli of the FLO category as we did in Study 2 for the stimuli of the OBJ category. Consequently, we needed again individual items that belong to a homogenous category as is the case for the individual pictures belonging to FACES and OBJ (i.e. chairs). We decided on front views of cars, because cars are omnipresent in our environment emcompassing by default a certain degree of experience, learning, exploration and encoding routine, but also faceness. Car fronts, indeed, elicited face-like processing and visual exploration biases (Rossion et al., 2003; Windhager et al., 2010), as should be expected when testing for a

pareidolia effect. Hence, we googled for car front pictures so that the FLO Category would be equally homogenous to the other categories (e.g. size, gray-scaled, centered).

4.4 STUDY 3

For Study 3, we formulated the same predictions and applied the same methodology as already done for Study 2. Yet, we improved the homogeneity of the items belonging to the FLO category. We homogenized the perceptual features within the FLO category to facilitate IE (Ashworth, et al., 2008; Thierry et al., 2007). We used items belonging to the same category (i.e. car fronts), likely to elicit a pareidolia effect. Thus, we could expect a comparable homogeneity of individual items per category for the FACE, FLO, and OBJ category. Thus, we also enhanced the likelihood to detect associations between pareidolia liability and Mentalism. In particular, we expected a stronger pareidolia effect (IE: FLO) as a function of "diametrical" rather than "shared" autistic and schizotypic traits, i.e. a stronger pareidolia effect with increasing Mentalistic scores (i.e. positive schizotypy, autistic mentalizing deficits).

4.4.1 PARTICIPANTS

Our sample consisted of a new sample of 48 undergraduate students (20 women; age: mean±sd: 20.90±2.20). We recuited participants in a comparable way to our recruitement for Studies 1 and 2.

4.4.2 SELF-REPORT QUESTIONNAIRES

Instruments and scoring was analogue to Study 2 (see Study 2, Materials & Methods, Self-Report Questionnaires).

4.4.3 VISUAL RECOGNITION TASK

4.4.3.1 STIMULI

Stimuli and stimulus features were similar to those presented in Study 2 (Study 2: Material & Methods: Visual Recognition Task: Stimuli), apart from using car front stimuli for the FLO

category, because of the face-like behavioural and neural correlates they elicit (Kühn et al., 2014; Windhager et al., 2008). We assembled 80 car front stimuli (browsing the internet using keywords such as "front car") of various types, brands and epoch (Figure 7). All images were turned to grey-scaled images, centered, and resized to fit to 300 pixels canvas, and to occupy a surface comparable to surfaces occupied by faces. The car fronts were centered to the centre between front lights, analogue to faces' nasion. Front lights of cars seem to guide visual exploration comparable to what eyes in faces do (Windhager et al., 2010). We additionally removed logos, writings, numbers or brand logos to prevent confounding verbal, numeric or semantic memorization strategies (see Figure 7). To further enhance the comporablity of items within and between catgories, we transformed stimuli to gray-scale, resized, and centered images using The GIMP 2.6.12. (http: //www.gimp.org) and Adobe Photoshop Elements 8 (Adobe Systems, San Jose, California). We showed each FLO (car front image) in the centre of a 300 pixels large circle (diameter of 10.6 cm on the screen). This circle was shown in the centre of a 480×480 pixels large dark grey square (13×13 cm on the screen) (see Figure 1).

Figure 7. Examples of car front stimuli used for the FLO category in Study 3.

4.4.3.1.1 PROTOCOL

The recognition paradigm used in Study 2 was reprogrammed on PsychoPy. All FACE and OBJ stimuli were the same as in Study 2. The only new stimuli were the new FLO stimuli. The overall study duration was similar to Study 2 (20 min). Also, Study 3 was run on computers running on Windows, with a different Eizo screen (diag. 60 cm), which explains the small difference in display size of stimuli (i.e. stimuli sizes 13 × 13cm instead of 13.98 × 13.98 cm; circle diameter 10.6 cm instead of 11.4 cm).

4.4.3.2 MEASURES AND STATISTICS

Analyses were similar to those performed in Study 2 (see Study 2, Materials & Methods, Self-report questionnaires, and Data Analyses), apart from the following differences. Overall, mean accuracy was 70%±11%, and mean RTs were 1899.15±356.51 ms. Variable skewedness were in the ±1 range for all variables apart for RT's for FACE:UPR and OBJ:UPR.

4.4.4 RESULTS

4.4.4.1 PARTICIPANTS

Our 48 participants (age: 20.90±2.20) comprised 20 women (age: 19.95±1.79) and 28 men (age: 21.57±2.23). There was a significantly equal number of women and men (χ^2=1.33, p=.248). Age normality, but not equality of variance, was respected. Thus, we performed bilateral Welch test on Age, with Sex as between-subject variable. Women were significantly younger than men (t(45.32)=-2.79, p=.008, d=-0.82), of about one and a half years.

4.4.4.2 SELF-REPORT QUESTIONNAIRES

Questionnaire dimension scores were computed for each dimension (Table 19). The personality questionnaire subscale scores were comparable to those reported from larger French-speaking undergraduate samples (sO-LIFE: Sierro, Rossier, Mason et al., 2016; AQ and PC scores: Sierro, Rossier & Mohr, 2016). All questionnaire scores were comparable to those from the normative data (Table 19).

Table 19. Descriptives for Study 3 for dimension scores of O-LIFE Short and AQ for each dimension. There were no significant differences between Study 3 and Normative sample scores.

		Study 3			Normative Sample					
Instrument	Dimension	mean	sd	α	mean	sd	*t*	*df*	*p*	*d*
sO-LIFE	Total	15.21	6.86	.88†	14.45†	6.28	0.82	1094	.414	0.12
	UnEx	3.67	3.00	.83†	3.73†	2.59	-0.16	1094	.876	-0.02
	CogDis	5.81	2.73	.84†	5.53†	2.82	0.67	1094	.501	0.10
	IntAn	2.23	1.88	.72†	2.14†	1.76	0.35	1094	.730	0.05
	ImpNon	3.5	2.37	.74†	3.04†	2.04	1.52	1094	.130	0.22
AQ-K	SocSki	15.98	4.74	.79††	16.13††	3.91	-0.26	967	.798	-0.04
	AttDet	11.75	3.88	.66††	11.43††	3.06	0.70	967	.487	0.10
	ComMind	10.35	2.74	.67††	10.10††	2.40	0.70	967	.485	0.10
	Ima	8.75	2.56	.49††	8.92††	2.36	-0.48	967	.628	-0.07
	RRBeh	11.46	2.64	.35††	11.12††	2.14	1.06	967	.290	0.16
PC score	PCSF-K	27.15	6.10	–	26.97††	5.02	0.24	967	.811	0.04
PC score	PCDF-K	18.05	6.84	–	16.90††	6.18	1.25	967	.212	0.19

mean=mean; sd=standard deviation; *t*=Student's t-test statistic; *p*= Student's t-test p-value; *d*= Cohen's *d* effect size
† normative sample based on *n*=1'048 respondents (Sierro, Rossier, Mason et al., 2016)
†† normative sample based on *n*=921 respondents (Sierro, Rossier & Mohr, 2016)
PCSF-K: PC score of Shared Features with AQ-K; PCDF-K: PC score of Diametrical Features with AQ-K
Global: total sO-LIFE score; UnEx: Unusual Experiences; CogDis: Cognitive Disorganization; IntAn: Introvertive Anhedonia; ImpNon: Impulsive Nonconformity
SocSki: Social Skills deficits; ComMind: Communication/Mindreading deficits; Ima: Imagination deficits; AttDet: Attention to Details; RRBeh: Routines/Repetitive Behaviours

4.4.4.3 VISUAL RECOGNITION TASK PERFORMANCE

Descriptives of IE indices for Accuracy and RT are reported in Table 20. To investigate IE, we first conducted a one-way repeated measures ANOVA on IE:Accuracy with Category as within subject variable. There was a main effect of Category ($F(2,94)=45.49$, $p<.001$, $\eta_G^2=.65$) (see Figure 8a). Planned contrasts (1,0,-1; -1,2,-1) were used to test whether IE would linearly decrease as a function of decreasing faceness of stimuli (from Face to OBJ). There was a significant linear decrease of IE across categories, in the expected direction ($t(94)=8.33$, $p<.001$, $r=.65$). The orthogonal contrast was also significant ($t(94)=4.65$, $p<.001$, $r=.43$). Post-hoc contrasts showed that the IE was maximal for faces and minimal for the other categories (2,-1,-1) proved to be significant ($t(94)=4.89$, $p<.001$, $r=.45$), whereas the contrast testing a maximal IE for faces and minimal for OBJ was not significant (1,-1,0; $t(94)=-0.14$, $p=.890$, $r=.01$). Post hoc comparisons showed that IE:FACE (0.12±0.08) was significantly larger than both IE:FLO (-0.02±0.11) and IE:OBJ (-0.02±0.08) (*p's* <.001). IE:FLO (-0.02±0.11) was not different from IE:OBJ (-0.02±0.08) ($p>.05$). The one-sample t-tests against μ=0 showed a significant IE for FACE ($t(47)=10.29$, $p<.001$, $d=3.00$), but none for OBJ ($t(47)=-1.00$, $p=.839$, $d=-0.29$) or FLO ($t(47)=-1.59$, $p=.940$, $d=-0.46$). The pareidolia index for Accuracy was well above zero ($t(47)=7.89$, $p<.001$, $d=1.14$), suggesting larger reliance on a configural processing strategy for FACE as compared to FLO stimuli.

Similarly, we conducted a one-way repeated measures ANOVA on IE:RT with Category as within-subject variable. There was a main effect of Category ($\varepsilon_{GG}=.88$, $F(1.76,82.74)=37.65$, $p<.001$, $\eta_G^2=.25$) (see Figure 8b). The planned contrast (1,0,-1) testing for a linear decrease of IE across categories was significant ($t(94)=7.37$, $p<.001$, $r=.61$), so was the orthogonal contrast (-1,2-1, $t(94)=-4.58$, $p<.001$, $r=.43$). Post-hoc contrast testing for a maximal IE for faces and a minimum IE for the other categories (2,-1,-1) was significant ($t(94)=8.67$, $p<.001$, $r=.67$), whereas the contrast testing for a bigger IE for FLO as compared to OBJ was not significant ($t(94)=0.28$, $p=.782$, $r=.03$). We conducated post-hoc comparisons using pairwise t-tests with Bonferroni correction, since sphericity assumptions were violated. IE:FACE (307±288) was significantly different from IE:FLO (21±197) and IE:OBJ (31±194) (*p's* <.001), but IE:FLO was not different from IE:OBJ ($p>.05$). The unilateral one-sample t-tests against μ=0 showed a significant IE for FACE ($t(47)=7.40$, $p<.001$, $d=2.16$), but none for the FLO ($t(47)=0.74$, $p=.232$, $d=0.11$) and OBJ ($t(47)=1.12$, $p=.135$, $d=0.33$) categories. The pareidolia index for RT was significantly larger than 0 ($t(47)=7.39$, $p<.001$, $d=1.07$), suggesting larger reliance on a configural processing strategy for FACE as compared to FLO.

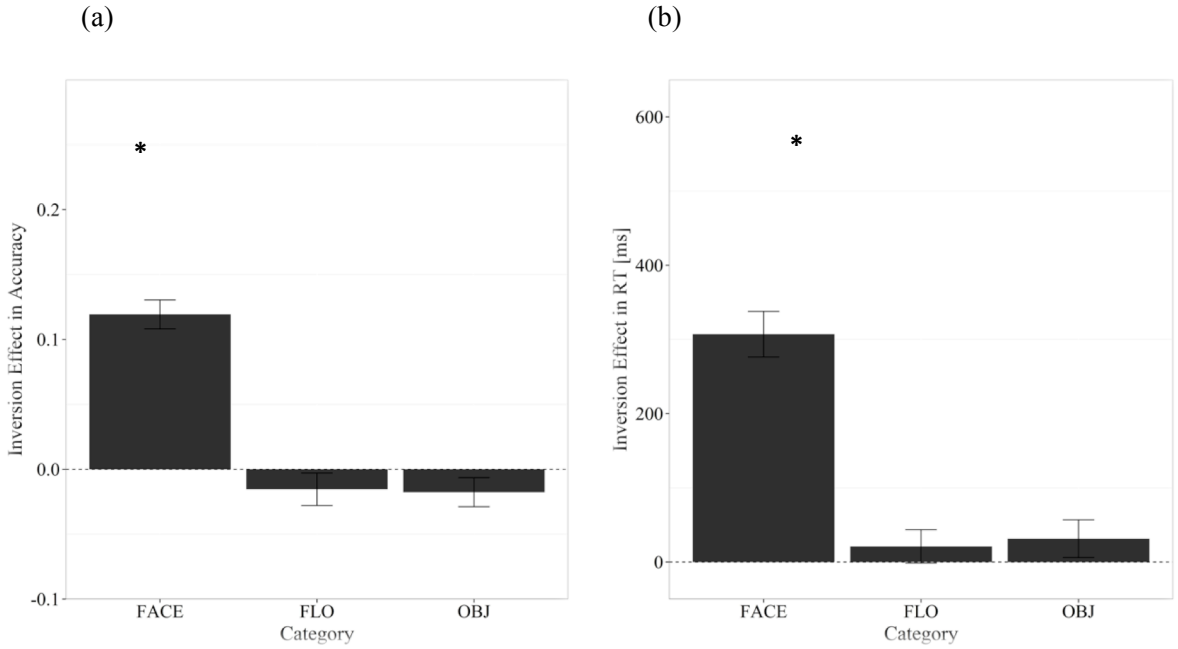
Table 20. Descriptives for Study 3 for Accuracy and RT performances, as a function of Category and Orientation.

	Accuracy		RT	
Measures	mean	sd	mean	sd
FACE:INV	0.71	0.09	2084.88	477.03
FACE:UPR	0.83	0.08	1777.81	290.49
FLO:INV	0.66	0.08	2021.53	330.35
FLO:UPR	0.64	0.10	2000.51	297.65
OBJ:INV	0.69	0.09	1770.8	272.86
OBJ:UPR	0.68	0.11	1739.39	270.31
IE:FACE	0.12	0.08	307.08	287.56
IE:FLO	-0.02	0.11	21.02	196.85
IE:OBJ	-0.02	0.08	31.41	194.60
IE:FACE-FLO	0.13	0.12	286.06	268.25

mean=mean; sd=standard deviation

FACE: face stimuli; FLO: face-like object stimuli; OBJ: object stimuli
INV: inverted orientation; UPR: upright orientation; IE: Inversion effect
IE:FACE-FLO: Pareidolia Index

Figure 8. Inversion effect for (a) Accuracy and (b) RT (with OBJ:Chairs, and FLO:Car Fronts). Error bars represent one standard error with correction for repeated measures. Stars indicate IEs that are significantly different from 0 when performing one-sample t-tests (* *p*'s<.050).



4.4.4.4 RELATIONSHIPS BETWEEN PERSONALITY SCORES AND VISUAL RECOGNITION PERFORMANCE

The regression models were non-significant (Table 21), with the exception of one model showing a trend. This trend was observed for Accuracy measures, i.e. for a model in which pareidolia indices in accuracy were predicted by PC scores (R^2=.13, F(2,45)=3.21, p=.050). Larger PCDF-K scores associated with significantly *smaller* pareidolia effect for Accuracy (i.e. larger pareidolia index, IE:FACE-FLO), as represented by Figure 9. The analogue regression model with UnEx, ComMind and Ima as predictors was not significant (R^2=.12, F(3,44)=1.984, p=.130). Yet, larger ComMind scores significantly associated with a larger pareidolia effect (i.e. smaller pareidolia index, IE:FACE-FLO), as represented by Figure 10. For RTs, the model predicting a pareidolia effect in RT with PC scores as predictors was non-significant (R^2=.01, F(2,45)=0.25, p=.782), as were analogue ones with UnEx, ComMind and Ima as predictors (R^2=.08, F(3,44)=1.30, p=.286). We observed a trend of larger Ima scores to associate with a larger pareidolia effect in RTs (i.e. smaller pareidolia index, IE:FACE-FLO), as represented by Figure 11.

Table 21. Regression of Accuracy and RT pareidolia effect as outcome measures, as a function of PCDF-K and PCSF-K, ComMind and Ima scores, as predictors.

	Outcomes	Predictors	R^2	β	*t*(46)	*p*	IC 2.5 %	IC 97.5 %
Accuracy	IE:FACE-FLO		*.13*°					
		PCSF-K		-.209	-1.484	.145	-0.010	0.001
		PCDF-K		**.313**	**2.227**	**.031**	**0.001**	**0.010**
Accuracy	IE:FACE-FLO		.12					
		UnEx		.120	0.816	.419	-0.007	0.016
		ComMind		**-.310**	**-2.151**	**.037**	**-0.026**	**-0.001**
		Ima		.000	0.002	.998	-0.013	0.013
RT	IE:FACE-FLO		.01					
		PCSF-K		.051	0.341	.735	-0.011	0.015
		PCDF-K		.085	0.567	.574	-0.008	0.015
RT	IE:FACE-FLO		.08					
		UnEx		-.129	-0.881	.383	-0.038	0.015
		ComMind		.009	0.059	.953	-0.028	0.030
		Ima		*-.267*	*-1.826*	*.075*	*-0.059*	*0.003*

° $p \leq .100$; * $p < .050$;** $p < .001$
PCSF-K: PC score of Shared Features with AQ-K; PCDF-K: PC score of Diametrical Features with AQ-K
UnEx: Unusual Experiences; ComMind: Communication/Mindreading deficits; Ima: Imagination deficits
IE:FACE-FLO: Pareidolia Index

Figure 9. Scatter plot of Accuracy pareidolia effect (IE:FACE-FLO), as a function of "diametrical" or Mentalism score (PCDF-K) for Study 3.

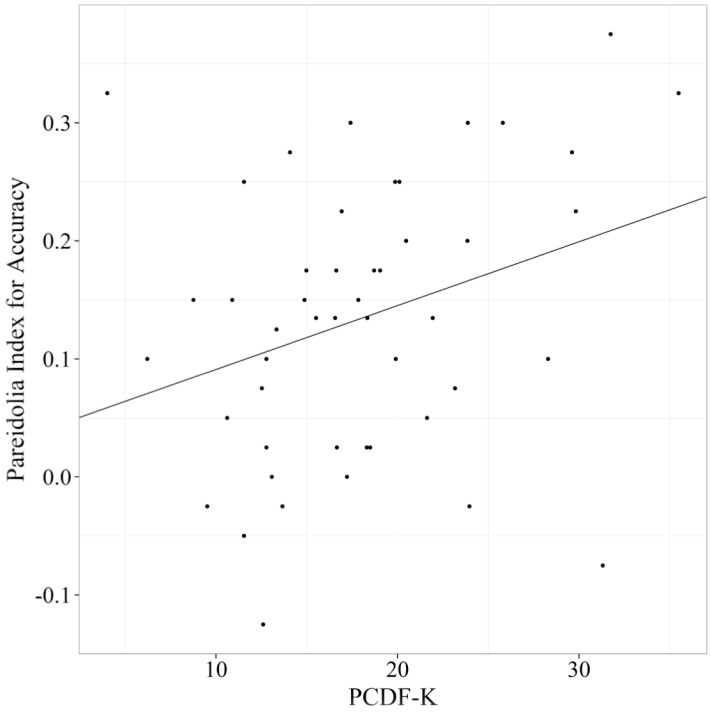


Figure 10. Scatter plot of Accuracy pareidolia effect (IE:FACE-FLO), as a function of autistic mentalizing deficit in communication/mindreading (ComMind) for Study 3.

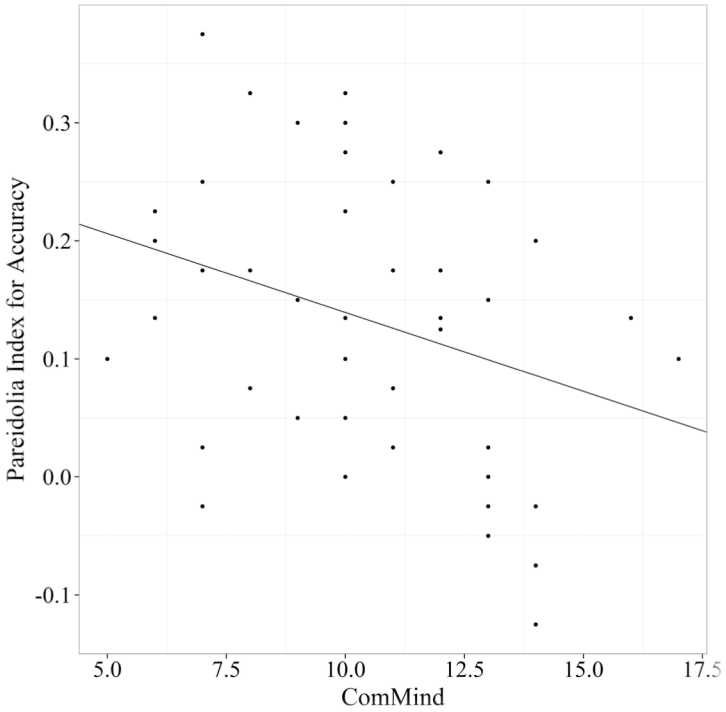
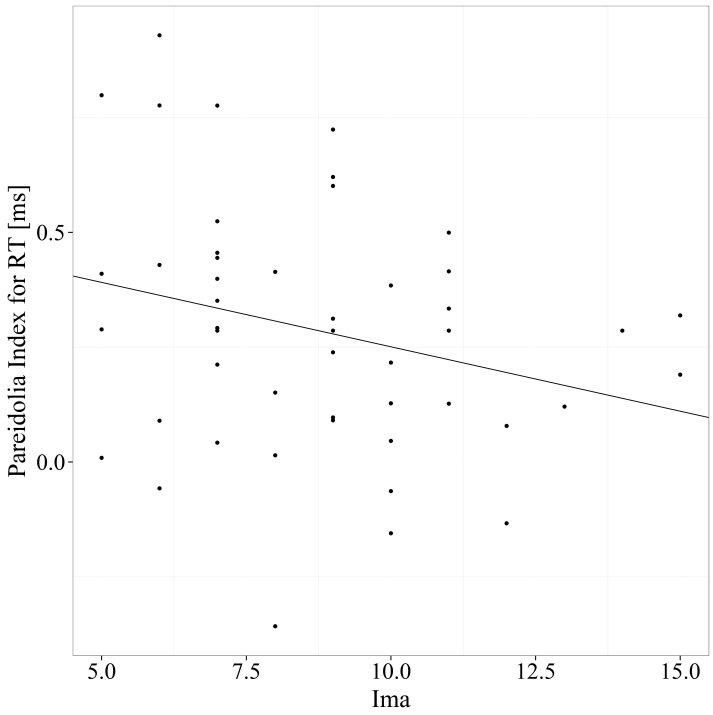


Figure 11. Scatter plot of RT pareidolia effect (IE:FACE-FLO), as a function of autistic mentalizing deficit in social imagination (Ima) for Study 3.



The analogue regressions with IE variables and raw behavioural variables as outcome variables and UnEx, ComMind and Ima as predictors are shown in Table 22. For Accuracy, all regression models were non-significant (all *p's* >.05). Yet, a trend was observed for the model of OBJ:UPR (R^2=.14, F(3,44)=2.33, p=.087). Larger ComMind scores significantly associated with larger Accuracy for OBJ:UPR and INV, at the expense of a tendency to longer RTs for OBJ:INV. Larger UnEx scores significantly associated with smaller Accuracy for FACE:UPR and FLO:UPR. For RTs, all regression models were non-significant (*p's* >.05). Larger Ima scores tended to associate with shorter RTs for FLO:UPR.

Table 22. Regressions with IE indices and raw behavioural variables as outcome measures and UnEx, ComMind and Ima as predictor variables.

		β's for Accuracy				β's for RT		
	R^2	UnEx	ComMind	Ima	R^2	UnEx	ComMind	Ima
IE:FACE	.05	-.122	-.212	.053	.05	-.193	.029	-.142
IE:FLO	.10	-.222	.184	.040	.04	-.107	.030	.157
IE:OBJ	.01	-.072	.064	-.069	.08	.140	.164	-.211
FACE:UPR	.11	**-.313***	-.045	-.128	.06	-.151	.182	-.051
FACE:INV	.08	-.187	.149	-.170	.07	-.208	.128	-.117
FLO:UPR	.12	**-.301***	.146	.002	.08	-.151	.066	*-.254°*
FLO:INV	.01	-.090	-.052	-.048	.06	-.200	.078	-.135
OBJ:UPR	*.14°*	-.154	**.315***	-.127	.06	-.154	.154	.071
OBJ:INV	.12	-.121	**.315***	-.092	.08	-.053	*.269°*	-.080

° $p \leq .100$; * $p < .050$;** $p < .001$
UnEx: Unusual Experiences; ComMind: Communication/Mindreading deficits; Ima: Imagination deficits
FACE: face stimuli; FLO: face-like object stimuli; OBJ: object stimuli
INV: inverted orientation; UPR: upright orientation; IE: Inversion effect

4.4.5 SHORT DISCUSSION

In Study 3, we expected significant IEs for all three categories, decreasing from FACE, through FLO's car fronts to OBJ's standardized chairs. Additionnaly, we expected that pareidolia effect (i.e. IE:FACE-FLO) would be significant and that its magnitude would reflect the diametrical relationships between positive schizotypy hyper- and autistic hypo-Mentalistic traits.

We replicated the IE:FACE for Accuracy and RTs (Studies 1 and 2), but not for the IEs for FLO (Study 1 for Accuracy; Studies 1 and 2 for RTs) or OBJ (Study 2 for Accuracy; Studies 1 and 2 for RTs). The overall performance replicated previous performances (Study 2; Kim et al., 2010). The IE for the FACE category was present for both Accuracy and RT and was larger than those for the FLO and OBJ categories. We had expected that the homogenization of the stimuli in the FLO category (car fronts) would increase the IE in Study 3, as it seemed having done for OBJ (chairs) in Study 2. Yet in Study 3, neither the car nor the chair stimuli resulted in significant IE (Accuracy, RT). Hence, the inconsistency with which non-FACE stimuli yield face-like IE (pareidolia-

proneness) casts doubt on this paradigm's efficiency in measuring pareidolia. Obviously, this does not exclude this paradigm's potential in assessing pareidolia-proneness as a function of participants' personality dimensions. We observed distinct performances in basic visual processing with hyper-Mentalistic positive schizotypy associating with impaired face(-like) configural processing, and hypo-Mentalistic autistic traits associating with preserved configural processing and advantage for non-face(-like) stimuli processing.

In line with our expectations, the "diametrical" (PCDF-K) but not the "shared" traits (PCSF-K) significantly predicted variation of pareidolia proneness (as assessed with the pareidolia index), but in the opposite direction to Study 1. The "diametrical" or mentalizing dimension (PCDF-K) significantly associated with the pareidolia index. Larger scores in the PCDF-K (contrasting elevated positive schizotypic with lower autistic traits) associated with a *smaller* pareidolia effect in Accuracy (i.e. a larger IE:FACE-FLO). At first glance, these results contradict our and the diametrical model's predictions (Crespi & Badcock, 2008). Yet, as we will discuss in the following, the "diametrical" dimension may once more reflect differences in basic visual processing rather than pareidolia-proneness.

As concluded for Study 1, the relationships between both IE indices (IE:FLO) and behavioural variables with personality traits (e.g. ComMind) might reflect systematic biases in basic visual processing rather than pareidolia-proneness. Contrary to what has been expected for the Mentalistic dimension (PCDF-K), larger ComMind scores associated with a *larger* (rather than *smaller*) pareidolia effect in Accuracy (i.e. smaller IE:FACE-FLO). This association mirrors and likely underlies the observed association between PCDF-K and pareidolia index for Accuracy, because ComMind scores contribute negatively to the diametrical Mentalistic score (PCDF-K). To reiterate, this finding for ComMind scores is at odd with our and the diametrical model's predictions (Crespi & Badcock, 2008). Also, we found no evidence of a genuine pareidolia effect (no association of larger ComMind scores with larger IE:FLO and smaller FLO:INV for Accuracy). Instead of pareidolia-proneness, larger ComMind scores associated with preserved performance for UPR stimuli (contrary to UnEx), in line with recent studies about preserved configural processing in ASp (Hedley et al., 2014; Stevenson et al., 2016; Weigelt et al., 2012), and improved Accuracy for OBJ stimuli, irrespectively of their orientation, possibly related to a preference for objects (Wolf et al., 2008) and/or gaze avoidance (Senju & Johnson, 2009; Tanaka & Sung, 2013), as seen in association with autistic traits (Davis et al., 2016).

Likewise, Ima did not show a convincing association with pareidolia effect, it did not associate with impaired configural processing, but associated with improved OBJ processing. Larger Ima scores tended to associate with a larger pareidolia effect (i.e. smaller IE:FACE-FLO) for

RTs. As for ComMind, this paradoxical result did not resist to the test (no association of larger Ima scores with either larger IE:FLO or FLO:INV in RTs). Instead of a pareidolia effect, Ima may reflect intact performance for stimuli with a facial configuration, as seen in ASp (see also Hedley et al., 2014; Stevenson et al., 2016; Weigelt et al., 2012), and an advantage for faster FLO:UPR processing. We suggest that Ima reflects an object-like processing bias for cars' FLO explained by attenuated social imagination or fantasy-proneness (Crespi et al., 2016), for instance resulting from gaze avoidance (Davis et al., 2016; Senju & Johnson, 2009; Tanaka & Sung, 2013).

Similarly, hyper-Mentalistic positive schizotypy (UnEx) did not associate with any variation in pareidolia-proneness, but associated with worse processing of face configuration, as already observed in and suggested for results of Study 1. Larger UnEx scores associated with reduced IE:FACE due to worse processing of stimuli with UPR face configuration (FACE, FLO). Comparable to Study 1, these findings again suggest that positive schizotypy (UnEx) associate with worse processing of UPR Face stimuli, possibly a configural processing deficit, as reported for positive schizotypy (Batty et al., 2014; Larøi et al., 2007), individuals at ultra-high risk for schizophrenia (Kim et al., 2010), and schizophrenia patients, notably with positive symptoms (Butler et al., 2008; Chambon et al., 2006; Shin et al., 2008; Walther et al., 2009).

In sum, we suggest that instead of *diametrically opposite* pareidolia-proneness (based on Crespi & Badcock, 2008), Mentalistic traits can result in *distinct* face/object processing styles. Enhanced positive schizotypy (UnEx) may associate with impaired processing of face-like configurations, and enhanced autistic mentalizing deficit traits (ComMind, Ima) may associate with preserved configural processing. Although behavioural performances were similar for FLO and OBJ, they showed different associations with personality traits, suggesting pareidolia effects that depend on individual differences. Indeed, an autistic OBJ processing advantage did not extend to stimuli with a face-like configuration (i.e. FLO), while schizotypy FACE configural deficits extended to stimuli with a face-like configuration (FLO). Hence, ambiguous FLO stimuli may help in distinguishing between an autistic profile with an advantage for non-face(-like) stimuli, and a positive schizotypy profile with face configural deficits extending to face-like stimuli.

4.4.6 LIMITATIONS

Limitations of Study 3 pertain again to questionnaires, behavioural variables, stimuli, and homogenization issues. We refer to the short discussion of Study 2, where we detailed our reservations concerning our questionnaires (see also Sierro, Rossier & Mohr, 2016). Moreover, several behavioural variables were not normally distributed (FACE:UPR and OBJ:UPR),

potentially biasing results. Our main limitation, however, concerns the absence of significant IE for the FLO:Cars category. Although standardization and homogenization of stimuli influences face and object processing more than category itself (Ashworth et al., 2008; Thierry et al., 2007), the standardization of FLO (only using similarly oriented front views of cars) did not induce a significant IE (although we observed one in Study 1 using heterogenous images). Also, we did not replicate the significant IE for OBJ:Chairs of Study 2. These results highlight the difficulty to obtain stable IEs for non-FACE stimuli, and thereby compromise our ability to account behaviourally for pareidolia-proneness. However, standardization and homogeneization were not required for us to obtain an IE with the rather heterogenous FLO in Study 1. Other studies support the improvement of stimuli, or simply adoption of standardized FACE, FLO (i.e. Cars), and OBJ to get a behavioural IE. For instance, Windhager et al. (2010) found a face-like visual exploration for homogenous FLO images. These points will be further discussed in detail in the general discussion.

4.5 COMPARISON BETWEEN STUDIES 1, 2 AND 3 RESULTS

4.5.1 MEASURES AND STATISTICS

We compared ages, self-report scores, and visual recognition performance across all 3 studies. For participants' ages, we performed two-ways ANOVA on age with Sex and Study as between-subject variables. Also, we performed two MANOVAs on sO-LIFE and AQ-BC scores, with Sex and Age as between-subject variables. Note that we used dichotomized AQ-BC scores so that we could estimate the variation of autistic traits based on a common measure across all 3 studies. For visual recognition task performance, we performed two repeated measures ANOVAs, one on Accuracy, the other on RTs, with stimuli Category as a within-subjects variable, and Study as a between-subjects variable. We report *F*, *df*, *p*-values. Finally, we compared the magnitude of pareidolia indices in Accuracy and RT throughout all three studies using one-way ANOVAs with pareidolia indices as dependent variables and Study as independent variable. We report *F*, *df*, *p*, and partial eta squared (η_P^2).

4.5.2 AGES

The mean age did not differ between Sexes, $F(1,138)=1.73$, $p=.190$, $\eta_p^2=.01$), but did as a function of Study ($F(2,138)=5.78$, $p=.004$, $\eta_p^2=.08$). The interaction between Sex and Study was also significant ($F(2,138)=3.29$, $p=.040$, $\eta_p^2=.05$). Post hoc comparisons showed that the mean age

was comparable for participants in Study 1 (22.64±4.15) and Study 2 (23.79±4.7) and for participants in Study 1 (22.64±4.15) and Study 3 (20.9±2.2) (p>.05). Participants in Study 2 (23.79±4.7) were older than those in Study 3 (20.9±2.2) (p<.05). The interaction effect can be explained by a younger mean age of women in Study 3 (19.95±1.79) as compared to women in both Study 1 (23.42±4.37) and Study 2 (23.17±3.55), and as compared to men in Study 2 (25.67±7.02) (p's <.05). The men in Study 3 (21.57±2.23) were significantly younger as compared to men in Study 2 (25.67±7.02) (p <.05). All other comparisons were non-significant (all other p's >.05).

4.5.3 SELF-REPORT QUESTIONNAIRES

Scores in sO-LIFE dimensions (Total score excluded) differed between Studies (Pillai's Trace=.14, F(8,272)=2.51, p=.012), but not between Sexes (Pillai's Trace=.14, F(4,135)=1.76, p=.140). The interaction between Sex and Study was marginally significant (Pillai's Trace=.11, F(8,272)=1.90, p=.060). The respective descriptive data can be found in Tables 9, 15 and 20.

Post hoc comparisons showed that CogDis scores differed between studies, (F(2,138)=8.39, p<.0019; all other p's >.05). CogDis scores were comparable for Study 1 (3.54±3.13) and Study 2 (4.10±2.62) (p>.05), but were lower in Study 1 (3.54±3.13) than Study 3 (5.81±2.73), and lower in Study 2 (4.10±2.62) than Study 3 (5.81±2.73) (p's <.05). ImpNon scores significantly differed as a function of Study (F(2,138)=3.93, p=.022), and also for the interaction between Study and Sex (F(2,138)=3.39, p=.037). Post hoc comparisons showed that ImpNon scores were significantly smaller in Study 2 (2.35±1.88) as compared to Study 3 (3.5±2.37) (p<.05). Study 2 and Study 3 scores did not significantly differ from Study 1 (2.79±1.89) (all other p's >.05). Interaction was explained by significantly larger ImpNon scores in Study 3 Males (3.79±2.62) as compared to Study 1 Females (2.05±1.43) and Study 2 Males (1.67±1.30) (p's <.05; all other p's >.05).

Scores in AQ-BC dimensions (Total score excluded) did not significantly differ as a function of Studies (Pillai's Trace=.10, F(10,270)=1.41, p=.175), Sex (Pillai's Trace=.03, F(5,134)=0.88, p=.499), or interaction between Sex and Study (Pillai's Trace=.05, F(10,270)=0.69, p=.736).

4.5.4 VISUAL RECOGNITION TASK PERFORMANCE

The repeated measures ANOVA on IE:Accuracy showed a main effect of Study (F(2,141)=6.26, p=.002, η_G^2 =.03), a main effect of Category (ε_{GG} =0.95, F(1.9,267.9)=65.39,

p<.001, η_G^2=.23), and an interaction between Study and Category (ε_{GG}=0.95, F(3.8,267.9)=5.08, p<.001, η_G^2=.04). Post hoc comparisons showed that IE:Accuracy did not differ between Study 1 (0.06±0.17) and Study 2 (0.08±0.17) (*p's* >.05). Yet, IE:Accuracy was larger in Study 2 than Study 3 (0.03±0.11) (p<.05). Also, IE:Accuracy was larger for FACE (0.16±0.16) than both FLO (-0.00±0.14) and OBJ (0.02±0.11) (*p's* <.05), whereas IE in Accuracy did not differ between FLO and OBJ (p>.05). The interaction between Study and Category was mainly explained by the large IE:Accuracy for OBJ (Chairs) in Study 2 (see Tables 10, 16 and 21 for means±sd). IE in Accuracy for FACE did not differ between studies (*p's*>.05), and were larger than IE:FLO for all Studies (*p's*<.05). IE:Accuracy for FACE of each Study were larger than IE:OBJ for Studies 1 and 3 (*p*'s<.05). IE in Accuracy for OBJ in Study 2 was significantly smaller than IE:FACE in Study 2 (p<.05), but did not differ from IE:FACE in Study 1 and IE:FACE in Study 3 (*p's*>.05). To put it shortly, the IE in Accuracy for OBJ (Chairs) in Study 2 was equally large as the IE in Accuracy for FACE in Studies 1 and 3. Also, the IE in Accuracy for OBJ in Study 2 was larger than each of IE:FLO and IE:OBJ for each other Studies (all *p*'s<.05). The IE of OBJ in Study 2 was also larger than IEs of OBJ in Studies 1 and Study 3, and larger than IE for FLOs in each Studies 2 and 3 (*p*'s<.05; all other *p*'s>.05).

The repeated measure ANOVA on IE:RTs showed significant main effects of Study (F(2,141)=3.63, p=.029, η_G^2=.02), and of Category (ε_{HF}=0.68, F(1.37,192.66)=28.91, p<.001, η_G^2=.11), but no interaction between Study and Category (ε_{HF}=0.68, F(2.73,192.66)=0.91, p=.430, η_G^2=.01). Post hoc comparisons showed that IE:RT were significantly longer in Study 1 (222.15±543.42) than in Study 2 (109.65±211.62) (p<.05), and tended to be longer in Study 1 than Study 3 (119.83±264.63) (p<.10). IE:RT did not differ between Studies 2 and 3 (p>.05). Post hoc comparisons showed that IE:RT were significantly larger for FACE (320.25±528.87) than both FLO (71.89±246.84) and OBJ (59.49±183.58) (*p's* <.05), whereas IE:RT did not differ between FLO and OBJ (p>.05).

4.5.5 PAREIDOLIA INDICES

We compared the pareidolia indices for Accuracy and RT across studies (see Tables 10, 17 and 22 for descriptive values). The pareidolia index for Accuracy differed between studies (F(2,93)=4.77, p=.010, η_P^2=.06). Tukey post hoc tests showed that pareidolia indices for Accuracy were comparable for Study 1 and Study 3 (p>.05), and each was smaller than the one of Study 2 (*p's* <.05; all other *p*'s>.05). Hence, the *pareidolia effects* were stronger in Study 1 and Study 3 as

compared to Study 2. For RT, there was no significant effect of Study on pareidolia indices ($F(2,93)=0.33$, $p=.718$, $\eta_P^2=.01$).

4.6 GENERAL DISCUSSION

In our 3 Studies, we tested whether the Mentalism scores, "diametrically opposing" positive schizotypy (UnEx) and autistic mentalizing deficits (ComMind, Ima), could predict, respectively, larger and smaller pareidolia-proneness in unaffected undergraduate students. The main findings were that "diametrically opposite" Mentalistic traits (PCDF-BC/-K) rather than the "shared" ones (PCSF-BC/-K) associated with face(-like) processing, but pareidolia-proneness did not vary as a function of "diametrically opposite" ASp hypo-Mentalism and PSp hyper-Mentalism (Crespi & Badcock, 2008), nor was face processing simply enhanced by the latter and impaired by the former (Crespi & Badcock, 2008). Instead, we found evidence of "distinct" basic visual processing biases for ASp and PSp corresponding to current literature on the topic. In PSp, we identified a configural processing deficits and a possible local bias (Kim et al., 2010; Panton, Badcock, & Badcock, 2016). In ASp, we identified preserved configural processing, a possible preference for non-social stimuli and difficulty with standardized stimuli (Rhodes et al., 2013; Stevenson et al., 2016; Weigelt et al., 2012; Wolf et al., 2008). Here, we review our findings and discuss their potential implications for models on ASp-PSp relationships.

Our experimental protocol resulted in IE, comparable performances and results, as reported in the literature. Once we had raised the number of stimuli (Studies 2 and 3), we replicated previous reports of an overall 70% accuracy (Kim et al., 2010). We reliably induced IE for Face in each study. Yet, IE for FLO and OBJ were smaller and could not be observed reliably (they were absent in Study 3). Haxby et al. (2000) argued that IE is not exclusive but *disproportionate* for Face, and may exist for OBJ too. Caharel et al. (2013) showed that the intermediate category of FLO may not result in a significant IE, despite showing face-like neural correlates (Caharel et al., 2013; Churches et al., 2009; Hadjikhani et al., 2009; Windhager et al., 2010). Importantly, an overall IE may be masked precisely because of interindividual variability, for instance in personality traits.

"Shared" traits (PCSF-BC/K) did neither predict pareidolia proneness nor general behavioural performance. We observed minor differences in scores between studies (e.g. for CogDis), but variation in this scale might only have affected "shared" but not "opposite" Mentalistc traits. Indeed, we considered positive schizotypy (UnEx scores) to be of major importance to our studies (Belayachi et al., 2014; Fyfe et al., 2008; Sierro, Rossier & Mohr, 2016), and UnEx scores

were indeed related to our behavioural measures. Before taking up the UnEx results in more detail, we here conclude that "shared" traits were not relevant to account for pareidolia-proneness or basic visual performance, as assessed in the present Studies. As negative and disorganized traits overlap for ASp and PSp in healthy population (Dinsdale et al., 2013; Ford & Crewther, 2014; Sierro, Rossier & Mohr, 2016) and clinical populations (Malaspina et al., 2014), it is theoretically reassuring and not surprising that they do not distinguish between ASp and PSp cognitive styles in face processing.

In contrast, "diametrically opposite" Mentalistic traits associated with face processing, at least in Studies 1 and 3 – yet, with increased pareidolia-proneness in Study 1 and decreased pareidolia-proneness in Study 3. While the former result would support Crespi & Badcock's (2008) diametrical model, the latter would challenge it. After having considered the contribution of individual variables (rather than indices), we concluded that instead of pareidolia-proneness, the behaviour more likely reflected basic visual processing association with Mentalism's traits, i.e. positive schizotypy (UnEx) and autistic mentalizing deficits (ComMind, Ima). In particular, positive schizotypy associated with worse configural processing of face(-like) stimuli, and a local bias beneficial to object processing, both of which have been considered potential endophenotypes of SSD (patients: Chambon et al., 2006; Joshua & Rossell, 2009; Shin et al., 2008; Walther et al., 2009; unaffected relatives: Calkins et al., 2005; Gur et al., 2015; individuals with schizotypy or at ultra high-risk for schizophrenia: Batty et al., 2014; Kim et al., 2010; Larøi et al., 2007). In contrast, ComMind associated with a preserved configural processing bias as seen in ASp (Hedley et al., 2014; Stevenson et al., 2016; Weigelt et al., 2012), with a possible local bias preference (Stevenson et al., 2016; see also Happé & Frith, 2006), and a preference for recognition of objects without a face(-like) configuration (Pallett, Cohen, & Dobkins, 2013; Wolf et al., 2008), possibly reflecting gaze avoidance (Senju & Johnson, 2009; Tanaka & Sung, 2013). Ima apparent association with configural deficit was problematic. Speculatively, Ima associated with reduced fantasy-proneness (Crespi et al., 2016) compromising FLO encoding in Study 1 oppositely to UnEx (Merckelbach et al., 2000), and a possible predictive encoding difficulty resulting in a general slowness to respond to standardized stimuli in Study 2, as seen in ASD (Rhodes et al., 2013).

Contrary to the "diametrical model" (Crespi & Badcock, 2008), our results indicate that hyper- and hypo-Mentalism traits associate with "*distinct" basic visual processing styles, rather than "diametrically opposite"* pareidolia-proneness (Crespi & Badcock, 2008). We can neither conclude that larger pareidolia-proneness links to larger Mentalism. Either the basic visual processing styles may have masked the relationships between Mentalism and pareidolia-proneness, or the two are not associated. Since our assessment of pareidolia-proneness is based on visual

processing, we cannot disentangle them. Hence, our results neither support, nor disprove Crespi and Badcock (2008) diametrical model. Instead, our results displace the focus from "diametrically opposite" pareidolia-proneness to "distinct" cognitive styles related to more fundamental functions and mechanisms. We discuss potential mechanisms below.

Our protocol revealed *distinct* face recognition performances between autistic and schizotypy, due to our encoding/retrieval durations, basic perceptual deficits, category-specific processing advantage or encoding styles. Our memorization task's short encoding duration targeted encoding deficits reported in SSD face recognition (i.e. 3 secs, like Chambon et al., 2006; Chen et al., 2009; see also: Butler et al., 2008), and not the longer durations reportedly normalizing SSD performance (i.e. 5 sec., Schwartz et al., 2002). In contrast, our task's encoding-retrieval duration (3-4 min) seemed not sufficient to impair performance as seen in ASD (>20min in Weigelt et al., 2012). As a result, we identified deficits associated with positive schizotypy rather than autistic traits. As for PSp, fundamental perceptual integration deficits may underlie the configural deficits and featural bias for face(-like) stimuli associated with positive schizotypy, as seen in schizophrenia and individuals with disorganized schizotypy (Feigenson, Gara, Roché, & Silverstein, 2014; Horton, & Silverstein, 2011). As for ASp, ComMind-associated preference for OBJs (Wolf et al., 2008) and/or an aversion for "unpleasant" face and, in particular gaze (Davis et al., 2016; Tottenham et al., 2014) may, for instance, stem from passive gaze omission or active gaze avoidance (Senju & Johnson, 2009; Tanaka & Sung, 2013), resulting in advantage for OBJ processing. Finally, an internal encoding style associated with positive schizotypic traits (Belayachi et al., 2014; Brosnan et al., 2013) might have impaired encoding quality, leading to faster but less accurate responses, whereas an external encoding style associated with autistic traits (ComMind, Ima), as shown in ASD (Brosnan et al., 2014), might have increased examination and encoding of stimuli, resulting in more accurate but slower responses (Lewicki, 2005).

Our results have implications for further predictions related to the diametrical model (Crespi & Badcock, 2008), and ASp-PSp models (Chisholm et al., 2015). From our results, the diametrical model's (Crespi & Badcock, 2008) prediction of enhanced social cognition in PSp and impaired social cognition in ASp might not apply to face processing. Predictions based on the diametrical model (Crespi & Badcock, 2008) need to integrate latest knowledge on cognitive styles and endophenotype candidates in ASp and PSp. Basic face and object processing peculiarities ought to be considered prior pareidolia-proneness, because they are more prevalent and fundamental processes, and endophenotype candidates in ASp (Fiorentini, Gray, Rhodes, Jeffery, & Pellicano, 2012; Rhodes et al., 2013) and PSp (Calkins et al., 2005; Gur et al., 2015). Indeed, Mentalism may manifest distinctly depending on cognitive functions and processing levels considered, as recently

proposed for cognitive styles (Kozhevnikov et al., 2014). Hence, current models of ASp-PSp relationships (Chisholm et al., 2015) ought to be adapted to integrate "shared", "diametrically opposite", but also "distinct" features, as exemplified by "distinct" face processing in ASp and PSp.

In addition to ASp and PSp personality, gender may influence the remaining inconsistencies in face processing better than stimuli and protocol. Comparison between several studies showed that changes in stimuli categories and sampling biases may explain only partly the inconsistent results across studies. Across all 3 Studies, IE:Accuracy but not IE:RT varied. Congruently, pareidolia index varied in Accuracy but not in RT. The main puzzling differences are the increase in pareidolia index in Study 2 as compared to others, owing to a nonsignificant FLO IE. This likely perturbed relationships with personality traits and explain their differences with other studies. Also, IE for OBJ (Chairs) was surprisingly large in Study 2 as compared to Study 3. The inconsistent results in Study 2 for FLO and OBJ are difficult to explain, because FLO stimuli were similar between Studies 1 and 2, and OBJ ones were similar in both Studies 2 and 3. Since the notable difference in Study 2 is the overrepresentation of women, gender effects may have altered the relationships between personality and basic visual processing.

Eventhough ASp-PSp models emphasized biological sex differences in different ways (Brosnan et al., 2010; Crespi & Badcock, 2008), gender differences should also be considered might also alter the relationships between basic visual processing and personality. Possibly, the over-representation of women in Study 2 may explain the larger IE for OBJ:Chairs. Also, UnEx in Study 2 rather consists in a local bias than a configural deficits, suggesting UnEx reflects featural or local bias in women (Rhodes et al., 2013). Conversely, UnEx in men may rather reflect configural deficits, usually based on a majority of male SSD patients (e.g. Chambon et al., 2006; Butler et al., 2008; Walther et al., 2009). Although sex-differences are assumed to be key in ASp-PSp relationships (Brosnan et al., 2010; Crespi & Badcock, 2008), future studies should use instruments or procedures ruling out socio-demographic influences, before concluding to sex-specific face recognition endophenotypes (e.g. SocSki for men; AttDet for women: Rhodes et al., 2013; see also Halliday et al., 2014).

4.6.1 LIMITATIONS AND FUTURE PERSPECTIVES

Our studies featured several limitations pertaining to sampling biases, sample sizes, psychometric instruments and protocol. Despite our studies' samples differed in ages and sex ratios, they may only partly explain the different correlation pattern we found between visual recognition

performance and personality across Studies. Further studies should control for demographic variables such as sex ratios, and clarify their relationships with questionnaires' scores and behavioural performance. Our results are limited by the properties of the autistic and schizotypic traits instruments we used (e.g. Sierro, Rossier, Mason et al., 2016; Sierro, Rossier & Mohr, 2016). Several studies criticized AQ factor structure and reliability, recommending improvement of this instrument (e.g. Hurst, Mitchell et al., 2007; Sierro, Rossier & Mohr, 2016). Hopefully, the switch from dichotomized AQ-BC to AQ-K did not suggest any bias, because correlations between corresponding dimensions were large. sO-LIFE factor structure and scales reliability were more robust, with the exception of ImpNon (Fonseca-Pedrero et al., 2015; Sierro, Rossier, Mason et al., 2016). Globally, sO-LIFE and AQ-BC scores did not vary as a function of study (with the exception of sO-LIFE's CogDis and ImpNon). Although the variations in sO-LIFE's CogDis and ImpNon across studies may have affected the PCSF and PCDF scores, the key Mentalistic dimensions (UnEx, ComMind and Ima) remained stable and allowed us to properly test our hypotheses. Our sample sizes were small in comparison to those to comparable face recognition studies (e.g. 150-240 participants; Halliday et al., 2014; Rhodes et al., 2013). Given the small effect size and possible sex-specific effects, further studies might require a 1.5 to 2-fold larger samples to reach an acceptable power (e.g. 30 women in Rhodes et al., 2013).

Quantifying pareidolia-proneness represented the major challenge of this research, raising issues of stimuli standardization, both in terms of semantic category and visual properties (Ashworth et al., 2008; Thierry et al., 2007). Our stimuli varied in number, in difficulty, in homogeneity, and in their ability to elicit IE (irrespectively of homogeneity). Possibly, the larger number of stimuli in Studies 2 and 3 caused the smaller variabilities in IEs as compared to IEs in Study 1, in particular for FACE. Further experiments ought to use ecological, validated and standardized sets of stimuli, such as those we used in Study 3. Yet, standardization of visual features and homogenization of the semantic category did not increase the IEs of non-FACE stimuli, against recent reports (Ashworth et al., 2008; Thierry et al., 2007). Carefully measuring stimuli homogeneity may help further studies to have equivalent face and non face stimuli, and to equate reliance on configural processing (Pallett & MacLeod, 2011; Thierry et al., 2007).

Beyond stimuli features, our task may not be a good measure of behavioural pareidolia-proneness for two reasons: the existence of a behavioural FLO IE is unclear, and IE might not only account for a face-like processing. IE may not be detectable as a behavioural IE, while showing face-like neural correlates (Caharel et al., 2013). Neural correlates may be preferable to IE for quantifying pareidolia-proneness (EEG: Caharel et al., 2013, Churches et al., 2009; eyetracking: Windhager et al., 2010; and fMRI: Hadjikhani et al., 2009, Kühn et al., 2014). Still, even the N170

face-specificity was challenged (Thierry et al., 2007), so that face-specific neural correlates still need to be further confirmed, independently from its particular relevance for PSp (Feuerriegel et al., 2015; Tsunoda et al., 2012). Alternative pareidolia-proneness measures would also be informative relatively to face processing broadly speaking (Caharel et al., 2013; Falck-Ytter, 2008). Theoretically, our operationalization of pareidolia-proneness relies on the controversial assumption that IE reflect a face-like processing of stimuli. However, configural sensitivity extended to non-facial/non-social stimuli (i.e. Greebles) as a function of expertise (Gauthier & Tarr, 1997). Hence, IE disruption of configural information may reflect expertise with a stimulus category rather than face-specificity (Diamond & Carey, 1986; but see McKone, Kanwisher, & Duchaine, 2006). Controlling or measuring expertise as a covariate may be helpful to further studies.

Further experiments interested in finding "opposite" or "distinct" performance patterns between ASp and PSp could also other protocols or measure other behavioural variables. The simple old-new recognition paradigm we used does not allow to identify specific contribution of encoding duration, encoding-retrieval delay or memory load. Simplier discrimination protocols might prove useful to investigate encoding (e.g. Wallace et al., 2010). As for stimuli recognition, manipulating encoding duration might target PSp deficits (Butler et al., 2008; Chambon et al., 2006), while manipulating encoding-retrieval delay might target ASp face processing deficits (Weigelt et al., 2012). As for distinction between internal and external cognitive styles, Signal Detection Theory could be used to allow a better measurement of the performance of participants, possibly showing differences in criterion (Brosnan et al., 2013, 2014; Lewicki, 2005). Finally, other behaviours crucial for social interactions ought to be investigated. Facial emotion recognition could also be investigated in ASp and PSp, since it was widely investigated in ASD and SSD (Amminger et al., 2012; Wallace et al., 2010). Also, gaze processing should be investigated since it may explain at the same time an ASp face processing deficits through avoidance, and possibly lack of expertise (Senju & Johnson, 2009; Tanaka & Sung, 2013), "diametrically opposite" joint attention (Langdon et al., 2006; Ristic et al., 2005), and gaze direction attribution (Hooker & Park, 2005; Matsuyoshi et al., 2014), contrasting ASp and PSp on a Mentalistic dimension.

4.7 CONCLUSION

Across 3 studies, we could not confirm the hypothesis that pareidolia-proneness varies as a function of Mentalism, as would be expected from the diametrical model (Crespi & Badock, 2008). We confirmed that "shared" negative/disorganized traits were not relevant to predict pareidolia-

proneness. In contrast, although "diametrically opposite" Mentalistic traits associated with behavioural results, we found no consistent association of higher (lower) Mentalistic traits with higher (lower) pareidolia-proneness. Instead, Mentalistic traits associated with basic face and object processing. Positive schizotypy (UnEx) associated with worse configural processing of face-like stimuli and a local bias for object processing, as previously reported in PSp (Batty et al., 2014; Butler et al., 2008; Chambon et al., 2006; Joshua & Rossell, 2009; Panton et al., 2016; Shin et al., 2008). Autistic mentalizing deficits (ComMind) associated with already reported absence of configural deficits in ASp (Hedley et al., 2014; Stevenson et al., 2016; Weigelt et al., 2012), and a bias or preference for object recognition devoid face(-like) configuration in ASD (Pallett et al., 2013; Wolf et al., 2008), or (for Ima) broad configural deficits possibly reflecting lower fantasy-proneness (Crespi et al., 2016) or adaptive coding deficits (Rhodes et al., 2013). We suggest that hyper- and hypo-Mentalistic traits associate with distinct basic visual processing styles rather than diametrically opposite pareidolia-proneness. Before positing diametrical or shared processing styles, we stress that research about ASp-PSp should integrate the "distinct" features. Indeed, distinct processing styles, deficits or associated mechanisms are apparent in existing neurocognitive endophenotype research (Gur et al., 2015; Lainhart & Lange, 2009; Snitz et al., 2006; Sucksmith et al., 2011). Since ASp-PSp models cannot be reduced to either shared, distinct or diametrical features, they ought to integrate altogether shared, diametrical *and also* distinct features, as no existing model do (Chisholm et al., 2015). Future studies ought to further develop better ways to measure pareidolia-proneness, disentangling it from basic face-object processing, and better understand gender differences.

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5 GAZE PROCESSING AND MENTALISM[19]

Abstract

Autism spectrum disorders (ASD) and schizophrenia spectrum disorders (SSD) share a common history and clinical features. Since the 1970[ies], however, they have been considered distinct diagnostic entities. Recent studies question this separation arguing again for their relatedness due to shared negative symptoms [Rausch & Johnson, 2008]. Crespi and Badcock [2008] theorized on "diametrical" features opposing autistic, hypo-functional social cognition (hypo-Mentalism) to psychotic, hyper-functional social cognition (hyper-Mentalism). These "diametrical" features would extend to the healthy end of these spectra (personality traits). Psychometric studies support both "shared" and "diametrical" autistic and schizotypic traits. We tested for similar support in cognitive measures. We investigated in 68 university students whether behavioural measures of social cognition (i.e. gaze sensitivity: gaze direction perception, gaze cueing liability) mirror such "shared" and "diametrical" traits in healthy undergraduates. We hypothesized that positive schizotypy would go along with a hyper-sensitivity towards gaze and autistic traits with a hypo-sensitivity towards gaze. We found (i) superior gaze direction accuracy with enhanced positive schizotypy traits and lower autistic mentalizing deficits, respectively, and (ii) lower gaze cueing liability with enhanced autistic mentalizing deficits (no effect for schizotypy), but only in men. The first result supports the diametric model by Crespi & Badcock, but on statistically disputable ground. The second result shows an autistic, male-specific gaze under-sensitivity, potentially reflecting a candidate endophenotype of ASD. We are discussing our results with the wish to present a potentially more comprehensive understanding of ASD-SSD relationships. In particular, we propose to add "distinct" features to the "shared" and "diametrical" features.

Keywords: schizotypy; autism; endophenotypes; gaze direction, gaze cueing

[19] Sierro, G. & Mohr, C. Autistic and schizotypic traits predict diametrical performance patterns in gaze direction and distinct performance in gaze cueing. *In preparation.*

5.1 INTRODUCTION

Autism Spectrum Disorders (ASD) and Schizophrenia Spectrum Disorders (SSD) share common historical origins, despite being now considered distinct psychopathologies. Kanner (1943) coined the term "autism" referring to social withdrawal described in schizophrenia patients (Bleuler, 1911). Subsequently, until the 70^{ies}, infantile autism was considered a subtype of psychosis characterized by an inability to relate to others (Kolvin, 1971; Rutter, 1972). Infantile autism was dissociated from the group of childhood psychoses mostly on the ground of the former's earlier age of onset (about 2 years of age). Since the publication of these studies, ASD and SSD have been considered separate psychiatric diagnostic categories, and still are in the recent 5th edition of the Diagnostic Statistical Manual of Mental Disorders (*DSM-5*; APA, 2013). Despite this separation, ASD and SSD remain frequently linked.

The two conditions share phenomenological similarities, i.e. negative as well as positive symptoms (Hallerbäck et al., 2012; Konstantareas & Hewitt, 2001). Broadly, negative symptoms here refer to deficits and positive symptoms to abnormal or eccentric features (Malaspina et al., 2014). Rausch and Johnson (2008) link ASD and SSD due to their overlap in both negative and positive symptoms. They argued that positive SSD symptoms represent a cognitive subtype of autistic stereotypies. Speaking also for a link between ASD and SSD, studies reported on comorbidities and misdiagnoses (Hallerbäck, et al., 2012; Konstantareas & Hewitt, 2001; Nylander & Gillberg, 2001; Perlman, 2000). For instance, Hallerbäck et al. (2012) showed that more than half of their SSD patients with paranoia met criteria for ASD.

More recently, researchers have taken a new interest in differences and similarities for ASD and SSD. In particular, various groups consider that differences and similarities inform on aetiology including genetic mechanisms (e.g. Abu-Akel & Bailey, 2000; Brosnan et al., 2010; Crespi & Badcock, 2008; King & Lord, 2011; Rausch & Johnson, 2008). A first group of researchers emphasize similarities, arguing that they reflect common underlying mechanisms for ASD and SSD, but which become differently expressed, e.g. as a function of genetic combinations influencing age of onset or severity (King & Lord, 2011; Rausch & Johnson, 2008). Rausch and Johnson (2008) argue that both negative and positive symptoms are shared between ASD and SSD and that genes influencing age of onset and severity distinguish ASD and SSD. This theory emphasizes shared features and does not predict diametrical or opposite behavioural patterns between ASD and SSD, and no extension to healthy phenotypes.

A second group emphasizes differences between ASD and SSD, argueing for instance that these conditions are diametrically opposite disorders of the social brain (Brosnan et al., 2010;

Crespi & Badcock, 2008; see also Abu-Akel & Bailey, 2000). As outlined further below, Crespi and Badcock (2008) proposed that this opposition stems from opposing over-expressions of maternal and paternal genes, causing respectively an over-developed social brain in SSD (also called hyper-Mentalism) and an under-developed social brain in ASD (also called hypo-Mentalism; see Badcock, 2004). Crucially, this theory goes beyond gene coding for age of onset and severity proposing instead diametrical phenotypes for ASD and SSD, extending from genes to behaviour, and from clinical to healthy personality levels. Most important to our study, part of the recent evidence feeding this debate has been based on psychometric evidence from healthy populations, i.e. studies that are based on dimensional models of psychopathology (Brosnan, et al., 2010; Dinsdale et al., 2013).

Dimensional models assume that disorders are extreme variants of normal phenotypes. According to this framework, healthy personality features are quantitatively minor but qualitatively similar to acute clinical symptoms present in patients (Claridge, 1997; Constantino & Todd, 2003; Meehl, 1990). Moreover, the healthy expressions might represent vulnerability markers of the illness (e.g. Claridge, 1997; Constantino & Todd, 2003; Meehl, 1990). In case of the SSD, researchers described schizotypy in the general population (e.g. Claridge, 1997; Meehl, 1990). Schizotypic traits represent a heritable and genetic vulnerability to SSD (Grant et al., 2014; Mason & Claridge, 2006). In the case of ASD, researchers described autistic traits in non-affected relatives of ASD individuals (i.e. the BAP, e.g. Bailey et al., 1998; Lai et al., 2013), and in the general population (Baron-Cohen et al., 2001; Constantino & Todd, 2003). Likewise, autistic traits would represent a heritable genetic liability to ASD (Hoekstra et al., 2007; Lundström et al., 2012).

Researchers commonly use self-report questionnaires to assess schizotypic and autistic traits (Hurst, Nelson-Gray, Mitchell, & Kwapil, 2007 for an example). In the case of schizotypy, common self-report questionnaires are the Short Oxford-Liverpool Inventory of Feelings and Experiences (O-LIFE) which exists in a short form (sO-LIFE) (Mason et al., 2005), the Schizotypal Personality Questionnaire (Raine, 1991), and the Wisconsin Schizotypy Scales (Winterstein, Silvia et al., 2011). In the case of autistic traits, the most common self-report questionnaire is the Autism Spectrum Quotient (AQ; Baron-Cohen et al., 2001), although other instruments exists (e.g. Social Responsiveness Scale by Constantino, Davis et al., 2003; Broader Autism Personality Questionnaire by Hurley et al., 2007). Scores from such self-report questionnaires associate with biological and behavioural markers, or endophenotypes, in both SSD (see Barrantes-Vidal et al, 2015; Cohen, Mohr, Ettinger, Chan, & Park, 2015; Ettinger et al., 2015 for recent overviews) and ASD (Dawson et al., 2002; Sucksmith et al., 2011), including schizotypic traits (Cappe, Herzog, Herzig, Brand, & Mohr, 2012; Chkonia et al., 2010) and autistic traits (Wallace et al., 2010;

Matsuyoshi et al., 2014) in the general population. An endophenotype refers to a subset of illness markers that are stable, heritable, found in patients, overrepresented in their relatives, but also found to a lesser extent in the general population (Gottesman & Gould, 2003). Hence, studies with healthy populations can inform on potential aetiological mechanisms underlying these illnesses including possible differences.

At this point, it is relevant going back to the assumption that ASD and SSD are diametrically opposite disorders of the social brain (Crespi & Badcock, 2008), because we can now formulate predictions regarding behavioural correlates of ASD and SSD. Crespi and Badcock (2008) proposed that phenotypes of ASD versus SSD should yield opposite cognitive styles related to social cognition, such as gaze processing and agency detection. Eye gaze processing is particularly suitable in this regard, because it informs on emotional and social cognitions such as deception, empathy, ToM, and perspective taking (Emery, 2000; Itier & Batty, 2009; Kennedy & Adolphs, 2012). Eye gaze processing can, thus, inform on hypo-Mentalistic under-sensitivity to gaze in ASD versus hyper-Mentalistic over-sensitivity to gaze in SSD (Badcock, 2004). Previous studies already showed different types of gaze processing impairments in ASD and SSD. In ASD, under-sensitivity to eye gaze in ASD has been demonstrated through deficits in GD discrimination, gaze following, joint attention, Mentalistic understanding of gaze and gaze avoidance (Baron-Cohen, Campbell, Karmiloff-Smith, Grant, & Walker, 1995; Klin et al., 2002; Ristic et al., 2005; Wallace et al., 2006). In SSD, over-sensitivity to eye gaze has been demonstrated through an excessive GC effect, the exaggerated self-attribution of gaze contact and an over-proportional response to others' GD (Langdon et al., 2006; Tso et al., 2012). Overall, impaired eye gaze processing might impede efficient mindreading and social skills in both ASD and SSD (Hooker & Park, 2005; Ristic et al., 2005).

In the current study, we used two gaze processing paradigms, i.e. GD perception and GC of attention (i.e. attentional redirection by a perceived GD; Driver et al., 1999). These were used to assess whether performance differences match opposing psychometric features of schizotypic and autistic traits. The gaze processing tasks have been powerful tools to elicite opposite impairments in ASD and SSD (e.g. Frischen et al., 2007; Hooker & Park, 2005; Langdon et al., 2006; Tso et al., 2012; Wallace et al., 2006), and some of them are discussed to represent ASD endophenotype candidates (e.g. Matsuyoshi et al., 2014; Wallace et al., 2010). With regard to GD perception, patients with ASD did not show the commonly observed advantage for GD perception when (i) eyes were shown in whole faces as compared to eye sections only and (ii) eye gaze was straight as compared to averted (Wallace et al., 2006). The latter finding was also observed in unaffected relatives of patients with ASD (Wallace et al., 2010). Individuals with higher as compared to lower

autistic traits were less likely to interpret gaze as self-directed (Matsuyoshi et al., 2014). In contrast, patients with schizophrenia over-attributed averted eye gaze as directed towards them, as compared to controls (Hooker & Park, 2005; Rosse et al., 1994; Tso et al., 2012). With regard to GC, children with ASD showed regular GC, but did not show an expected advantage for gaze over arrows (Senju et al., 2004). GC deficits seem stronger in ASD adults (Ristic et al., 2005; Vlamings et al., 2005) than ASD children (Kylliäinen & Hietanen, 2004; Senju et al., 2004; see Frischen et al., 2007 for a review). GC effects in healthy populations were worse when having enhanced autistic traits (Bayliss, Di Pellegrino et al., 2005). In contrast, patients with schizophrenia showed a faster orienting response subsequent to a GD cue, as compared to controls (Langdon et al., 2006).

Considering (i) opposite gaze processing biases in ASD and SSD and (ii) the debate on the relationship between ASD and SSD, we investigated GD (Wallace et al., 2006) and GC (Jones, Main, Little, & DeBruine, 2011) performances within the same student population assessing both their autistic (AQ: Baron-Cohen et al., 2001; Kloosterman et al., 2011) and schizotypic traits (sO-LIFE). If the model by Crespi and Badcock (2008) holds true, we predict diametrically opposite performance patterns in these eye gaze processing tasks. Based on recent psychometric studies (Dinsdale et al., 2013; Sierro, Rossier & Mohr, 2016), we expected that high positive schizotypy would associate with a hyper-Mentalistic style (i.e. oversensitive to GD, and GC) and high autistic traits (mentalizing deficits) with a hypo-Mentalistic style (i.e. undersensitive to GD, and GC). Also, we expected that men might be less sensitive to gaze as compared to women (see Bayliss, Di Pellegrino et al., 2005; Matsuyoshi et al., 2014).

5.2 MATERIAL AND METHODS

5.2.1 PARTICIPANTS

We recruited 78 students (35 women) from two local universities (University of Lausanne, Ecole Polytechnique Fédérale de Lausanne) with a mean (±SD) age of 21.90 (±2.88, range 18–30). Participants were recruited through personal contact or experimental hours scheme. They received either course credit equivalents or monetary incentives. They were informed about the main topic and overall goal of the research. It was stressed that they were free to participate, free to quit whenever they wanted, and that all information collected would be treated strictly anonymously. Prior to participation, participants provided written informed consent. The study was conducted in accordance with the guidelines of the Declaration of Helsinki (World Medical Association, 2013).

5.2.2 SELF-REPORT QUESTIONNAIRES

5.2.2.1 AUTISTIC TRAITS QUESTIONNAIRE

Autistic traits were assessed with the 50-item Autism Spectrum Quotient (AQ-BC; Baron-Cohen, Wheelwright Skinner et al., 2001) including 24 reversely formulated items. We here base our study on a psychometrically improved and shortened version of this instrument (henceforth AQ-K; Kloosterman et al., 2011), following a recent French validation study in a comparable, but larger sample (Sierro, Rossier & Mohr, 2016). The AQ-K features 28 items, grouped along 5 dimensions: Social Skills and Interest deficits (SocSki, 8 items, e.g. "I enjoy social occasions."), Communication/Mindreading deficits (ComMind, 5 items, e.g. "I find it easy to "read between the lines" when someone is talking to me."), Routines/Repetitive Behaviours (RRBeh, 5 items, e.g. "I prefer to do things the same way over and over again."), Social Imagination deficits (Ima, 5 items, e.g. "When I'm reading a story, I can easily imagine what the characters might look like."), and Attention to Details (AttDet, 5 items, e.g. "I tend to notice details that others do not."). Participants indicate their agreement with each item using a 4-point Likert scale ("definitely agree", "agree", "don't agree", "definitely disagree") attributing respectively, 4, 3, 2 and 1 point to "definitely agree", "agree", "don't agree", and "definitely disagree" (for reverse formulated, respectively, 1,2,3 and 4 points) (e.g. Lau, Kelly, & Peterson, 2013). We computed dimension scores by summing up the points attributed for each item's response in a given dimension. Normative values for the Dutch AQ-K can be found in Kloosterman et al. (2011) and for the French AQ-K in Sierro, Rossier and Mohr (2016).

5.2.2.1.1 SCHIZOTYPIC TRAIT QUESTIONNAIRE

Short Oxford-Liverpool Inventory of Feelings and Experiences questionnaire (sO-LIFE, Mason et al., 2005, French version, Sierro, Rossier, Mason et al., 2016). The 43-item English sO-LIFE assesses positive schizotypy (Unusual Experiences [UnEx]), 12 items, e.g. "Are your thoughts sometimes so strong that you can almost hear them?"), negative schizotypy (Introvertive Anhedonia [IntAn]), 10 items, e.g. "Do you prefer watching television to going out with people?"), Cognitive Disorganization (CogDis, 11 items, e.g. "Are you easily confused if too much happens at the same time?") and Impulsive Non-Conformity (ImpNon, 10 items, e.g. "Do you at times have an urge to do something harmful or shocking?"). Participants indicate for each statement whether it is true or false. The number of positive responses (negative responses are reversely coded) is summed for

each subscale so that higher scores indicate higher schizotypy. Normative English values can be found in Mason et al. (2005). The validated French version and normative French values for student populations can be found in Sierro, Rossier, Mason et al. (2016).

5.2.3 COMPUTERIZED GAZE PROCESSING TASKS

The GD task and attentional GC task were programmed on E-Prime 2.0 Pro. running on Mac with Windows OS.

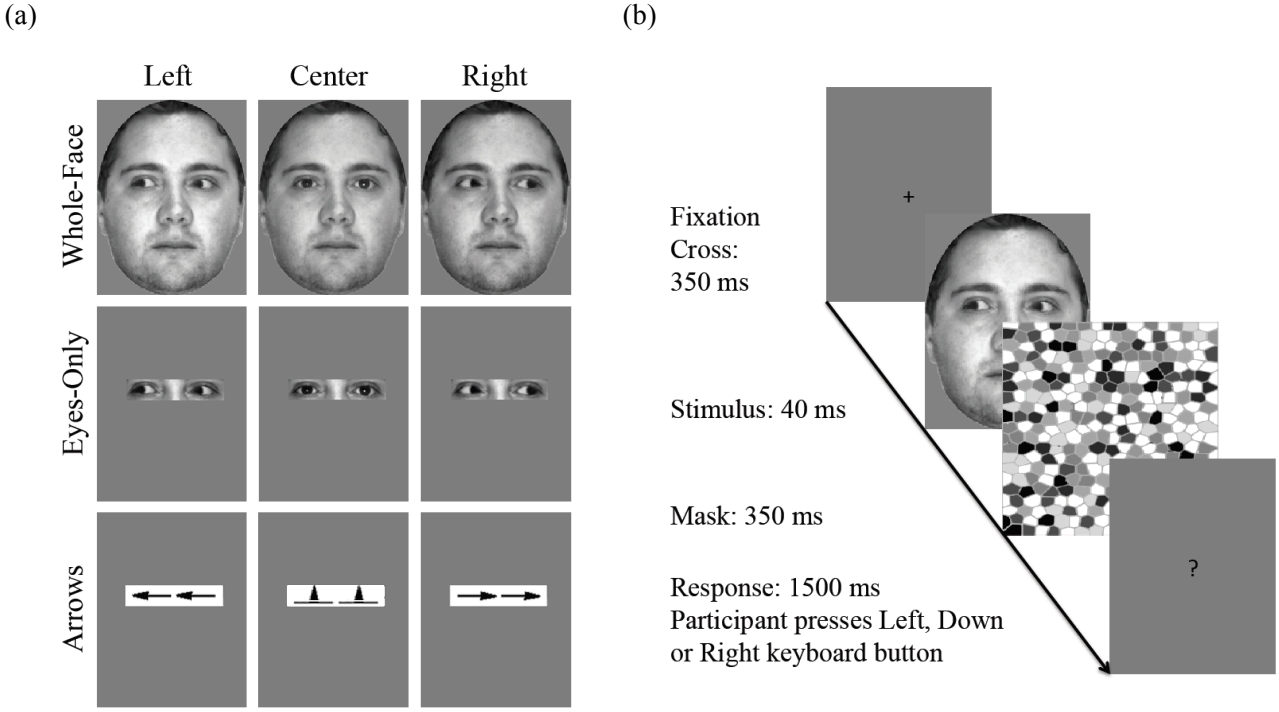
5.2.3.1 GAZE DIRECTION TASK

We replicated the task used in Wallace et al. (2006, 2010). We obtained the original stimulus material from Simon Wallace (University of Oxford). The stimuli consisted of 12 Whole-Face stimuli (7.65 cm width, 10 cm height), 12 Eyes-Only stimuli (eye region cut out from each of the 12 Whole-Face stimuli, approximately ~7 cm width, ~1.5 cm height), and 12 Arrow stimuli (size were matched to the Eyes-Only stimuli; see Figure 12a). The gaze of the Whole-Face stimuli and Eyes-Only stimuli were either straight ahead, to the right, or to the left. For the Arrow stimuli, the arrow was either pointing downward, leftward or rightward (Figure 12a). Accordingly, we had 108 different stimuli that we presented twice (216 expertimental trials). In Wallace et al. (2006), the authors presented stimuli for three different durations (40, 70 and 100 ms). They found that duration did not affect the detection of deficits in ASD or relatives. Thus, we presented stimuli for 40 ms to shorten the experiment, and to prevent serial processing and geometric analyses of eye gaze (Wallace et al., 2006, 2010). Overall, we presented trials in pseudo-randomized order, including three breaks to avoid fatigue effect. After each block, participants could take a break and continue the experiment when ready. We distributed the 216 trials across the four experimental blocks (54 trials each) plus one practice block (we randomly drew eight stimuli from all possible ones) using a random number generator (www.random.org). Then, we programmed E-Prime such that the 54 trials per block were randomized, so that trial order differed between participants. This block-wise pseudo-randomization was necessary to implement breaks within the E-Prime protocol.

An actual trial consisted of the presentation of a central fixation cross (0.8 cm × 0.8 cm; 350 ms), followed by the stimulus presentation (40 ms). A mask was directly presented afterwards (350 ms). A blank screen followed for a maximum of 1500 ms during which the participant had to respond (Figure 12b). The participant had to indicate stimulus direction by pressing one of 3

keyboard arrow buttons (left arrow for "Left"; down arrow for "Center"; right arrow for "Right") using the dominant hand. The next trial started when participants had made a response or after the 1500 ms response interval (Figure 12b). During the practice block, the experimenter stayed with the subject to make sure the subject understood the instructions. We assessed percent correct responses (accuracy) and RTs for correct responses for each participant and for each of the 9 conditions separately. The overall task took about 12 min to complete.

Figure 12. Gaze Direction experiment. Illustration of one example for each stimulus category (a). In (b), we show the sequence of events).

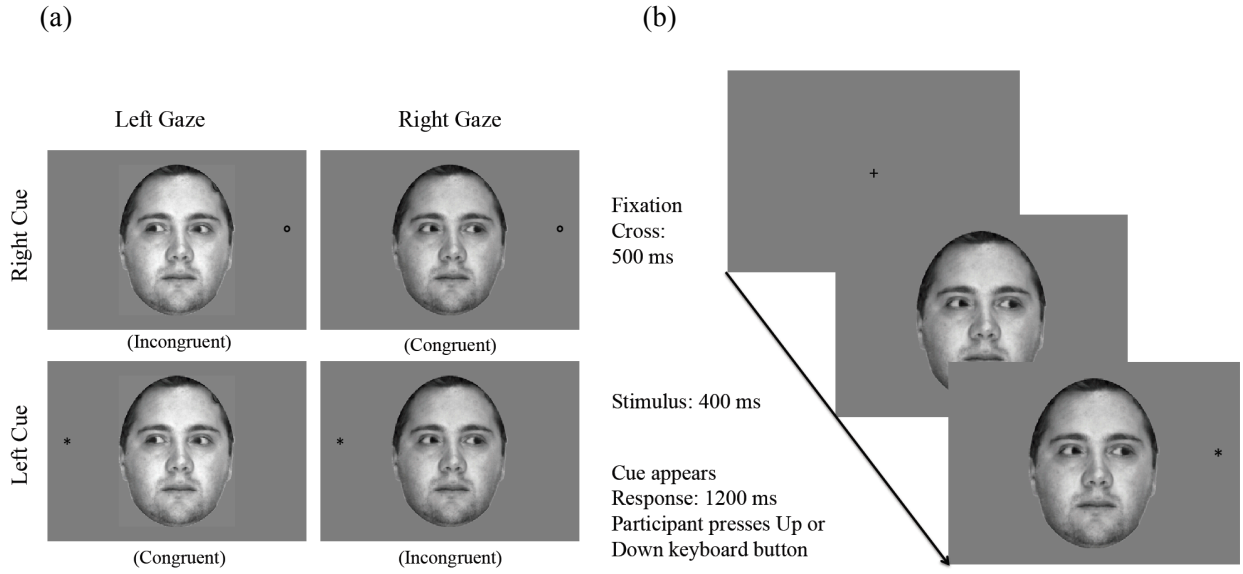


5.2.3.2 *GAZE CUEING TASK*

We performed the GC task paradigm used in Driver et al. (1999) and Jones et al. (2011). However, instead of their stimuli, we showed the Whole-Face stimuli also shown in the GD task (see also Figure 13). We used 12 Whole-Face identities (7.65 cm wide, 10 cm height), looking in two possible GDs (left, right), resulting in 24 possible face stimuli. Along with faces, we presented two possible target stimuli (star or circle measuring 1.2 cm × 1.2 cm), presented in two different locations (either to the left or right from the face; star or circle measuring 1.2 cm × 1.2 cm), at 13 cm from the centre of the screen (Figure 13a). In total, face identity, gaze direction and cue location produced 96 different trials. We presented each trial twice, so that the experiment comprised a total of 192 trials (excluding training trials). To avoid fatigue effects, we included three breaks between four experimental blocks. After each block, participants could take a break and continue the

experiment when ready. We pseudo-randomized trial order in the same way as described for the GD task. We distributed all 192 trials between four experimental blocks (48 trials each). These followed a practice block of 10 stimuli that were randomly drawn from all possible stimuli. We set e-Prime such that trial order was pseudo-randomized in trial order within each block, so that trial order differed between participants.

Figure 13. Gaze Cueing experiment. Illustration of some examples of stimuli (not all combinations are shown) (a). In (b), we show the sequence of events for a Gaze Cueing trial.



An actual trial consisted of the presentation of a fixation cross (0.8 cm × 0.8 cm; for 500 ms) followed by a face stimulus. A target stimulus appeared near the face 400 ms later. Both face and target remained on the screen for a maximum of 1200 ms (Figure 13b). During this time, participants had to indicate whether the target stimulus was a star or a circle by pressing the corresponding button ("up arrow" or "down arrow") with the dominant hand. We counterbalanced response buttons between participants: half responded with the "arrow up" and "arrow down" button for a circle and star target, respectively, the other half responded with the "arrow up" and "arrow down" button for the star and circle target, respectively. As described in Jones et al. (2011), we explicitly instructed participants not to take into account the GD of centrally presented faces. The experimenter stayed with participants during the practice block to make sure the subject understood the instructions. Then, the experimenter left the testing room for the duration of the experimental blocks. We recorded percent correct responses (accuracy) and RTs for correct responses for each participant and the 12 conditions separately. The task took about 12 min to complete.

5.2.4 OVERALL PROCEDURE

Within two weeks prior laboratory testing, participants received detailed study information and provided written informed consent. Subsequently, participants filled in the demographic and self-report questionnaires online. In the laboratory session, they performed the computer tasks in a given order (GD task followed by the GC task). Participants were seated 57 cm from the computer screen (eye-screen distance).

5.2.5 DATA ANALYSES

We initially tested 78 participants, but excluded 10 participants because of incomplete questionnaires, did not perform the computer tasks, or made an excessive amount of errors (when compared to the remaining population). The descriptives and final analyses are based on the remaining 68 participants (women *n*=32). They had a mean (±SD) age of 21.60 (±2.33). For RT data from both tasks, we deleted individual RTs that were 3 SD above the individual mean (< 5% of trials per participant). Following previous studies, we used the accuracy and RT data for the GD task (Wallace et al., 2006, 2010), and the RT data only for the GC task (Jones et al., 2011). In both gaze performance tasks, participants' accuracy rate was close to ceiling (GD task: 94% ±10% correct responses, GC task: 95% ±6% correct responses). Mean RTs amounted to 532.80±97.14 in GD task, and to 505.67±56.13 in GC task. From these participants, we had complete data for questionnaires as well as demographic information. We computed descriptives of questionnaires and behavioural scores comprising means and standard deviations (SD).

To account for Mentalistic traits, we adopted a double strategy. **For the first strategy, we once computed** two PC scores representing "shared" and "diametrical"/Mentalistic traits among autistic and schizotypic ones (Dinsdale et al., 2013; Sierro, Rossier, Mason et al., 2016). We also selected schizotypic and austic traits representing hyper-Mentalistic (UnEx) and hypo-Mentalistic traits (ComMind, Ima), respectively. The PC scores were based on previous PCAs, in which questionnaire subscale scores were weighted and compiled into "shared" and "diametrical" components (see Sierro, Rossier & Mohr, 2016 for details). In short, to obtain the two PC scores, we multiplied each AQ-K and sO-LIFE dimension score with the respective loadings obtained from the previous PCA, before summing them into PC scores (Chapter 3; see Dinsdale et al., 2013). Based on the latter studies, one PC score represents the "Shared Features" (PCSF-K) and another PC score represents the "Diametrical Features" (PCDF-K). Higher PCSF-K scores reflect more pronounced negative traits (for schizotypy e.g. IntAn; for autistic social deficits e.g. SocSki).

Higher PCDF-K scores reflect higher positive schizotypic traits (e.g. UnEx) over autistic mentalizing deficit traits (e.g. ComMind).

For the second strategy, we chose positive schizotypy (UnEx) to represent hyper-Mentalistic features and autistic mentalizing deficits to represent hypo-Mentalistic features (ComMind, Ima; Sierro, Rossier & Mohr, 2016). We predicted that *higher* positive schizotypy and *higher* autistic mentalizing deficits would associate with, respectively, *increased* versus *decreased* gaze sensitivity. For this reason, we retained PCSF-K (as a control), PCDF-K, UnEx, ComMind and Ima as predictors for gaze processing behaviour.

For GD and GC tasks, we first tested whether our population showed the expected gaze performance biases. For the GD task, we performed separate repeated measures ANOVAs, one on Accuracy and one on RTs, with direction (Centre, Averted) and stimulus type (Arrows, Whole-Faces, Eyes-only) as repeated measures[20]. We report effect sizes throughout (generalized eta squared i.e. η_G^2; Olejnik & Algina, 2003). We also report results of planned *t*-tests comparing Center vs. Averted conditions, Social (Arrows) vs. Non-social ones (Whole-face, Eyes-only), as well as Whole-face vs. Eyes-only. When sphericity assumption was met, we used Tukey post-hoc test, otherwise we used *t*-tests with Bonferroni correction. For the sake of simplicity and reproducibility with Wallace et al. (2006), we computed indices representing the gain of Accuracy and RTs that controls experienced f.i. in Center as compared to Averted gaze conditions, and in Non Social as compared to Social conditions (Wallace et al., 2006). For the GC task, we determined congruent trials (GD matches target side, e.g. left gaze and left-sided target) and incongruent trials (GD does not match target side, e.g. left gaze and right-sided target; see also Jones et al., 2011). We subtracted RTs of congruent trials from RTs of incongruent trials obtaining one index (GC Index) representing the time costs of incongruent vs congruent attentional redirection by gaze. Thus, higher GC indices indicate stronger GC effects, i.e. a greater liability to have one's attention redirected by an observed gaze. To test for a general GC effect, we performed a unilateral one-sample *t*-test against $\mu=0$ on the GC Index. We postulated that GC indices would be superior to zero and positive ($\mu>0$). Following the literature (see Bayliss, Di Pellegrino et al., 2005), we compared the GC indices between men and women and used a two-sample independent *t*-test. Given a trend towards significance, we performed one-sample t-tests against $\mu=0$ for each sex separately. Additionnally,

20 For GD task, we first performed a one-way between factors ANOVA on RTs performance as dependent variable with "response button" and "symbol type" as between factors variable. Since neither "symbol type" ("star", "circle"), nor counterbalancement of response button influenced the performance, these variables were not further taken into account.

we compared the magnitude of our GC Index with the one in our reference task (Jones et al., 2011) using independent *t*-tests.

Subsequently, we wanted to test if autistic and schizotypic dimension scores predict GD and GC performance biases using regression analyses, with either PC scores or specific dimension scores (UnEx, ComMind, Ima) as outcome measures. In particular, we assessed whether (i) *higher* schizotypic and autistic negative trait scores (IntAn, SocSki), (ii) *lower* positive schizotypic scores (UnEx), and (iii) *higher* autistic mentalizing deficit traits (ComMind, Ima) predict *lower* performance at GD and GC measures.

For GD, we retained Accuracy and RT indices for Center vs. Averted, and Social vs. Non Social comparison indices[21]. Wallace et al. (2006, 2010) reported a GD effect for direct gaze in ASD and BAP, and center gaze conditions distinguished between ASp and neurotypicals. Hence, we also retained Accuracy and RT behavioural variables for Center:Whole-Face and Center:Eyes-Only conditions. In a first approach, we performed multiple regressions with aforementioned GD indices as outcome variables, and personality variables as predictor variables, once using the compiled PC scores (PCSF-K, PCDF-K) and once the individual, pre-selected Mentalistic dimension scores (UnEx, ComMind, Ima). In a second approach, we performed similar multiple regressions, but added the aforementioned center gaze GD variables as outcome variables. For the GD regression models, we added the personality variables in one step as predictor variables, because sex was not considered relevant to this task (Wallace et al, 2006, 2010).

For GC, we performed multiple regression analyses (the same personality scores as predictor variables) with the GC index for RTs (Bayliss, Di Pellegrino et al., 2005) as outcome variable. Here, we included sex as a predictor variable, because GC indices showed a trend towards a sex effect in neurotypicals (see also Bayliss, Di Pellegrino et al., 2005). Hence, we performed hierarchical regressions with Sex at the first step of the model, personality variables at the second step, and the sex × personality interaction at the third step. The comparison between the sexes was based on the difference between women and men GC indices (women – men GC indices), so that positive β or *b*'s would represent higher GC indices in women as compared to men, and vice-versa.

For the regression analyses, we report the whole models' R^2, F, df, p-values in the text in addition to the ΔR^2 for hierarchical regressions. For each significant regression model, we also report β (standardized slopes), *b* (slopes), standard errors (se), t, df, p-values for each regression predictor. All predictors of hierarchical regressions were centered. All variables were univariate normal (i.e.

[21] We decided to drop Whole-Face:Eyes-Only comparison indices and all subsequent regressions with personality variables, to reduce the number of regressions and potential false positives (all p's>.05).

no values exceeded ±3.27), except for GD's Social vs Non Social conditions. We report violations of regression assumptions. For all analyses, we kept the statistical significance threshold (*p)* at α<.05. All statistical analyses and graphs were performed with R (R Core Team, 2014).

5.3 RESULTS

5.3.1 PARTICIPANTS AND SELF-REPORT QUESTIONNAIRE SCORES

The mean (±sd) age differed between sexes with men (*n*=36; 22.22±2.51) being older than women (*n*=32; 20.91±1.92), Welch independent sample t-test (*t*(64.67)=-2.40, *p*=.019, *d*=-0.58). Descriptives for questionnaire scores are reported in Table 23. In this Table, we also show that questionnaire scores were comparable to scores from normative samples (Sierro, Rossier, Mason et al., 2016; Sierro, Rossier & Mohr, 2016).

5.3.2 GENERAL BEHAVIOUR IN THE GAZE PROCESSING TASKS

5.3.2.1 GAZE DIRECTION TASK

Descriptives for GD task are reported in Table 24. The repeated measures ANOVA on GD (Central, Averted) and Stimulus Type (Whole-Face, Eyes-Only, Arrows) as within-subject factors on Accuracy showed a main effect of Stimulus Type (ε_{HF}= 0.795, F(1.59,107)=14.60, p<.001, η_G^2=.04), no significant main effect of Direction, F(2,134)=0.45, p=.503, η_G^2=.00), and a significant Stimulus Type × Direction interaction (ε_{GG}=0.949, F(1.9,127)=6.69, p=.002, η_G^2=.02). Figure 14 represents the Accuracy performance in the GD task. Planned t-tests showed that Accuracy did not differ between Center vs Averted condition (p>.05), whereas Accuracy was significantly higher for Non-social (Arrows) than Social (Whole-Faces, Eyes-only) conditions (p<.05). Accuracy for Whole-Faces was superior to Accuracy for Eyes-only (p<.05). Also, Accuracy for Non Social (Arrows) vs Social stimuli (Whole-Face, Eyes-only) was stronger for the Averted as compared to the Center condition (p<.05). Post-hoc comparisons were significant throughout (all p-values < 0.05): Accuracy was higher for Arrows (0.97±0.07) as compared to both Whole-Faces (0.93±0.10) and Eyes-Only (0.91±0.12) with performance being also superior for Whole-Faces than Eyes-Only. Post-hoc comparisons showed that the significant interaction between Stimulus Type × Direction originated from a superior performance for the Averted:Arrow condition as compared to Center:Eyes-Only, Center:Arrow and Averted:Eyes-Only conditions, respectively (*p's*<.05). When considering all Averted conditions, we found that performance was superior for Averted:Arrows

than Averted:Whole-Face and Averted:Eyes-Only (*p's*<.05), respectively, whereas Averted:Whole-Face and Averted:Eyes-Only did not differ (*p's*>.05; all other *p's*>.05).

Table 23: Descriptive values for questionnaire dimension scores gathered in the current study, and compared to a normative samples. Results comparing the current questionnaire scores and the questionnaire scores from the normative data are also provided (student t-test statistics)

		The present study			Normative Sample					
Instrument	Dimension	mean	sd	α	mean	sd	*t*	*df*	*p*	*d*
sO-LIFE	Global	15.07	6.21	.88†	14.45†	6.28	0.79	1114	.428	0.10
	UnEx	3.82	2.50	.83†	3.73†	2.59	0.29	1114	.775	0.04
	CogDis	5.24	2.73	.84†	5.53†	2.82	-0.85	1114	.400	-0.10
	IntAn	2.66	2.03	.72†	2.14†	1.76	**2.06**	**1114**	**.043**	**0.27**
	ImpNon	3.35	1.69	.74†	3.04†	2.04	1.45	1114	.152	0.17
AQ-K	SocSki	16.56	4.63	.79††	16.13††	3.91	0.75	987	.458	0.10
	AttDet	11.99	2.96	.66††	11.43††	3.06	1.50	987	.137	0.19
	ComMind	10.07	2.15	.67††	10.10††	2.40	-0.11	987	.913	-0.01
	Ima	8.85	2.61	.49††	8.92††	2.36	-0.21	987	.831	-0.03
	RRBeh	10.93	2.04	.35††	11.12††	2.14	-0.74	987	.462	-0.09
PC score	PCSF-K	27.48	5.53	–	26.97††	5.02	0.74	987	.463	0.10
PC score	PCDF-K	17.91	5.63	–	16.90††	6.18	1.42	987	.160	0.17

mean=mean; sd=standard deviation; *t*=Student's t-test statistic; *p*= Student's *t*-test p-value; *d*= Cohen's *d* effect size

† normative sample based on *n*=1'048 respondents (Sierro, Rossier, Mason et al., 2016)

†† normative sample based on *n*=921 respondents (Sierro, Rossier & Mohr, 2016)

PCSF-K: PC score of Shared Features with AQ-K; PCDF-K: PC score of Diametrical Features with AQ-K

Global: total sO-LIFE score; UnEx: Unusual Experiences; CogDis: Cognitive Disorganization; IntAn: Introvertive Anhedonia; ImpNon: Impulsive Nonconformity

SocSki: Social Skills deficits; AttDet: Attention to Details; ComMind: Communication/Mindreading deficits; Ima: Imagination deficits; RRBeh: Routines/Repetitive Behaviours

Table 24: Performance measures (accuracy and RT) obtained from the GD and GC task (n = 68). Results are given as means and standard deviations (sd) for the different gaze conditions.

		Accuracy		RT	
	Measures	mean	sd	mean	sd
GD	Center Arrow	0.95	0.09	527.92	42.23
GD	Center Eyes-Only	0.93	0.07	559.54	30.34
GD	Center Whole-Face	0.94	0.08	555.87	35.34
GD	Averted Arrow	0.98	0.06	473.30	36.93
GD	Averted Eyes-Only	0.90	0.08	546.58	38.10
GD	Averted Whole-Face	0.93	0.07	533.61	32.53
GD	Center vs Averted	0.03	0.39	174.80	231.43
GD	Whole-Face vs Eyes-Only*	0.07	0.19	24.21	71.74
GD	Non Social vs Social*	0.29	0.45	317.03	223.46
GC	Congruent	–	–	495.99	47.09
GC	Incongruent	–	–	500.17	46.04
GC	Gaze Cueing Index	–	–	4.16	13.37

* For RT conditions: Social vs Non Social costs; Eyes-Only vs Whole-Faces costs.
GD: Gaze Direction; GC: Gaze Cueing of attention

The analogue ANOVA on RTs showed significant main effects of Stimulus Type (ε_{HF}=0.758, F(1.52,102)=76.8, p<.001, η_G^2=.06) and Gaze Direction (F(1,67)=38.5, p<.001, η_G^2=.03) as well as a significant Stimulus Type × Gaze Direction interaction (F(2,134)=24.5, p<.001, η_G^2=.01. Planned t-tests showed that RT were significantly slower for the Center as compared to the Averted Gaze condition (p<.05). We also found that non-social stimuli (Arrows) were processed significantly faster than social stimuli (Whole-faces, Eyes-only) (p<.05). Whole-Face stimuli were processed significantly faster than Eyes-only stimuli (p<.05). Also, the slower RTs for Center vs Averted was stronger for Non Social stimuli (Arrows) as compared to social stimuli (Whole-Face, Eyes-only; p<.05). Figure 15 represents RT performance for the GD task. Post-hoc comparisons showed that for Stimulus Type, Arrows were processed faster than both Eyes-Only and Whole-Faces, and also that Whole-Faces were processed faster than Eyes-Only (all

p's<.05). Post-hoc tests also showed that, for Direction, Averted stimuli were processed significantly faster as compared to Center stimuli (all *p-values* < .05). Also, Center:Eyes-Only and Center:Whole-Faces took significantly more time to be processed as compared to Averted:Whole-Faces. Moreover, for Center stimuli, Center:Arrow were processed significantly faster than Center:Eyes-Only and Center:Whole-Faces, respectively (*p's*<.05), whereas Center:Eyes-Only and Center:Whole-Faces did not differ (*p*>.05). Likewise, for Averted stimuli, Arrows were processed significantly faster than both Eyes-Only and Whole-Faces (*p's*<.05), whereas Eyes-Only and Whole-Faces did not differ (*p*>.05; all other *p's*>.05).

Based on these results, we computed for both Accuracy and RT data respective indices representing two types of contrasts: "Center vs Averted" and "Social vs Non-Social". For both Accuracy and RT, the "Center vs Averted" index accounted for the advantage in processing Averted as compared to Center stimuli (Left and Right, taken together), irrespectively of their category (Arrows, Eyes-Only or Whole-Faces). For the "Non-Social vs Social" index on Accuracy, we accounted for the advantage when processing Non Social stimuli (Arrows) as compared to Social (Eyes-only, Whole-Face), irrespectively of Direction. Conversely, for RT, the "Social vs Non Social" index accounted for the advantage for processing Non Social (Arrows) over Social stimuli (Eyes-only, Whole-Face), irrespectively of Direction (see Table 24 for descriptives).

Figure 14. Mean accuracy in the Gaze Direction task [proportion correct] as a function of stimulus Type and Direction (error bars represent one standard error of the mean).

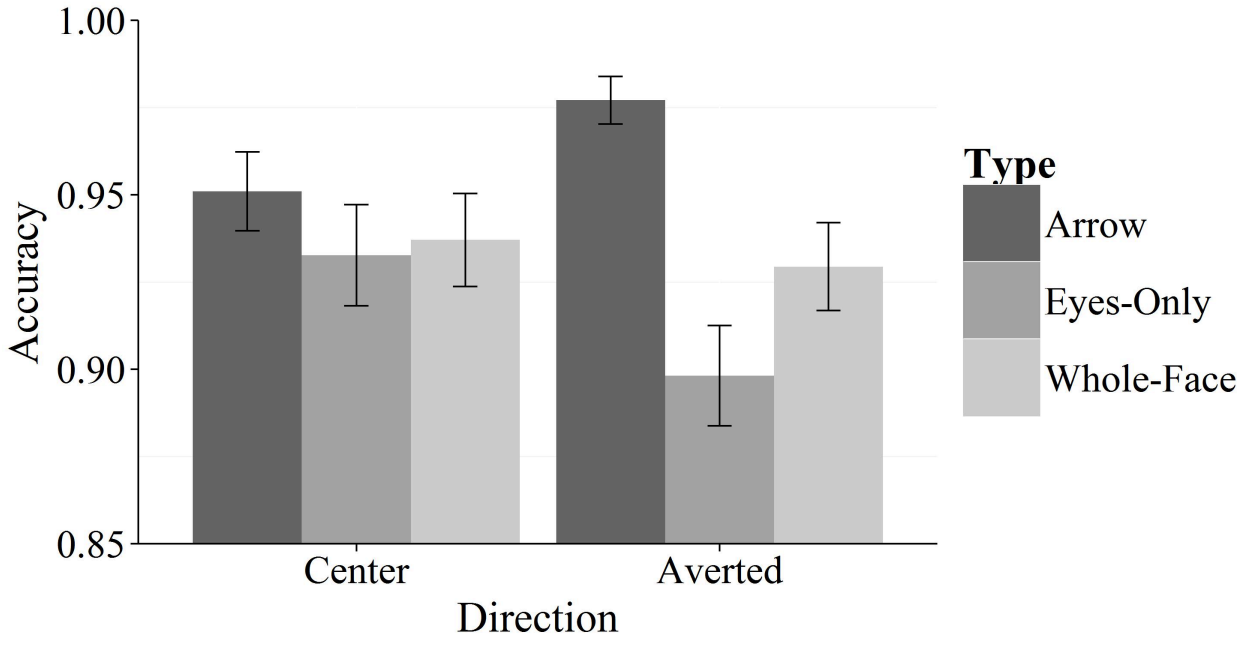
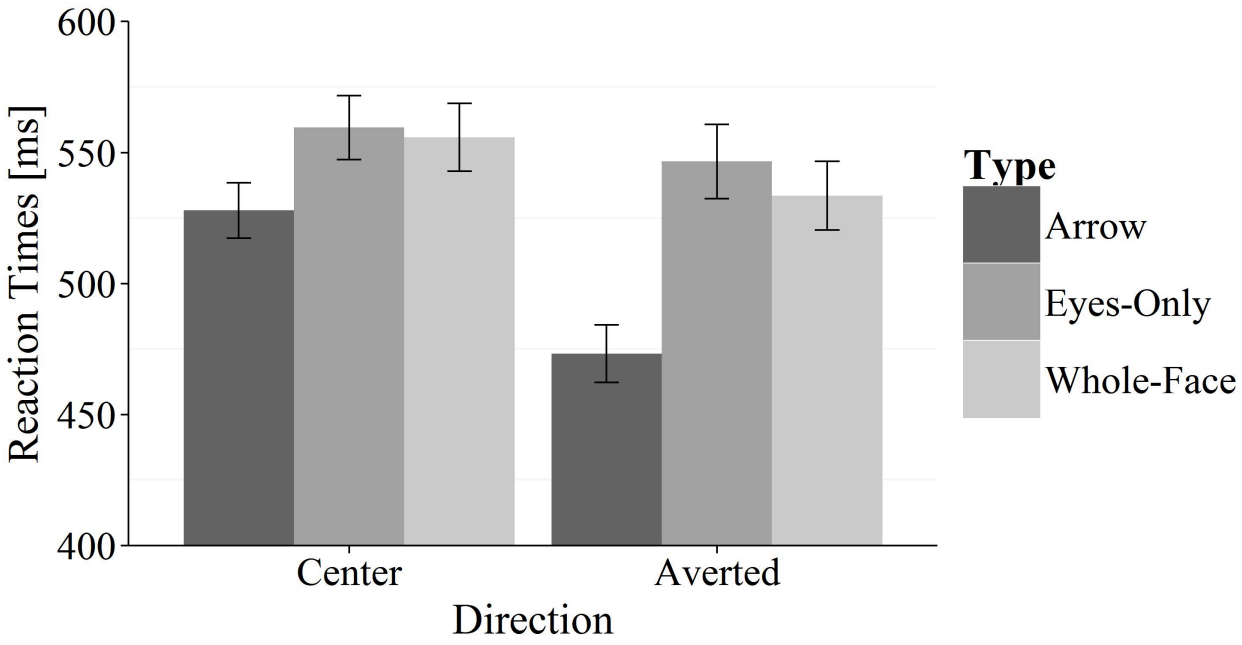


Figure 15. Mean reaction times [ms] in the Gaze Direction task as a function of stimulus Type and Direction (error bars represent one standard error of the mean).



5.3.2.2 GAZE CUEING TASK

Descriptives for the GC task are reported in Table 24. The one-sample *t*-test on the GC index (against μ=0) was significant ($t(67)=2.56$, $p<.006$, $d=0.31$). The positive GC index score (4.16±13.36) indicates that participants yielded faster responses for consistent as compared to inconsistent trials. When comparing our GC index with the GC index (8.75±12.52) of Jones et al. (2011), the difference was not significant ($t(86)=1.43$, $p=.158$, $d=0.36$). As the mean GC index tended to be larger for women (7.51±11.7) than men (1.17±14.19) ($t(66)=2.00$, $p=.050$, $d=0.48$), we tested whether the GC effect was significant (i.e. whether GC index was significantly larger than 0) for women but not men. Womens' GC index (7.50±11.71) was significantly larger than zero ($t(31)=3.62$, $p<.001$, $d=1.30$), while mens' GC index did not significantly differ from zero (1.17±14.19; $t(35)=0.50$, $p=.312$, $d=0.17$).

5.3.3 RELATIONSHIPS BETWEEN GAZE PROCESSING TASKS AND PERSONALITY MEASURES

5.3.3.1 REGRESSION ANALYSES

5.3.3.1.1 GAZE DIRECTION TASK…

5.3.3.1.1.1 …USING GD INDICES FOR ACCURACY AND RTs

All models for Accuracy and RT indices were non-significant, apart from some trends (see Table 25 for each model's R^2's, and each predictor's β's). For differences in accuracy between Center and Averted conditions, neither the model with PC scores (i.e. PCSF-K and PCDF-K scores) (R^2=.032, F(2,65)=1.08, p=.344), nor the analogue model with Mentalistic dimensions (i.e. UnEx, ComMind, Ima) (R^2=.036, F(3,64)=0.79, p=.503) were significant. For differences in accuracy for Social vs Non-Social conditions, the model with PC scores was not significant (R^2=.006, F(2,65)=0.19, p=.827), but the analogue model with Mentalistic dimensions showed a statistical trend (R^2=.103, F(3,64)=2.45, p=.072). Higher Ima scores associated with a larger difference between accuracy for Social and Non-Social conditions (β=.289, b=0.050, se=0.021, t(64)=2.43, p=.018). Although no leverage effect was reported, normality of residuals and homoscedasticity were not respected, which would limit the generalizability of the model.

For GD RT indices, we observed that neither PC scores nor Mentalistic scores predicted performance measures. For differences in RT between Center vs Averted conditions, neither the model with PC scores (R^2=.077, F(2,65)=2.70, p=.075), nor the analogue model with Mentalistic dimension scores (R^2=.095, F(3,64)=2.25, p=.091) were significant, despite both showing statistical trends. The first trend showed that higher PCSF-K scores associated with a larger RT difference between Center and Averted gaze (β=.276, b=11.918, se=5.808, t(65)=2.05, p=.044), whereas higher PCDF-K scores showed the opposite trend (β=-.257, b=-10.910, se=5.703, t(65)=-1.91, p=.060). The model on Mentalistic dimensions did not show significant relationships. In both cases, normality of residuals, homoscedasticity were respected and there was no leverage effect. For differences in RT between Social and Non-Social conditions, neither the model using PC scores (R^2=.029, F(2,65)=0.95, p=.390), nor the model using Mentalistic dimension scores (R^2=.061, F(3,64)=1.38, p=.256) were significant.

5.3.3.1.1.2 …USING THE ORIGINAL GD ACCURACY AND RTs MEASURES

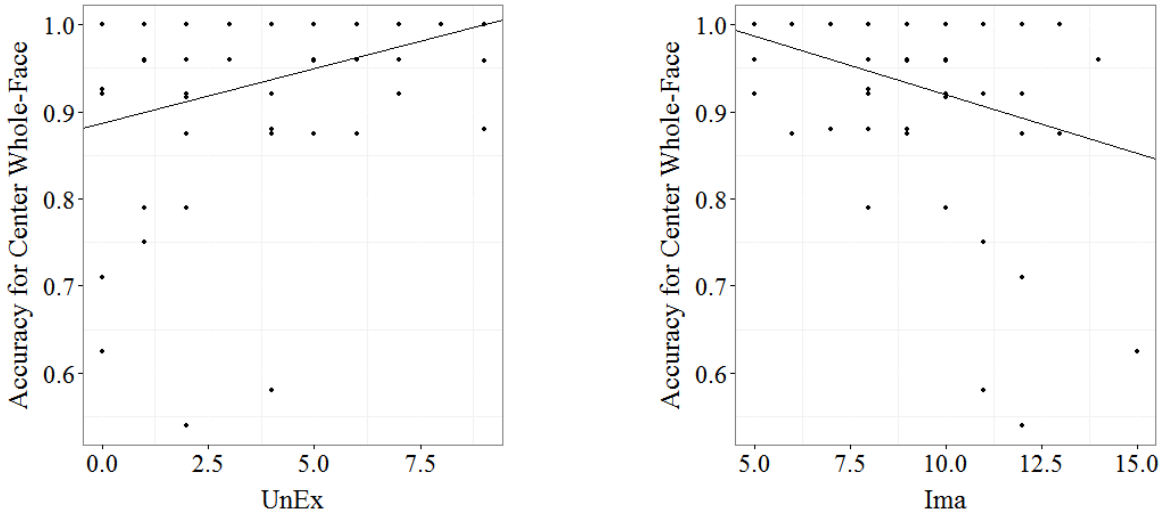
The models for Accuracy and RT variables predicted by either PC or Mentalistic scores were not significant (all *p*'s>.05), with the exception of a significant result for Accuracy Center:Whole-Faces with Mentalistic scores as predictor variables (see Table 25 for each model's R^2's, and each predictor's β's). The regression model on PC scores did not account for Accuracy Center:Whole-Face performance (R^2=.05, F(2,65)=1.89, p=.160), but the regression model on the Mentalistic dimensions was significant (R^2=.21, F(3,64)=5.73, p=.002): *Higher* UnEx associated with *better* Accuracy for Center:Whole-Face performance (β=.31, b=0.013, se=0.005, t(64)=2.70, p=.009; see Figure 16a), whereas *higher* Ima associated with *lower* Accuracy Center:Whole-Face performance (β=-.35, b=-0.013, se=0.004, t(64)=-3.12, p=.003; see Figure 16b). ComMind was unrelated to Accuracy Center:Whole-Face performance (β=.10, b=0.005, se=0.005, t(64)=0.86, p=.391). Although no influential case was detected (all Cook's distances <0.5), regression diagnostics showed that residuals were not normally distributed and homoscedasticity assumption was violated (these observations cast doubt onto the generalizability of this model).

Table 25. Standardized β of regressions on Gaze Direction Indices and variables with centered personality dimensions as predictors (n=68).

			β			β		
	Outcomes	R^2	PCSF-K	PCDF-K	R^2	UnEx	ComMind	Ima
Accuracy	Center vs Averted	.032	-.187	.154	.036	.118	-.033	-.130
Accuracy	Non Social vs Social	.006	.042	.047	*.103°*	-.068	-.166	**.289***
RT	Averted vs Center	*.077°*	**.276***	*-.257°*	*.095°*	-.090	*.241°*	.110
RT	Social vs Non Social	.029	.178	-.023	.061	-.054	.135	.177
Accuracy	Center Whole-Face	.055	-.191	*.250°*	**.212***	**.312***	.100	**-.348***
Accuracy	Center Eyes-Only	.054	*-.227°*	.220	.073	.176	-.030	-.186
RT	Center Whole-Face	.042	.104	.136	.083	.134	.130	*.234°*
RT	Center Eyes-Only	.036	.126	.093	.079	.120	*.226°*	.157

° *p*<.100; * *p*<.05
PCSF-K: PC score of Shared Features with AQ-K; PCDF-K: PC score of Diametrical Features with AQ-K
UnEx: Unusual Experiences; ComMind: Communication/Mindreading deficits; Ima: Imagination deficits

Figure 16. Scatter plot depicting the relationship between the accuracy for Center Whole-Faces and (a) UnEx scores and (b) Ima scores .



5.3.3.1.2 GAZE CUEING TASK

For GC effects, we observed that both "diametrical" and "shared" traits significantly predicted gaze sensitivity, although somewhat differently in women and men. We report the regression model statistics in the text together with the detailed outcome for each predictor in Table 26. At the first step of our regression models, sex tended to improve the model with women showing a trend towards larger GC effects than men (R^2=.06, F(1,66)=3.98, p=.050; β=-.12, b=3.17, se=1.59, t(66)=2.73, p=.050). At the second step, PC scores did not predict GC effects (R^2=.08, F(3,64)=1.77, p=.162). At the third step, the PC scores accounted for variance in the GC Index (R^2=.18, F(5,62)=2.68, p=.030). Yet, neither PCSF-K nor PCDF-K scores significantly predicted GC effects. Interactions between Sex and both PCSF-K and PCDF-K highlighted an important role of sex in GC (Table 26). When performing regressions *separetely* for men and women, however, we found that the previous results were driven by men. In women, the model on PC scores was not significant (R^2=.08 F(2,29)=1.22, p=.310). Neither PCSF-K (β=.34, b=.647, se=0.414, t(29)=1.56, p=.130), nor PCDF-K (β=-.21, b=-0.401, se=0.418, t(29)=-0.96, p=.345) associated with GC effects in women. In men, the model on PC scores tended to be significant (R^2=0.16, F(2,33)=3.10, p=.059). Here, higher PCSF-K tended to associate with a *smaller* GC effect (β=-.34, b=-0.961, se=0.481, t(33)=-2.00, p=.054), whereas higher PCDF-K tended to associate with a *larger* GC effect (β=.34, b=.93, se=0.457, t(33)=2.04, p=.050).

Table 26. Hierarchical regressions on Gaze Cueing Index with sex and centered personality dimensions as predictors (n=68).

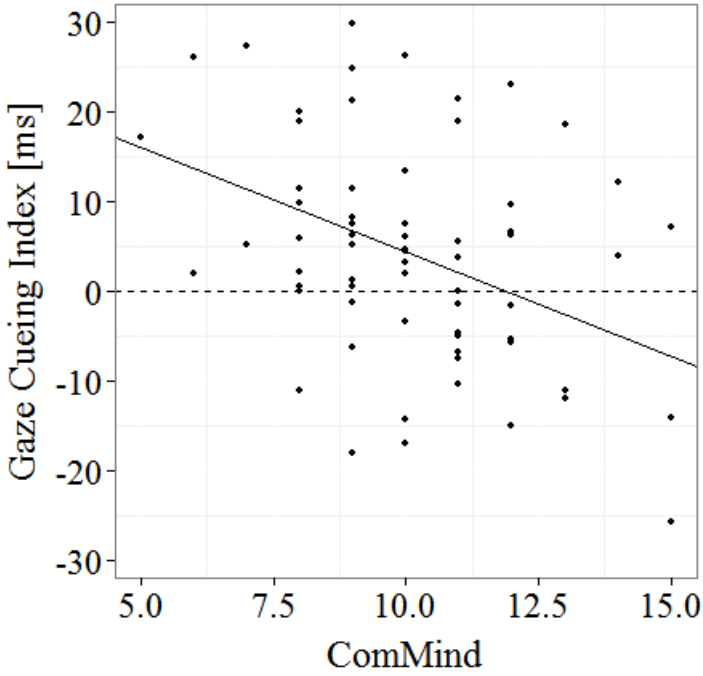
	Predictors	ΔR^2	β	*b*	se	*t*(64)	*p*
Step 1		**0.06***					
	Sex		*.12*	*3.062*	*1.545*	*1.98*	*.052*
Step 2		0.02					
	PCSF-K		-.09	-0.211	0.328	-0.64	.522
	PCDF-K		.16	0.374	0.322	1.16	.250
Step 3		**0.10***					
	Sex × PCSF-K		**.33***	**4.442**	**1.762**	**2.52**	**.014**
	Sex × PCDF-K		**-.14***	**-3.744**	**1.757**	**-2.13**	**.037**
Total R^2		**.18***					
Step 2		**.16***					
	UnEx		-.14	-0.758	0.632	-1.20	.235
	ComMind		**-.37**	**-2.316**	**0.726**	**-3.19**	**.002**
	Ima		-.14	-0.712	0.579	-1.23	.223
Step 3		.07					
	Sex × UnEx		.02	0.394	0.628	0.63	.533
	Sex × ComMind		.21	1.106	0.796	1.39	.170
	Sex × Ima		*.18*	*1.128*	*0.576*	*1.96*	*.055*
Total R^2		**.29***					

° $p \leq .100$; * $p < .050$;** $p < .001$
PCSF-K: PC score of Shared Features with AQ-K; PCDF-K: PC score of Diametrical Features with AQ-K
UnEx: Unusual Experiences; ComMind: Communication/Mindreading deficits; Ima: Imagination deficits

For GC effects, at the second step, the model using mentalistic dimension scores as predictors was significant (R^2=.21, F(4,63)=4.25, p=.004). At the third step, the complete model with additional interaction with sex was also significant (R^2=.29, F(7,60)=3.43, p=.004). Higher ComMind scores predicted a smaller GC effect (Figure 17). Also, the Sex × Ima interaction tended to be significant. Separate regression analyses for men and women confirmed an interaction with sex. In women, the model on the Mentalistic dimensions was not significant (R^2=.01, F(3,28)=0.14, p=.937). Neither UnEx (β=-.04, b=-0.166, se=0.859, t(28)=-0.19, p=.848), ComMind (β=-.08, b=-

0.581, se=1.383, t(28)=-0.42, p=.678), nor Ima (β=.09, b=0.356, se=0.779, t(28)=0.457, p=.651) scores associated with GC effect in women. In contrast, in men, the model on the Mentalistic dimensions was significant (R^2=.38, F(3,32)=6.54, p=.001). Higher ComMind (β=-.50, b=-2.794, se=0.826, t(32)=-3.38, p=.002) as well as Ima (β=-.32, b=-1.90, se=0.846, t(32)=-2.26, p=.032) scores predicted a smaller GC effect, while UnEx scores were unrelated to GC effects (β=-.15, b=-0.954, se=0.916, t(32)=-1.04, p=.305).

Figure 17. Scatter plot of Gaze Cueing Index as a function of ComMind scores.



5.4 DISCUSSION

Gaze processing is key to intact social cognition and abnormal in both autism and schizophrenia. It is unknown whether ASp and PSp gaze processing deviations reflect "shared"/similar (King & Lord, 2011) or opposite "diametrical" social cognition profiles (Crespi & Badcock, 2008). In a within-subject design, we investigated in a French speaking Swiss sample whether gaze processing behaviour matches diametrical Mentalistic traits as assessed with validated French autistic and schizotypic trait questionnaires (Sierro, Rossier & Mohr, 2016). Using regression models, we confirmed that larger gaze sensitivity could be explained by larger diametrical Mentalistic traits (PCDF-K scores). Higher hyper-Mentalistic positive schizotypy (high

PCDF-K scores) associated with larger gaze sensitivity, whereas high autistic hypo-Mentalistic traits (low PCDF-K scores) associated with lower gaze sensitivity. Since gaze sensitivity varied along a continuum of diametrical "Mentalistic" traits, our results support the diametrical model of ASp and PSp (Crespi & Badcock, 2008). At the same time, having larger autistic hypo-Mentalistic traits (ComMind, Ima) and being male best explained lower gaze sensitivity in GC, replicating and clarifying similar previous findings (Alwall et al., 2010; Bayliss, Di Pellegrino et al., 2005).

Beyond using the PC scores to directly test the diametrical model, we also found a decreased accuracy for straight ahead looking whole-faces as a function of larger Ima scores in GD, and lower GC as a function of ComMind, mostly in men. These latter findings support lower gaze sensitivity as an endophenotype of ASD (Matsuyoshi et al., 2014; Scheeren & Stauder, 2008; Wallace et al., 2010). In contrast, higher positive schizotypy (UnEx) associated with larger gaze sensitivity in the GD but not in the GC task. The lack of associations between higher hyper-Mentalistic positive schizotypy traits and higher gaze cueing replicates previous inconsistent findings in SSD individuals (Akyiama et al., 2008; Hooker & Park, 2005; Langdon et al., 2006; Magnée et al., 2011). We finally found some evidence that "shared" (PCSF-K) traits tended to associate with *lower* gaze sensitivity, mirroring previously reported associations between larger total autistic traits (notably introverted and negative ones) and *lower* gaze sensitivity (e.g. Alwall, Johansson, & Hansen, 2010; Bayliss, Di Pellegrino et al., 2005; Ponari, Trojano, Grossi, & Conson, 2013). This latter finding suggests that gaze processing deficits may also owe to "shared" traits between ASp and PSp (King & Lord, 2011).

Overall, variation in gaze sensitivity seems better explained by "diametrical" Mentalistic traits (PCDF-K) rather than "shared" negative traits (PCSF-K). The "shared" negative traits (PCSF-K) only tended to associate with a gaze under-sensitivity. For the GD task, larger PCSF-K scores tended to associate with smaller Center vs Averted Accuracy. For the GC task, despite a significant interaction between PCSF-K and sex, larger PCSF-K scores only tended to associate with a smaller GC effect in men. These findings correspond with reports of a smaller GC effect associated with larger introversion scores (Ponari et al. (2013). Indeed, negative "shared" schizotypy and autistic traits (e.g. SocSki, IntAn) represent introverted traits, as indicated by negative correlations with Extraversion (Sierro, Rossier & Mohr, 2016). In contrast, higher gaze sensitivity (GD and GC) associated with higher hyper-Mentalistic traits (PCDF-K), and lower hypo-Mentalistic traits (PCDF-K), supporting the diametrical model (Crespi & Badcock, 2008). In the GD task, Accuracy for identifying direction of direct gaze (Center:Whole-Faces) was higher with *higher* positive schizotypy (UnEx) and *lower* autistic Ima scores. However, in the GC task, the association between a larger Mentalistic score (PCDF-K) and a larger GC effect stemmed from men and autistic

mentalizing deficit traits (ComMind; see below). Reduced GC might be a "distinct" feature, that is autistic specific. Although "shared" negative traits may bear some relevance with respect to gaze sensitivity, the "diametrical" Mentalistic traits, in particular the hypo-Mentalistic subset, could better explain gaze sensitivity (Crespi & Badcock, 2008), supporting an integrative model combining "shared", "diametrical" and "distinct" features.

Our results imply that existing ASp-PSp relationship models ought to be combined to explain all the relationship features of the disorders. Models emphasizing shared deficits and shared aetiologies between ASD and SSD (King & Lord, 2011; Rausch & Johnson, 2008) may explain the association of social introverted/negative traits with gaze under-sensitivity. Introverted/negative traits may reflect shared social motivation deficits (social anhedonia), possibly expressed in introversion (Ponari et al., 2013). Among the hypotheses of atypical autistic gaze processing (Senju & Johnson, 2009 for a review), the amygdalar hypo-arousal hypothesis of atypical eye gaze may explain the present findings. Dawson et al. (2005) proposed that an early deficit in social motivation would cascade into a lack of activation of the brain's amygdalar and reward system. By inference, this would result in a difficulty to learn about the reward of social stimuli, and in turn in a fdysfunction of cortical specialization and social stimuli processing, notably gaze and face. Nevertheless, these models may not be sufficient to account for ASp-PSp relationships in gaze processing. The diametrical model's opposition between autistic hypo-Mentalism and schizotypic hyper-Mentalism (as PCDF-K score) might be necessary to explain, respectively, lower and higher gaze sensitivity (GC and GD). In addition to "shared" and diametrical features (see Chisholm et al., 2015), we should also consider "distinct" features. GC effects associated with hypo-Mentalistic autistic deficit traits (ComMind) and did not associate with hyper-Mentalistic positive schizotypy (UnEx) traits, suggesting that gaze under-sensitivity is for now a "distinct" feature of the autistic phenotype (Matsuyoshi et al., 2014; Wallace et al., 2010).

In contrast to autistic mentalizing deficit traits (ComMind, Ima), positive schizotypy only showed weak evidence of central gaze over-sensitivity, reflecting inconsistent findings about PSp gaze sensitivity in the literature (Akiyama et al., 2008; Hooker & Park, 2005; Langdon et al., 2006; Magnée et al., 2011; Pino, Mariano, Valchera, Valenti, and De Berardis, 2015). In the GD task, positive schizotypy (UnEx, as a trend for PCDF-K) associated with a larger Accuracy in Center Whole-Face, suggestive of increased gaze sensitivity. However, here, participants performed at ceiling, and several assumptions of the regression model were violated (residuals normality and homoscedasticity). In the GC task, larger UnEx scores were not associated with the GC effect, reflecting contradictory reports in schizophrenia (over-sensitivity: Langdon et al., 2006; under-sensitivity: Akiyama et al., 2008; Pino et al., 2015; no difference: Magnée et al., 2011). Positive

schizotypy did not associate with gaze under-sensitivity and may reflect gaze over-sensitivity to self-directed central stimuli, for instance at shortly displayed gazes (our GD experiment), as seen in schizophrenia patients (Hooker & Park, 2005; Langdon et al., 2006; Langdon, Seymour, Williams, and Ward, 2016; Tso et al., 2012). At higher display durations, positive schizotypy may instead associate with smaller GC, as seen in schizophrenia patients (Akiyama et al., 2008; Langdon et al., 2016; Pino et al., 2015), in particular in chronic patients (Dalmaso, Galfano, Tarqui, Forti, & Castelli, 2013), and be caused by a prolonged gaze examination (Magnée et al., 2011) rather than diminished gaze sensitivity.

Higher autistic mentalizing deficit traits (ComMind, Ima) associated with a *lower* liability to GC. This observation is compatible with previously reported findings in ASp, and stresses the relevance of hypo-Mentalistic traits in ASp (Bishop et al., 2006; Sierro, Rossier & Mohr, 2016). In the GC task, we found that with higher ComMind scores, participants (in particular men) showed a lower liability to have their attention redirected by gaze. In men only, Ima showed a similar association to smaller GC. Our results replicate and clarify the smaller GC effect reported in neurotypicals as a function of higher total autistic traits, especially in men (Bayliss, Di Pellegrino et al., 2005), in individuals with ASD (Kylliäinen & Hietanen, 2004; Ristic et al., 2005; Senju et al., 2004; Vlamings et al., 2005), and ASD children parents as compared to controls' parents (Scheeren & Stauder, 2008; Tajmirriyahi, Nejati, and Pouretemad, 2016). The involvement in reduced GC of the hypo-Mentalistic subset of autistic traits (i.e. mentalizing deficit traits: ComMind, Ima) corresponds to a previous report suggesting the involvement of lower empathy in reduced GC (Alwall et al., 2010). Such hypo-Mentalistic traits likely underlie the association of lower GC with larger total AQ scores (Bayliss, Di Pellegrino et al., 2005).

Autistic hypo-mentalistic traits may lower gaze sensitivity through various mechanisms, such as passive gaze avoidance (Senju & Johnson, 2009 for a review). In line with Scheeren and Stauder (2008), autistic mentalizing deficits may associate with a smaller attentional engagement to gaze resulting in lower gaze sensitivity. Among the hypotheses of atypical autistic gaze processing (see Senju & Johnson, 2009 for a review), the hypo-arousal model (Dawson et al., 2005; see also Klin et al., 2002), and the model positing a defect of a putative "communication intention detector" module (Baron-Cohen, 1995) do not seem relevant to explain a possible lack of attentional engagement to gaze. The existence of such a module was not confirmed (Itier & Batty, 2009), and hypo-arousal theory was criticized (Senju & Johnson, 2009; Tanaka & Sung, 2013). An active avoidance of eye gaze in those high in autistic mentalizing deficits traits is unlikely (hyper-arousal hypothesis; Dalton et al., 2005; Tanaka & Sung, 2013), because participants were instructed to fixate the stimuli, they did process stimuli, yet just with a lower accuracy for GD, and slower RTs

for congruent trials in GC. For these reasons, even though our design does not allow us to favour one over the other hypothesis, we consider it more plausible that gaze was passively ignored or insufficiently processed ('fast-track modulator' model, Senju & Johnson, 2009). Building on Senju and Johnson (2009), our results would reflect minor abnormalities in the subcortical route subserving quick processing of gaze, which would result in minor attentional engagement deficits in those high in autistic traits.

Autistic mentalizing deficit traits (ComMind, Ima) might reflect a continuum of gaze sensitivity as a candidate ASp endophenotype (Matsuyoshi et al., 2014; Sucksmith et al., 2011; Wallace et al., 2010), involved in ASp social deficits (Jellema et al., 2009). Deficits in a social cognitive empathizing dimension underlying autistic traits (Bishop et al., 2004; Sierro, Rossier & Mohr, 2016), such as mindreading (ComMind) and social imagination deficits (Ima) are expected to relate to gaze processing (Emery, 2000), for several reasons. First, gaze processing areas are anatomically connected to mentalizing brain networks (Kennedy & Adolphs, 2012). Second, abnormal central gaze processing endophenotype may originate in early life (Elsabbagh et al 2009; Dalton et al., 2007), remain until adulthood (Scheeren & Stauder, 2008; Wallace et al., 2010), and impair the development of mentalizing, resulting in social deficits (Itier & Batty, 2009; Jellema et al., 2009; Kuhn et al., 2010). In summary, our results suggest that sensitivity to gaze may vary on a continuum from high to low, could be a putative endophenotype of ASp (Matsuyoshi et al., 2014; Scheeren & Stauder, 2008; Sucksmith et al., 2011; Wallace et al., 2010), and be responsible for resultant social deficits (Itier & Batty, 2009; Jellema et al., 2009). Such social deficits might establish later in life as self-reported hypo-Mentalistic autistic deficits.

Although we confirmed the previously reported men-specificity of reduced GC as a function of autistic traits (e.g. Bayliss, Di Pellegrino et al., 2005; Scheeren & Stauder, 2008), effects of gender or cultural variables should not be dismissed. Indeed, we found smaller gaze sensitivity as a function of autistic mentalizing deficit traits (GD: Ima; GC: ComMind, Ima) in men but not in women, as previously reported in ASp for GD (Matsuyoshi et al., 2014), and GC (Bayliss, Di Pellegrino et al., 2005; Scheeren & Stauder, 2008). Irrespectively of autistic traits, several studies showed a superior gaze sensitivity in women as compared to men, either in GD or GC tasks (Alwall et al., 2010; Ashwin, Ricciardelli, & Baron-Cohen, 2009; Bayliss & Tipper, 2005; Deaner, Shepherd, & Platt, 2007). In addition, autistic scores are higher in men (e.g. Baron-Cohen, Wheelwright, Skinner et al., 2001), whereas EQ scores are higher in women (Wakabayashi et al., 2006b), possibly reflecting a female advantage in mentalizing (Christov-Moore et al., 2014). However, cultural or gender influences on questionnaire responding were reported for AQ (Broadbent et al., 2013), for EQ (Allison et al., 2011), and for negative schizotypy (Winterstein,

Ackerman et al., 2011). Aside from a general sex-difference in GC, either genuine trait differences or gender-specific response biases might have lowered women's scores, to the point of masking any association with gaze task performance. Hence, before concluding that an endophenotype candidate might be sex-specific (Matsuyoshi et al., 2014), gender and cultural influence should be ruled out, and trait measures should be proven invariant between genders and cultures (see f.i. Allison et al., 2010 for EQ).

5.5 LIMITATIONS

Our behavioural results mostly replicated previous reports, and therefore are unlikely to explain our results with personality or sex. For instance, we largely replicated GD results formerly reported in neurotypicals and individuals with autism (Wallace et al., 2006, 2010). We also replicated GC results in healthy controls (Jones et al., 2011). GD tasks showed more accurate processing of Whole-Face over Eyes, and Center over Averted Gaze (only Eyes-Only), and faster processing of Whole-Faces over Eyes-Only, and Social over Non Social stimuli (Wallace et al., 2006). However, we reported a *slower* processing of Center as compared to Averted Gazes, whereas Wallace et al. (2006) reported a *faster* one. GC task showed a significant GC effect, whose magnitude did not significantly differ with the one of Jones et al. (2011). Hence, limitations may rather pertain to psychometric tools and metholodological aspects of our tasks.

Our use of AQ may feature limitations, we would not claim for the sO-LIFE (Sierro, Rossier & Mohr, 2016 for a discussion; Fonseca-Pedrero et al., 2015). The AQ is popular, yet suffers from reliability issues (e.g. Ima; Hurst, Mitchell et al., 2007) and lacks a consensual factor structure (e.g. Kloosterman et al., 2011; Lau, Gau et al., 2013; Sierro, Rossier & Mohr, 2016). In our GD task, the Center Arrow may have biased participants' responses, and their associations with personality traits (e.g. Center vs Averted conditions), because it is a non ecological stimulus. Also, our RTs were globally shorter as compared to those reported by Wallace et al. (2006) who tested older participants who by default might have longer RTs. For the GC experiment, our GC effect was not significantly different from the one in Jones et al. (2011), but was smaller when compared to other studies (Lachat, Conty, Hugueville, & George, 2012 for a summary; Senju et al., 2004), and non-significant in men (see: Bayliss, Di Pellegrino et al., 2005). Using shorter Stimulus Onset Asynchrony (SOAs) might increase GC magnitude (Jones et al., 2010), and using a range of different SOAs (e.g. from 100 to 700 ms, Langdon et al., 2006) might improve the detection of associations with autistic *and* schizotypic traits, as they may occur at different SOAs (see also Langdon et al., 2016).

As a final comment, broader attentional deficits generalized to non-social stimuli might also partly explain gaze processing abnormalities (Bayliss, Di Pellegrino et al., 2005; Kuhn et al., 2010; see Landry & Parker, 2013 for a meta-analysis). In this respect, understanding social and non-social cueing performance and their neural correlates might gain from inclusion of non-social conditions, and from comparisons between different clinical groups (e.g. ASD vs attention deficit/hyperactive disorder [ADHD]: Tye et al., 2013).

5.6 CONCLUSION

We investigated gaze processing in healthy undergraduate participants, as a function of their "shared" and "diametrical" autistic and schizotypic traits. We tested gaze processing twofold (GD, GC). We expected "diametrical" traits, that is, the alleged hyper-Mentalistic positive schizotypic traits and hypo-Mentalistic autistic traits to relate, respectively, to gaze over-sensitivity, and gaze under-sensitivity (Crespi & Badcock, 2008). Our GD results supported the diametric model by Crespi and Badcock (2008), while we found no such support for the GC task. In the latter task, autistic mentalizing deficits (ComMind, Ima) in men associated with a reduced GC effect (see also Alwall et al., 2010; Bayliss, Di Pellegrino et al., 2005), with no result for positive schizotypy. On the downside, results in favour of opposite GD performance as a function of Mentalism showed limited generalizability on a statistical level. Results on an association between deficits in GC and autistic mentalizing deficits were statistically more convincing.

Beyond support for the Crespi and Badcock (2008) model, we highlight the link between autistic hypo-Mentalistic traits and reduced gaze sensitivity, in particular for GC, because this link points to the relevance of an "empathizing" or hypo-Mentalistic deficit dimension for ASp (Bishop et al., 2004; Sierro, Rossier & Mohr, 2016), a potential endophenotype candidate of ASD (Scheeren & Stauder, 2008). Future studies should test whether this proposition applies to men specifically (see also Matsuyoshi et al., 2014; Scheeren & Stauder, 2008). In this regard, ASp-PSp relationship models ought to be updated ideally integrating *altogether* "shared", "diametrical" and "distinct" features. Also, these models should build explanations "bottom-up" from previous research about ASp and PSp endophenotypes (Gur et al., 2015; Lainhart & Lange, 2009; Snitz et al., 2006; Sucksmith et al., 2011) to the higher levels of symptoms, personality or cognitive styles, such as Mentalism and Mechanism cognitive styles (Crespi & Badcock, 2008).

Acknowledgments

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6 GENERAL DISCUSSION

6.1 SHORT SUMMARY OF MAIN FINDINGS

The relationships between ASD and SSD are still unclear, some stressing "shared" features (King & Lord, 2011), others stressing "opposite" features (Crespi & Badcock, 2008). While "shared" features are not questioned, this thesis tested for behavioural correlates of "opposite" features to advance our understanding of ASp-PSp relationships (Crespi & Badcock, 2008). We tested healthy undergraduate students in Lausanne (Switzerland) validating the essential self-report questionnaires in French, and testing their relationship (Chapters 2 and 3), before assessing in addition social cognition (face and gaze processing) using computerized tasks (Chapters 4 and 5).

In Chapters 2 and 3, we showed acceptable reliability and validity for the French sO-LIFE (Sierro, Rossier, Mason et al., 2016). The French sO-LIFE showed a comparable structure to the English version with the best model being the 3-dimension model excluding ImpNon. For the AQ questionnaire, we replicated psychometrical weaknesses retaining a 5-dimension solution (AQ-K; in line with the solution presented by Klostermann et al., 2011), with item deletions and revised dimensions (in particular, Attention Switching deficits [AttSwi] became Routines/Repetitive Behaviors [RRBeh]; Communication deficits [Comm] became Communication/Mindreading deficits [ComMind]). For the relationship between the AQ-K and the sO-LIFE, we replicated "shared" traits (Dinsdale et al., 2013; Ford & Crewther, 2014; Hurst, Nelson-Gray et al., 2007; Russell-Smith et al., 2011), and "opposite" traits (Dinsdale et al., 2013; Del Giudice et al., 2014). We proposed that these "opposite" traits represent a Mentalism continuum, included in the diametrical model to distinguish ASp and PSp (Crespi & Badcock, 2008).

In Chapters 4 and 5, we tested whether psychometric observations of "opposite" traits would also be found in respective behaviour, namely social cognition (Crespi & Badcock, 2008). We tested liability to face pareidolia (Chapter 4) and gaze processing (Chapter 5). In both cases, we investigated whether hyper-Mentalism (positive schizotypy) would go with a tendency to see more meaning in social stimuli (faces, FLO, gaze direction) and hypo-Mentalism (autistic social trait deficits) with a tendency to see less meaning in social stimuli. We measured liability to face pareidolia assessing the IE for faces, FLO and OBJ categories. For gaze processing, we assessed GD and GC performance. For liability to face pareidolia (perceiving ambiguous stimuli (FLO) like a face), we found weak and inconsistent evidence in favour of the diametrical model (Crespi & Badcock, 2008). We concluded that our results are best explained by assuming "distinct" cognitive styles for schizotypy and autistic traits when performing this task, rather than "shared" or

"opposite" ones. For positive schizotypy, our results pointed to deficits in face configural processing and a local / featural processing strategy. For autistic mentalizing deficits (ComMind), our results pointed to preserved configural processing for face(-like) stimuli and a possible bias towards object recognition. For Ima, we observed a general configural processing deficit for standardized stimuli. Potentially, "distinct" cognitive styles when processing basic face information might have obscured pareidolia-proneness. In the case of gaze processing, we found evidence for "opposite" traits in the GD task, but for GC, we again found evidence for "distinct" cognitive styles (reduced gaze cueing could best be explained by higher hypo-Mentalistic traits; in particular in men). For both behavioural studies, we concluded that ASp-PSp relationship models should account for "shared", "diametrical" *and* "distinct" features. No existing model is doing so (Chisholm et al., 2015).

In the current Chapter 6, we outline implications of our results more widely, in particular their implications for the diametrical model. We propose alternative ways of conceiving and testing the diametrical model and other ASp-PSp relationship models. We discuss to what extent components of positive schizotypy (Chapter 6.2.) and autistic mentalizing deficit traits (Chapter 6.3.) truly represent hyper- and hypo-Mentalism, respectively (relatively to Chapters 2 and 3). We also examine whether these hyper- and hypo-Mentalistic traits are psychometrically (Chapter 6.4.1.) and behaviourally "opposite" (in face pareidolia and gaze sensitivity tasks; Chapter 6.4.2.). Susequently, we discuss the implications of our psychometric and behavioural findings for the diametrical model (Chapter 6.4.3.), notably discussing the concomittant existence of "shared" traits along "opposite" traits (including its limitations). Finally, we discuss limitations (Chapter 6.5.) and future challenges and perspectives (Chapter 6.6.). We highlight limitations of psychometric instruments and behavioural tasks (Chapters 6.6.1. and 6.6.2, respectively), and briefly discuss sex differences, notably those found in Chapter 5's gaze cueing task (Chapter 6.5.3.). Looking ahead, we (i) argue for and describe an alternative model capable of integrating the various relationships between ASp and PSp (Chapters 6.6.1. to 6.6.2.), (ii) discuss the issues and opportunities of using personality traits, cognitive styles or endophenotypes in ASp-PSp relationships research (Chapters 6.6.3, 6.6.4, and 6.6.5., respectively), and (iii) argue more broadly for a transdiagnostic approach when testing questions as those presented in this thesis (from Chapters 6.6.6. to 6.6.8.).

6.2 POSITIVE SCHIZOTYPY REPRESENTS HYPER-MENTALISM

Did we capture hyper-Mentalism on psychometric as well as behavioural levels?

6.2.1 PSYCHOMETRIC LEVEL

We assumed a priori that sO-LIFE's UnEx dimension represents positive schizotypy in the general population, and as such accounts for hyper-Mentalistic traits (Chapters 2 and 3). As we will show below, UnEx is conceptually close to hyper-Mentalism and at the same time opposite to allegedly hypo-Mentalistic traits. Behavioural measures were not mirroring these psychometric observations.

6.2.1.1 THE VALIDITY OF POSITIVE SCHIZOTYPY (UNEX)

We showed in the first experimental chapter (Chapter 2) that UnEx showed acceptable internal consistency or Cronbach's alpha. CFA supported the inclusion of UnEx as a schizotypic dimension (Cella et al., 2013; Lin et al., 2013). Comparison with other instruments (SPQ-B) further strengthened UnEx's validity. UnEx correlated positively with the SPQ-B analogue scale (Pos) (Chapter 2) as previously shown for SPQ's Cog-Per (Asai et al., 2011), and for corresponding WSS Magical Ideation dimension (Shofield & Mohr, 2014). This relationship suggests that sO-LIFE, SPQ(-B) and WSS dimensions measure similar constructs, although relying, respectively, on 4-, 3- and 2-dimension models (see also Gross et al., 2014). Elevated UnEx scores were reported in schizophrenia patients, as a function of positive symptoms (SAPS, Andreasen, 1984), supporting UnEx as a personality analogue to positive symptoms (Cochrane et al., 2010).

We consider that positive schizotypy, as measured by UnEx, represents hyper-Mentalism in conceptual, psychometric and empirical terms (Badcock, 2004; Crespi and Badcock, 2008). UnEx encompasses heterogeneous –although consistent– positive traits (for English O-LIFE: Claridge et al., 1997, Mason et al., 1995) accounting for magical belief, paranormal ideation, ideas of reference, and unusual sensory experiences (see also for SPQ, see Raine, 1991; WSS: Magical Thinking, Perceptual Aberrations, Winterstein, Silvia et al., 2011). Thus, not each UnEx item (e.g. perceptual aberrations) will tap hyper-Mentalistic features equally. Yet, most items make reference to external agents/intentionalities, such as supernatural entities (e.g. magical thought about gods, spirits, extra-terrestrials), belief into telepathy or overconfident mindreading (i.e. lack of boundary between self and others' minds), anthropomorphic interpretations of physical events (i.e. apophenia, attribution of meaning or intentionality to events), and human events (i.e. an exaggerated attribution of mental states to others; read English sO-LIFE items in Mason et al., 2005). These experiences can be

interpreted as involving over-functioning Mentalistic abilities (hence hyper-Mentalism; see also Abu-Akel, 1999; Abu-Akel & Bailey, 2000; Badcock, 2004; C. Frith, 2004).

Numerous empirical studies, indeed, indicate that positive symptoms in schizophrenia and positive schizotypic traits relate to hyper-mentalizing (e.g. Brosnan et al., 2010). Relationships were found when assessing empathy (Dinn, Harris, Aycicegi, Greene, & Andover, 2002; Brosnan et al. (2013), evoked apophenia (Fyfe et al., 2008), proneness to false alarms (Brugger & Graves, 1997), seeing faces in noise (Lindeman, Svedholm-Häkkinen, & Lipsanen, 2015), exaggerated perception of facial expressions (Uono, Sato and Toichi, 2015), ToM tasks (Clemmensen et al., 2014, 2015; Fretland et al., 2015), cooperation tasks (Backasch et al., 2013), as well as intention attribution tasks (Bara et al., 2009; Mohnke et al., 2015; Peyroux, Strickland, Tapiera, & Franck, 2014; Walter et al., 2009). Over-mentalizing in schizophrenia might, however, only apply to the cognitive and not the affective mental states (Lavoie, Jackson, Godmaire-Duhaime, Lacroix, & Achim, 2014). Noteworthy, hyper-mentalizing seems relevant to other conditions such as bipolar/manic patients (Usnich et al., 2015; but see Montag et al., 2011) or borderline PD (Andreou et al., 2015). Common underlying mechanisms may be a hyper-associative style, and/or an internal encoding style (Belayachi et al., 2013), conferring liability to apophenia and hyper-Mentalism's over-mentalizing tendencies (Brosnan et al., 2010; Fyfe et al., 2008).

6.2.2 BEHAVIOURAL LEVEL

6.2.2.1 PAREIDOLIA-PRONENESS AND FACE/FLO/OBJ PROCESSING

Contrary to our expectations, hyper-Mentalistic positive schizotypy did not associate with a larger liability for pareidolia-proneness (Chapter 4). We observed, instead, relationships with impaired configural processing or a featural bias for stimuli with face configurations. When positive schizotypy associated with the pareidolia index (i.e. once decreased and once increased pareidolia effect), alternative explanations fared better than those implying a genuine pareidolia effect. For instance, the smaller pareidolia index associated with UnEx (Chapter 4's Study 2) likely occurred because of decreased IE:Accuracy:FACE and not because of increased IE:Accuracy:FLO. Hence, we concluded that, instead of pareidolia-proneness, we observed more fundamental performance patterns associated with positive schizotypy. This conclusion was not restricted to one experiment. Previous SSD studies showed impaired face configural processing (Butler et al., 2008; Chambon et al., 2006; Joshua & Rossell, 2009; Shin et al., 2008) and deficits in encoding face configurations in schizotypy and schizophrenia (Batty et al., 2014; Chambon et al., 2006, respectively). Thus, in Study 2 (Chapter 4), our individuals with elevated positive schizotypy might have relied on featural

processing due to their configural processing deficits, as observed for SSD patients (Joshua & Rossell, 2009; Shin et al., 2008). In Study 3 (Chapter 4), however, we observed such configural deficits with larger positive schizotypy for front images of cars (FLO), resulting in face-like processing behaviour (Windhager et al., 2008, 2010). Potentially, pareidolia effects might have manifested indirectly in the configural deficit of high positive schizotypy scorers for processing standardized face-like images of car fronts. More likely, the association of positive traits with evoked apophenia and seeing faces in noise reported in the litterature (Chapter 6.2.1.1.) might have been blurred by stronger effects of basic face processing (Chapter 4). In sum, we doubt that the current method is ideally suited to test the diametrical model, instead showing that "distinct" features might also be relevant to SSp-ASp relationship models (see also Discussions in Chapters 4 and 5).

6.2.2.2 GAZE DIRECTION PROCESSING AND GAZE CUEING

More promising regarding our expectations was that hyper-Mentalistic (positive schizotypic) traits partly associated with GD accuracy, but not with GC liability (Chapter 5). In GD, PCDF-K and UnEx scores tended to associate with a larger Accuracy difference between Center and Averted conditions, in contrast to ComMind. Larger UnEx scores associated with larger Accuracy for Center Whole-Face stimuli, thus, showing an increased sensitivity to Centre gaze (see also Hooker & Park, 2005 in patients with schizophrenia), in contrast to what was reported for ASp (Wallace et al., 2006, 2010). Yet, for GC task, we did not observe increased GC liability with larger positive schizotypy, adding more evidence to already existant conflicting results (Akyiama et al., 2008; Dalmaso et al., 2013; Langdon et al., 2006, 2016; Magnée et al., 2011; Pino et al., 2015). In contrast to social perception tasks, higher-level social cognition tasks, such as intention attribution tasks, showed clearer associations between positive traits/symptoms to hyper-mentalizing (Chapter 6.2.2.1.).

6.2.3 SUMMARY

Positive schizotypy (as measured by UnEx) represents hyper-Mentalism psychometrically (Chapter 6.2.1.). Few evidence supported corresponding hyper-Mentalism behaviourally using social cognition tasks (Chapter 6.2.2.). The association of larger UnEx scores with larger accuracy for centre directed gaze confirmed the hypothesis of larger gaze sensitivity as a function of larger hyper-Mentalistic scores, in line with other researches (Chapter 5). Yet, GC liability did not

significantly vary as a function of larger hyper-Mentalistic scores, in line with inconsistent research findings in the literature (Chapters 5 and 6.2.2.2.). As for face/FLO/object recognition, hyper-Mentalistic traits rather reflected the face configural deficit in processing stimuli with a face-like configuration, in line with schizophrenia and schizotypy research (Chapters 4 and 6.2.2.1.). Possibly, the presence of similar configural processing deficit for the face-like car fronts may indicate an indirect pareidolia effect. Nevertheless, with the exception of gaze perception, our results indicate that psychometric hyper-Mentalism does not simply translate into larger pareidolia-proneness and larger gaze sensitivity (Chapters 4 and 5), as posited by the diametrical model (Crespi & Badcock, 2008). As for face(-like) processing, hyper-Mentalism as measured by positive schizotypy shows fundamental processing biases, possibly masking a genuine pareidolia effect. As we shall see below, we come to similar conclusions for hypo-Mentalistic autistic traits.

6.3 AUTISTIC MENTALIZING DEFICITS REPRESENT HYPO-MENTALISM

Did we capture hypo-Mentalism on psychometric and behavioural levels?

6.3.1 PSYCHOMETRICALLY

We assumed a priori that AQ's ComMind and Ima dimensions represent social and communication deficits in the general population, and as such accounts for hypo-Mentalistic traits (Chapter 3). As we will show below, Ima and CommMind are conceptually close to hypo-Mentalism, opposite to allegedly hyper-Mentalistic traits. Again, we do not find that behavioural measures mirror the psychometric ones.

6.3.1.1 VALIDATION OF AQ AND ITS USEFULNESS FOR UNDERSTANDING ASP-PSP RELATIONSHIPS

We showed in the second experimental chapter (Chapter 3) that AQ-K's ComMind and Ima dimensions may help us accounting for hypo-Mentalistic traits, and by inference mentalizing deficits. In this chapter, we also argued that, psychometrically, different factor solutions were proposed for AQ, and none was universally accepted (e.g. Baron-Cohen et al., 2001; Hurst, Mitchell, et al., 2007; Lau, Kelly et al., 2013). Moreover, for validation purposes, we lack direct empirical comparison with similar measures (e.g. SRS and BAPQ; Ingersoll et al., 2011).

Nevertheless, across studies, a coherent picture of AQ emerges, notably the presence of dimensions accounting for autistic empathising or mentalizing deficit traits (e.g. Bishop et al., 2004; Hurst, Nelson-Gray, et al., 2007; Lau, Kelly et al., 2013). This picture is congruent with the factor structure we retained for our sample, the one of AQ-K (Kloosterman et al., 2011). Furthermore, two AQ-K dimensions might help distinguishing between autistic and schizotypic traits, i.e. ComMind and Ima deficits. Below, we explain in which ways these AQ dimensions might help us in ASp-PSp.

6.3.1.2 COMMIND

A first AQ-K dimension, ComMind, helped us distinguishing schizotypic and autistic traits by accounting reliably and validly for hypo-Mentalism. Thus, ComMind has conceptual, and empirical relevance, despite its low reliability (Cronbach's alpha; Chapter 3, Kloosterman et al., 2011). To us, ComMind corresponds to "empathising" deficits seemingly specific to ASp (Bishop et al., 2004) grouping items from AQ-BC's Comm dimension, SocSki, and Ima dimensions. Most psychometric AQ questionnaire studies report on mentalizing deficits: "Understanding others" (Stewart & Austin, 2009), "Communication/Mindreading" (Austin, 2005; Hurst, Nelson-Gray et al., 2007; 1st study of Russell-Smith et al., 2011), "Understanding Others/Communication" (2nd study of Russell-Smith et al., 2011), "Mindreading" (Lau, Gau et al., 2013), or "Social Cognition" (Lau, Kelly et al., 2013). Only Hoekstra et al. (2011) did not retain an analogue dimension to Comm or ComMind. When using alternative instruments, researchers too report on dimensions resembling ComMind such as "Reading Facial Expression", "Expressive Language" (Kanne et al., 2012), "Conversational Skills" and "Expressiveness" (Dawson et al., 2007), "Pragmatic Language" (Hurley et al., 2007), or "Social Communication" and "Understanding Emotions" (Garnett et al., 2013). In a large clinical study on more than 20'000 ASD children (Steer, Golding, & Bolton, 2010), "Social Understanding" was confirmed as one of six autistic factors. In line with its validity, ComMind recalls *DSM-5* ASD criteria of social deficits in nonverbal communication and understanding relationships (APA, 2013, p. 50).

Crucially, ComMind and analogue dimensions measure everyday-life communication and mindreading deficits that are different from negative traits. Mindreading refers to the ability to draw inferences about other persons' thoughts, impaired in autism (Baron-Cohen, 2002), and is part of mentalizing abilities performed by the social brain network (Kennedy & Adolphs, 2012; see Chapter 1.5.4. for further details). In Chapter 3, we reported on a small correlation between enhanced ComMind scores and lower Extraversion, but no significant

correlation with Neuroticism. These observations support the theoretically sound assumption that ComMind deficits can neither be reduced to introverted nor anxious/depressive traits. The original AQ-BC's Comm dimension showed, indeed, small to moderate correlations with both Neuroticism, and Extraversion (Wakabayashi et al., 2006a) in addition to Agreableness (Austin, 2005). Given this reasoning and our results, we conclude that AQ-K's ComMind's mindreading deficits represent hypo-Mentalistic traits when testing the diametric model (Crespi & Badcock, 2008).

6.3.1.3 IMA

A second AQ-K dimension, Ima, may help us distinguishing between schizotypic and autistic traits. Ima forms part of hypo-Mentalism, notwithstanding criticism towards its reliability and validity (Chapter 3). Ima measures imagination deficits, and has been part of the original AQ (Baron-Cohen et al., 2001). Ima bears conceptual and clinical relevance for ASD and autistic traits (*DSM-IV*, APA, 1994; Ten Eycke & Müller, 2014). In *DSM-5*, lack of imagination is not a criteria of ASD itself, but is stated as an example of social deficits in ASD criteria (APA, 2013, p. 50). Ima was, however, criticized for its lack of internal reliability (e.g. Hurst, Mitchell et al., 2007). Moreover, in psychometric studies, Ima or an analogue dimension was not retained in about half of the studies (Austin, 2005; Hurst, Nelson-Gray et al., 2007; Lau, Gau et al., 2013; Lau, Kelly et al., 2013; 1st study of Russell-Smith et al., 2011). In the other half of the studies, Ima was retained, but its item number was frequently reduced or revised (Hoekstra et al., 2011; Kloosterman et al., 2011; 2nd study of Russell-Smith et al., 2011; Stewart & Austin, 2009). To our knowledge, alternative autism instruments do not feature Ima or any analogue dimension (Dawson et al., 2007; Garnett et al., 2013; Hurley et al., 2007; Kanne et al., 2012; Steer et al., 2010).

Based on our findings, we suggest that Ima may represent an aspect of hypo-Mentalistic traits. Ima conceptually reflects an autistic symptom, not reducible to any other broad personality dimension. Ima conceptually targets an autistic feature defined in *DSM-IV* (APA, 1994) and evoked in *DSM-5* (APA, 2013), i.e. a lack of imagination and fantasy life found in ASD children. Yet, Ima does not simply reflect imagination deficits but deficits in *social Imagination*, the ability to simulate the content of other's mind and behaviour (involved in pretend play, typically lacking in ASD children; see APA, 2013; see also Ten Eycke & Müller, 2014). The ability to simulate others' mind could be a complementary function to usual mindreading; another expression of mentalizing (Craig & Baron-Cohen, 1999; Kennedy & Adolphs, 2012). In our data, Ima showed a small negative correlation with Extraversion and no significant correlation with Neuroticism. Previously, AQ-BC's Ima showed small significant negative correlations with each dimension of the NEO-PI-R, but

Neuroticism (Wakabayashi et al., 2006a). Although these relationships are not very informative about Ima's nature, they nonetheless exclude the possibility that Ima can be mainly explained by broad personality dimensions. We suggest for these reasons that the lack of social Imagination reflects one aspect of hypo-Mentalistic tendencies characterizing ASD (Badcock, 2004; Crespi & Badcock, 2008).

Interpretation of ComMind and Ima as mentalizing deficits, underlying autistic traits and relevant to ASD, seems to be trivial and very much conceptual. We consider psychometric, behavioural and alternative lines of evidence to further evaluate whether we can interpret certain autistic traits as mentalizing deficits (Chapter 3). We will focus on *indirect* psychometric evidence.

6.3.1.4 INDIRECT PSYCHOMETRIC EVIDENCE

Indirect evidence suggests that autistic mentalizing deficits traits exist among AQ dimensions and allow distinguishing ASD/BAP from other groups (Bishop et al., 2004), notably SSD (Wouters & Spek, 2011). To our knowledge, direct evidence[22] is missing to support ComMind and Ima account for mentalizing deficits. In our case, indirect evidence refers to the abilities of dimensions underlying an "empathising" or mentalizing dimension (Bishop et al., 2004) to distinguish ASD/BAP from other neurotypical and clinical groups, notably SSD. When looking at such studies (summarized in Appendices Table 28), we can conclude that a mentalizing or empathising dimension (i) likely characterises ASD/BAP and (ii) allows distinction from other clinical or healthy groups (including SSD) based on score comparisons. Yet, not all AQ dimensions are equally relevant to identify ASD individuals among other groups (Appendices Table 28): SocSki, Comm (or analogue dimensions), and to a lesser extent Ima scores are systematically higher in (i) ASD groups than neurotypicals, (ii) BAP than non-BAP groups (i.e. parents with and without ASD children), and (iii) ASD groups than SSD groups. This overview additionally indicated that Comm and Ima distinguished ASD from specific anxiety disorders (SAD), obsessive-compulsive disorder (OCD) and attention deficit/hyperactivity disorder (ADHD), whereas SocSki could not. Likewise, AttDet rarely distinguished between the tested groups.

Comm and Ima scores, but not SocSki scores, distinguished between ASD and both OCD and SAD supporting the role of mentalizing deficits rather than social anhedonic deficits

[22] By direct evidence, we mean for instance correlations between ComMind, Ima or analogue autistic dimensions. We also think of correlations with specific mentalizing dimensions such as EQ or tasks requiring mentalizing abilities. As of now, AQ-K and other alternative factor structures are not sufficient evidence showing that autistic traits are mentalizing deficit traits (ComMind, Ima).

(Appendices Table 28). In contrast to ComMind and Ima, SocSki *partly* accounts for a "lack of social interest" that may be shared between ASD, OCD and SAD, as a nonspecific negative symptom (Kaiser et al., 2011; Malaspina et al., 2014). We suggest that the best dimensions to distinguish between ASD/BAP population and any other clinical or nonclinical population are, in order of importance, Comm, SocSki, Ima, and eventually AttSwi, but not AttDet. In AQ-K, SocSki was revised and solely consists of items measuring lack of *social interest*, without mentalizing deficit items. Hence, AQ-K's SocSki may not distinguish between ASD and other groups as AQ-BC's SocSki did. AQ-K's ComMind bundles mindreading deficit items and is the closest dimension from AQ-BC's Comm. Thus, ComMind may account for mentalizing deficits and ASD liability (a.o. BAP). AQ-K Ima is the reduced analogue to AQ-BC's Ima, including only social imagination deficits. Consequently, AQ-K Ima might relate to mindreading abilities (Craig & Baron-Cohen, 1999), and by inference to mentalizing deficits distinguishing between ASD and SSD liability. For these additional reasons, we propose that ComMind and Ima represent autistic mentalizing deficits, likely indexing ASD liability, possibly distinguishing ASD from SSD, and their respective personality traits.

6.3.2 BEHAVIOURAL LEVEL

6.3.2.1 PAREIDOLIA-PRONENESS AND FACE/FLO/OBJ PROCESSING

We identified relationships between hypo-Mentalistic autistic mentalizing deficits (ComMind) and pareidolia index, but withhold from interpreting it as pareidolia-proneness (Chapter 4). Like for UnEx, we did not find a significant increase of IE for FLO associated with autistic traits. This suggests that variations of pareidolia index as a function of autistic traits (e.g. ComMind, Ima) reflected specific face/object processing rather than genuine pareidolia-proneness.

Hypo-Mentalistic ComMind did not associate with configural processing deficits, and showed an advantage for processing OBJs (Chapter 4). Larger ComMind scores, contrary to positive schizotypy, did not show a configural impairment. This absence of configural impairment fits recent claims of preserved configural processing in ASD (Weigelt et al., 2012; but see Behrmann et al., 2006), and neurotypicals with heightened autistic traits (Hedley et al., 2014). Indeed, ASD would be characterized by deficits in face memory, unrelated to SSD configural encoding impairments (see Chapters 4 and 6.6.2.1.), but related to a larger sensitivity to encoding-retrieval durations (Weigelt et al., 2012). Our encoding-retrieval duration was probably too short to observe associations between autistic traits and face recognition performance. Moreover, higher

ComMind scores associated with UPR and INV OBJ stimuli in one experiment, suggesting they associated with improved processing of stimuli that do not feature face-like configuration, like face and FLO (cars) did. These results might be explained by various peculiarites reported in ASD: a preference for non-Face stimuli (Pallett et al., 2013; Wolf et al., 2008), a preference for featural processing (Lahaie et al., 2006; Nishimura, Rutherford, & Maurer, 2008), a lack of social motivation and reward value for social stimuli (Dawson et al., 2005; Klin et al., 2002), passive omission of gaze(-like) stimuli due to abnormal salience (Senju & Johnson, 2009), an active avoidance of hyper-arousing gaze(-like) stimuli (Dalton et al., 2005; Tanaka & Sung, 2013; Tottenham, et al., 2014), or the defect of a hypothetical brain module subserving social stimuli processing (Baron-Cohen, 1995, but see Itier & Batty, 2009).

Ima was not associated with a genuine pareidolia effect. Higher Ima associated with lower FLO performance in Study 1 (remember that higher UnEx scores associated with higher FLO performance). Ima may associate with decreased (Crespi et al., 2016) and UnEx with increased (Merckelbach et al., 2000) fantasy proneness. Possibly, a larger fantasy proneness (UnEx) associated with better FLO encoding, and a lower fantasy proneness (Ima) associated with worse FLO encoding. Also, the higher Ima scores in Study 2 tended to decrease accuracy for UPR Faces, like UnEx did. It reflects on face recognition deficits with larger total autistic traits scores reported in healthy controls (Halliday et al., 2014), possibly associated with the majority of women in Study 2. We also speculated that other deficits might have impaired performance associated with Ima, as previously observed in ASp (i.e. deficit in adaptive coding: Rhodes et al., 2013). To our knowledge, there are no published cognitive results particularly linked to Ima scores.

6.3.2.2 GAZE DIRECTION PROCESSING AND GAZE CUEING

In Chapter 5, we reported that hypo-Mentalistic autistic mentalizing deficit traits (ComMind, Ima) associated with autistic gaze under-sensitivity (see also Bayliss, Di Pellegrino et al., 2005; Matsuyoshi et al., 2014; Wallace et al., 2006, 2010). In GD, larger Ima scores associated with increased Accuracy differences between the Non-social and Social conditions. This difference was largely owed to the association between higher Ima scores and lower Accuracy for Center Whole-Face condition. Also, higher Ima and ComMind scores tended to associate with longer RTs for Center Whole-Face and Eyes-only conditions. Hence, autistic traits associated with worse performance in centre-directed gaze conditions, mostly Center Whole-Face, as reported in ASD and their relatives (Wallace et al., 2006, 2010). We treated these results with caution, because we had observed statistical limitations for this particular regression analysis.

In GC (Chapter 5), larger autistic mentalizing deficits (ComMind) associated with a decreased automatic redirection of attention by gaze (see also Bayliss, Di Pellegrino et al., 2005). Larger ComMind significantly associated with a smaller GC effect. The higher ComMind scores, the less was participants' attention redirected by Congruent or Incongruent gaze. Further sex-specific regressions showed that these relationships were only found for men. Moreover, larger Ima scores in men, but not in women, associated with a smaller GC effect, as ComMind scores.

In both GD and GC experiments (Chapter 5), autistic mentalizing deficit traits (ComMind, Ima) associated with a relative under-sensitivity to gaze, as reported for ASp (Baron-Cohen et al., 1995; Nummenmaa, Engell, von dem Hagen, Henseon, & Calder, 2012; Ristic et al., 2005; Wallace et al., 2006), specifically in men (see also Scheeren & Stauder, 2008; Wallace et al., 2010). Gaze processing abnormalities might reflect an ASD endophenotype (Matsuyoshi et al., 2014), in particular for front gaze processing (Wallace et al., 2010). For GC, several previous studies showed smaller GC effects as a function of autistic traits in healthy individuals (Bayliss, Di Pellegrino et al., 2005; Bayliss & Tipper, 2005), as has been shown in adults with ASD (Ristic et al., 2005). We can hypothesize that the association between total autistic traits and smaller GC effect owed largely to empathising or mentalizing deficits traits, such as ComMind and Ima. In line with this interpretation and our results, Alwall et al. (2010) reported an association between larger EQ scores and larger GC effect. This latter observation is not surprising, because EQ conceptually represents the inverse construct (i.e. high empathy) to ComMind and Ima (i.e. low empathy / empathy deficits).

Beyond treating gaze under-sensitivity as a putative ASD endophenotype, our data support centre gaze discrimination as a behavioural correlate of hypo-Mentalistic traits. Early deficits in gaze processing were found in healthy siblings of ASD children (Dalton et al., 2007; Elsabbagh et al., 2009), in ASD adults (Wallace et al., 2010), and unaffected parents of ASD children, in particular fathers (Scheeren & Stauder, 2008; Tajmirriyahi et al., 2016). Both observations satisfy the condition for centre gaze processing to represent an ASD endophenotype candidate (see also: Matsuyoshi et al., 2014). The endophenotype idea is supported by similar deficits in the healthy population (Wallace et al., 2010), and those with high autistic traits (Bayliss, Di Pellegrino et al., 2005; Matsuyoshi et al., 2014), notably as a function of mentalizing deficits traits (Chapter 5; Alwall et al., 2010). Possibly, early automatic gaze processing deficits (listed in Chapter 6.3.2.1.) contribute to the the development of joint attention, and mentalizing skills. Later in life, these deficits may manifest through self-reported hypo-Mentalistic traits, deviant gaze processing performance (Chapter 5; Wallace et al., 2010), and social deficits (Jellema et al., 2009; Kuhn et al., 2010). The severity of early gaze processing deficits, the environmental support, and the existence and efficiency of compensatory strategies would contribute in modulating the severity of resulting

mentalizing and social skills deficits, all along the autistic spectrum. As a result, hypo-Mentalistic traits, as measured by ComMind and Ima account for autistic gaze under-sensitivity, and represent the expected hypo-Mentalistic behavioural correlates of the diametrical model (Crespi & Badcock, 2008).

6.3.3 SUMMARY

Autistic mentalizing deficits (as measured by ComMind and Ima) seem to represent hypo-Mentalism, but psychometric evidence is yet scarce. Ima, in particular, shows reliability issues and uncertain validity. We presented some indirect evidence that these deficits correspond to hypo-Mentalism as assessed with social cognition tasks (Chapter 5). Higher ComMind and Ima scores associated with smaller gaze sensitivity, either for GD processing and GC liability (Chapter 5), in line with ASD and autistic traits research. Also, ComMind associated with enhanced performance for non-social OBJ recognition (Chapter 4). More than UnEx hyper-Mentalism, ComMind and Ima obviously relate to a lack of advantage for abnormal gaze processing, possibly related to gaze aversion or lack of reward value (Chapter 5). Hence, hypo-Mentalistic autistic traits are diametrical/opposite to hyper-Mentalistic traits. As we shall see, there will also be distinct ones.

6.4 OPPOSITE PSYCHOMETRIC AND BEHAVIOURAL RELATIONSHIPS: IMPLICATIONS FOR THE DIAMETRICAL MODEL

Did we capture hyper- vs. hypo-Mentalism opposition at psychometric and behavioural levels?

6.4.1 PSYCHOMETRICAL LEVEL

6.4.1.1 OPPOSITION BETWEEN HYPO- AND HYPER-MENTALISM

In line with previous research, our psychometric results (Chapter 3) support the idea that autistic hypo-Mentalism *opposes* positive schizotypy hyper-Mentalism (Badcock 2004; Crespi and Badcock, 2008). We observed small, but significant negative correlations between UnEx on the one hand, and ComMind and Ima on the other hand (see Chapter 3). Our PCA results confirmed that autistic and schizotypic traits relationships could be summarized by a two-factor solution. One factor represented “shared” negative/social anhedonic traits (e.g. IntAn, SocSki) and another factor diametrically opposite traits (PCDF-K). In the PCDF-K, positive schizotypy (UnEx) and autistic

mentalizing deficits (ComMind, Ima) loaded in opposite ways. Other studies had already found such negative correlations between autistic mentalizing deficits and positive schizotypic traits. Hurst, Nelson-Gray et al. (2007) first found negative relationships between SPQ's Cog-Per positive schizotypy and AQ's Ima and Comm, using zero-order and semipartial correlations. The second study by Russell-Smith et al. (2011) reported negative correlations between UnEx and Ima. Dinsdale et al. (2013) found negative relationships between positive schizotypy (UnEx) and some autistic traits (SocSki, Ima). Most studies did not discuss these negative correlations, because of their weakness (Hurst, Nelson-Gray et al., 2007; Russell-Smith et al., 2011). Dinsdale and colleagues interpreted this negative correlation, without explicitely considering a Mentalism continuum (Crespi & Badcock, 2008).

Other psychometric evidence supports the opposition between ASp and PSp looking at Mentalism or Empathizing dimension. Brosnan et al. (2010) found that larger EQ scores associated with larger positive schizotypy and psychotic traits scores (e.g. mania). Close to the Mentalism-Mechanism model (Badcock, 2004), this study proposed that positive schizotypy may associate with a psychotic-prone extreme female brain, analogue to the autistic extreme male brain from Baron-Cohen (2002) E-S theory.

Direct and indirect evidence support the relevance of mentalizing deficits (e.g. ComMind) as being opposite to positive schizotypy (UnEx). Ciaramidaro et al. (2014) showed opposite cerebral connectivity patterns when ASD and schizophrenia patients performed an intention attribution task. The ASD group showed decreased connectivity between right STS and right ventromedial PFC, while the schizophrenia group showed increased connectivity between these *same* areas. Abu-Akel, Apperly, Wood and Hansen (2015) recently reported on a diametrical modulation of the anterior and the posterior right TPJ in a mentalizing task. These authors had looked at autistic and psychotic traits in healthy participants. These opposite brain activation patterns in the *same* areas suggest a diametrical bias affecting the *same* mechanisms, and the *same* underlying aetiological pathway. Hence, we interpret them as strong support for the diametrical model.

Additional clinical evidence supports the relevance of a dimension like Ima, as opposite to UnEx. While a lack of imagination was reported in children with autism, the opposite was reported in children who would later develop schizotypal PD (Tantam, 1988; Wolff, 1991). Children who would later develop schizotypal PD showed increased fantasies in childhood, suggesting that imagination-proneness might be opposite between both groups. A recent study showed that children with schizotypal PD scored significantly higher in “fantasy/magical thinking” than ASD and typically developing children, while ASD and typically developing children did not differ from each other (Jones et al., 2015). The “fantasy/magical thinking” scale contained items relative to *social*

imagination (e.g. imaginary friends, make-believe plays), that corresponds to the inverse of Ima deficits dimension. This scale also loaded congruently with other positive traits (e.g. paranoia, apophenia, delusion, perceptual aberrations), similar to those measured by UnEx. Hence, fantasy-proneness, and social imagination in particular, might be relevant to distinguish between ASp and PSp, and may explain the relationship between UnEx and Ima traits in our results (Chapter 3).

In sum, literature and our results emphasize that autistic mentalizing deficit traits are opposite to positive schizotypic ones. These deficits would correspond to a Mentalism continuum (Crespi & Badcock, 2008). As a result, Mentalism is a promising dimension to distinguish between ASD and SSD, as proposed by Crespi and Badcock (2008) and others (Brosnan et al., 2010). In our behavioural studies, we considered UnEx to represent hyper-Mentalism, and ComMind and Ima hypo-Mentalism. Based on our PCA results, we also computed general indices accounting for hyper- and hypo-Mentalism (PCDF-K), notably based on UnEx, ComMind, Ima, but also other scores (Chapters 4 and 5). In the discussion of behavioural results below, we emphasized the effects of constitutive dimensions of these scores (UnEx, ComMind, Ima), as they could better account for the effect of personality on behaviour than PC scores (PCSF-K; PCDF-K).

6.4.2 BEHAVIOURALLY

6.4.2.1 PAREIDOLIA-PRONENESS AND FACE/FLO/OBJ PROCESSING

Hyper- and hypo-Mentalistic traits associated with face/FLO/object processing rather than pareidolia-proneness. These traits did not reveal *diametrically opposite* performance patterns as a function of *diametrical* hypo- and hyper-Mentalistic traits, but *distinct* ones (Chapter 4). With respect to face and OBJ processing, the diametrical model (Crespi & Badcock, 2008) would predict that hyper-Mentalistic traits of positive schizotypy would associate with improved face and FLO processing, whereas hypo-Mentalistic autistic mentalizing deficit traits would associate with improved OBJ and FLO processing relatively to face processing. Hyper- and hypo-Mentalistic profiles would have shown, respectively, a social/human stimulus processing bias and a non-social/non-human stimulus processing bias. Ambiguous stimuli, on the other hand, would be processed as a function of a pareidolia-proneness. We did not observe these tendencies.

We observed distinct performance patterns, instead of the opposite ones predicted by the diametrical model (Crespi & Badcock, 2008). Positive schizotypy associated with a difficulty processing face-like configuration stimuli (FACE, FLO:Cars' fronts), or a local bias for face stimuli. Autistic mentalizing deficits showed an advantage for processing OBJ stimuli (chairs),

irrespective of their orientation. No link was found with FLOs. These findings suggest distinct (rather than opposite) deficits. Positive schizotypic traits associated with configural processing deficits and/or a featural bias. These deficits are possibly due to an insufficient encoding, as seen in SSD patients and schizotypy (see Chapter 6.2.2.1.). Autistic mentalizing deficit traits tended to associate with a preserved configural processing as seen in ASD, patients and ASp. This preserved processing is possibly due to preference for object and/or avoidance of stimuli with gaze(-like) features (see Chapter 6.3.2.1.)

Pareidolia-proneness and basic visual processing could not permit us to confirm the diametrical model with a statistical opposition of similar mechanisms (e.g. pareidolia-proneness, face processing). Our results nonetheless associated face-like processing with hyper-Mentalism and OBJ processing with hypo-Mentalism (Chapter 4). Obviously, the diametrical model (Badcock, 2004; Crespi & Badcock, 2008) could account for these specific association patterns, or stimuli preferences, better than other models proposing shared deficits, and by inference shared mechanisms. Nevertheless, our results on relationships between pareidolia-proneness and personality traits cannot offer support to the diametrical model (Chapter 4). These results go along with a certain number of problems, and might reflect basic visual processing rather than an influence of Mentalism. Future research ought to be performed to clarify this issue.

6.4.2.2 GAZE DIRECTION AND GAZE CUEING

Gaze experiments provided a partial and spurious support to the diametrical model (Crespi & Badcock, 2008). Only central GD processing showed diametrically opposite performance as a function of hypo- and hyper-Mentalistic dimensions, while GC liability did not (Chapter 5). To sum up, GD perception confirmed opposite autistic hypo-sensitivity and schizotypic hyper-sensitivity to gaze, in line with ASD and SSD literature (Chapters 6.3.2.2, and 6.2.2.2. respectively). While promising, the statistical tests showed a ceiling effect violating assumptions of regression analyses. Assumptions, and consequently how much weight these results should carry. At first sight, GC's sensitivity appeared diametrically opposite (general PC score), confirming the diametrical model (Chapter 5). At second sight, however, autistic hypo-Mentalism associated with smaller GC's gaze sensitivity, a putative endophenotype of ASD (Chapter 6.3.2.2.). Positive schizotypy hyper-Mentalism showed no such association with GC's gaze sensitivity, in line with inconsistent results in SSD litterature (Chapter 6.2.2.2.). Our results are reminiscent of those by Sasson et al. (2007). These authors suggested that lower social orienting in ASD as compared to SSD or controls, represents a distinct abnormality in ASD.

Mirroring the conclusions for face/FLO/OBJ processing (Chapters 4 and 6.4.2.1.), the hyper-/hypo-Mentalism opposition of the diametrical model may not apply to all gaze-related tasks (Chapter 5). The relevance of hyper/hypo-Mentalistic opposition may depend on the type and level of cognitive processing. Possibly, simple detection of gaze cues relates to diametrically opposite biases for ASp and PSp, while gaze-induced attentional redirection is specific to ASp. Yet, gaze processing abnormalities might have distinct origins. ASp gaze processing abnormalities might stem from basic perceptual and attentional processes (e.g. passive avoidance, as claimed by Senju & Johnson, 2009; see also Chapter 6.3.2.1.). PSp gaze processing may stem from higher-level cognitive biases (e.g. gaze attribution, in Hooker & Park, 2005; see also Chapter 6.2.2.1.). In contrast to our social perception tasks (Chapters 4 and 5), only higher-level social cognition tasks (intention attribution) showed an opposition between ASp and PSp (Chapter 6.4.1.1.). *To sum up, ASp-PSp diametrical opposition in Mentalism can be found in high-level social cognition tasks (Chapter 6.4.1.1.) and self-reported personality measures (Chapter 3). Lower-level social perception tasks revealed mostly distinct relationships between ASp and PSp (Chapters 4 and 5).* We will discuss the idea of different kinds of ASp-PSp relationships as a function of cognitive level (psychometry, cognitive styles, endophenotypes; Chapters 6.6.3. to 6.6.5.).

6.4.3 IMPLICATIONS FOR CRESPI AND BADCOCK (2008) DIAMETRICAL MODEL

What are the implications of our results for the diametrical model and theories of ASp-PSp relationships?

Our results have few if any implication on the different ASp-PSp theories (see Chisholm et al., 2015 for a review). At the very least, our results underline the fact that no existing theoretical model can account for all results (ours, in the literature, see Chisholm et al., 2015). As highlighted at various places, we strongly propose an adaptation to any model in that "distinct" features for ASp and PSp should be considered, in addition to "shared" and "diametrical" ones.

Models that advocate partially overlapping/shared mechanisms and aetiologies can only partly explain our results (e.g. King & Lord, 2011; Rausch and Johnson, 2008). Rausch and Johnson's (2008) model could explain our psychometric overlaps, namely between negative/social anhedonia traits (e.g. SocSki, IntAn) and positive-like symptoms (e.g. UnEx, AttDet, RRBeh), reported in Chapter 3. As for behaviour results, the resembling detrimental effects of UnEx and Ima on the configural face processing could also be explained by similar aetiologies (Chapter 4). Yet, other results we obtained and the literature do not support the idea that common underlying

mechanisms relative to configural processing deficit are shared between positive schizotypy and autistic mentalizing deficits (Chapter 4).

Hence, it is possible that distinct mechanisms or impairments cause similar deficits. For instance, both ASD and SSD were reported to exhibit deficits in face processing and face memory, but stemming from different causes (Butler et al., 2008; Chambon et al., 2006; Weigelt et al., 2012). Without proper tasks that enable a distinction between underlying mechanisms, the configural face processing deficit of positive schizotypy, and the gaze avoidance or impaired salience of gaze may both lower the performance in face processing and face memory, and result in apparent similarity (Chapter 4). The problem with seemingly similar deficits of autistic and schizotypic traits is that it is not possible to know whether this similarity rests on similar or distinct underlying mechanisms or impairments. As a result, focusing on underlying mechanisms is crucial. Also, focusing on distinct performances may prove useful, since they necessarily refer to a difference somewhere in underlying mechanisms and impairments.

In parallel to finding evidence for overlap, we found *opposite* traits as a function of Mentalistic traits, partially supporting the diametrical model (Chapter 3) and behaviourally (Chapter 5). Psychometrically, while finding an important overlap between autistic and schizotypic traits, not all overlapped and some opposed (Chapter 3), as previously reported (Dinsdale et al., 2013; see also: Del Giudice et al., 2014). We observed diametrical (opposite) relationships between hyper-Mentalistic positive schizotypic traits (UnEx), and hypo-Mentalistic autistic mentalizing deficits (ComMind, Ima). Models emphasizing (partially) overlapping aetiologies (King & Lord, 2011; Rausch & Johnson, 2008) cannot account for these opposite features. In contrast, the diametrical model and the extended E-S theory (Baron-Cohen, 2002; Brosnan et al., 2010) are the only models able to account for opposite psychometric features between autistic and schizotypic traits.

Behaviourally, we also found *opposite* performance patterns as a function of Mentalistic traits, partially supporting the diametrical model (Chapter 5). Indeed, the larger the hyper-Mentalism, the better the accuracy for detecting centre gaze in whole-faces, and the larger the hypo-Mentalistic Ima traits, the smaller the accuracy in this condition. We also found an opposition in gaze cueing as a function of Mentalism PCSF-K score. Again, among the existing models, only the diametrical model (Crespi & Badcock, 2008) or the extended E-S model (Brosnan et al., 2010) could account for these opposite performance patterns. Models advocating shared deficits and overlapping aetiologies cannot account for these performances (King & Lord, 2011; Rausch & Johnson, 2008[23]). As a result, the diametrical model is the best suited to explain these results.

[23] Rausch and Johnson's (2008) model cannot account for these diametrical performances because (i) they did not clarify the status of mentalizing deficits as either shared, opposite or distinct between ASp and PSp,

Nevertheless, our results do not prove that these opposite performances necessarily stem from opposite Mentalistic biases in the same mechanisms: opposite performances could stem from distinct mechanisms acting at different levels (Chapter 6.4.2.2.).

In contrast, neither face/FLO/object, nor GC liability showed diametrical performances as a function of Mentalistic traits (UnEx, ComMind, Ima), yet distinct performances (Chapters 4 and 5). In both face/FLO/object task and GC liability (Chapters 4 and 5), the underlying mechanisms and impairments involved are likely *distinct*, rather than *opposite*. For face/FLO/object, the associations of positive schizotypy mostly pertained to face and FLO, whereas those of autistic mentalizing deficits (i.e. ComMind), mostly pertained to objects (Chapter 4). Also, for GC liability, only autistic mentalizing deficit traits (in men) associated with a smaller gaze sensitivity (Chapter 5). A PSp-specific face(-like) configuration processing deficit, possibly stemming from encoding deficits might have affected face recognition performance (Chapter 6.2.2.1.). In contrast, an ASp-specific preserved configural processing and aversion for face(-like) stimuli or preference for non-face ones might have improved object recognition (Chapter 6.3.2.1.), and decreased gaze sensitivity (Chapter 6.3.2.2.). Against the diametrical model (Crespi & Badcock, 2008), positive schizotypy and autistic mentalizing deficits mostly affected distinct behavioural experimental conditions instead of showing opposite effects on these same conditions.

Current models emphasizing shared and opposite features do not account for distinct deficits (Crespi & Badcock, 2008; King & Lord, 2011). We argue that new alternative and integrative models are necessary. Indeed, distinct deficits cannot be accounted for by a simple hypo- vs. hyper-Mentalistic dichotomy (Crespi & Badcock, 2008). Also, distinct deficits cannot be accounted for by explanations proposing shared deficits stemming from similar mechanisms and aetiologies in ASp and PSp (King & Lord, 2011; Rausch & Johnson, 2008). Models theorizing *partially* distinct aetiologies would be suitable. Nevertheless, no model currently accounts for the variety of relationships between ASp and PSp (Chisholm et al., 2015). We believe that ASp-PSp relationships would need to be represented by an alternative *multidimensional* model, integrating (i) *partially* shared, (ii) *partially* diametrically opposite and (iii) *partially* distinct deficits and their respective aetiological pathways.

In addition, such a model would have to integrate different ASp-PSp relationships across the same causal pathway. Within a given causal pathway (e.g. mentalizing deficits), personality self-

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and (ii) their model does not address the causes of distinct performances in mentalizing tasks between ASp and PSp.

assessment and high-level social cognition tasks might exhibit shared and opposite features between ASp and PSp (Chapter 3 ; Chapter 6.4.1.1.), whereas lower-level social perception tasks would feature distinct features (Chapters 6.4.2.1., 6.4.2.2.). *Against what was suggested by Crespi & Badcock (2008), the diametrical model's ASp-PSp opposition may neither be sufficient nor correct for all considered variables at all explanatory levels (see Chapters 6.6.3. to 6.6.5.).*

6.4.4 SUMMARY

Our data offered clear psychometric evidence but absent or spurious behavioural evidence in favour of the diametrical model. Psychometrically, we replicated previous evidence of shared and opposite personality traits between autistic and schizotypic sets. The opposite personality traits could be interpreted as positive schizotypy and the autistic mentalizing deficits, matching the diametrical model's hyper- and hypo-Mentalism (Chapter 3). Behaviourally, centre gaze processing was improved as a function of positive schizotypy (UnEx), but impaired as a function of autistic mentalizing deficits (Ima) (Chapter 5). Yet, the psychometric and behavioural opposition were respectively weak and flawed, unable to offer valid support for the diametrical model. Also, alternative models to the diametrical model might account for psychometric overlaps and distinct behavioural performance patterns (Chapters 4 and 5). Still, the diametrical model is the only model that could explain the opposition between positive schizotypy gaze over-sensitivity and autistic gaze under-sensitivity (Chapter 5). In addition, ASp-PSp relationships might be discontinuous across explanatory levels of an aetiological pathway (e.g. cognitive levels of mentalizing; Chapter 6.4.3). Among all the technical limitations questioning our conclusions, we note that no current model can account for ASp-PSp relationships we observed, nor any of those reported in the literature (Chisholm et al., 2015 for a review). Before outlining and discussing an alternative ASp-PSp relationships model, we detail the empirical and theoretical limitations of our research below as well as future perspectives.

6.5 LIMITATIONS, GENERAL COMMENTS AND POSSIBLE SOLUTIONS

Why had we no better psychometric and behavioural results, and what are the possible strategies to obtain them?

6.5.1 PSYCHOMETRICALLY

6.5.1.1 GENERAL LIMITATIONS REGARDING MENTALISM

We argue that our findings are limited by our measurement of Mentalism. The opposite correlations between positive schizotypy and autistic mentalizing deficit dimensions were small, and may not be a convincing argument for Mentalism, understood as the diametrical opposition between hyper- and hypo-Mentalism (Chapter 3). Some authors did not consider such correlations (Hurst, Nelson-Gray et al., 2007; Russell-Smith et al., 2011), while other authors did (Chapter 3; Dinsdale et al., 2013). In any case, the presence of negative correlations between autistic and schizotypic traits is a robust observation (Chapter 3; Dinsdale et al., 2013; Hurst, Nelson-Gray et al., 2007; Russell-Smith et al., 2011). Either a robust artefact is driving the negative correlations, or a robust relationship. We do not think the former possibility is likely. Also, we can explain the reason why the opposition between positive schizotypy and autistic mentalizing deficits was small, by analyzing positive schizotypy and autistic mentalizing deficits scales.

6.5.1.2 LIMITATIONS RELATIVE TO POSITIVE SCHIZOTYPY AND HYPER-MENTALISM

We present one possible reason why we have observed small negative correlations between hyper- and hypo-Mentalistic traits, i.e. positive and impulsive schizotypy scales were not designed to account for hyper-Mentalism (Chapter 3). In general, positive schizotypy scales were not designed to account for hyper-Mentalism, neither UnEx (Mason et al., 1995, 2005) nor SPQ's Cog-Per (Raine, 1991). Both UnEx and Cog-Per measure several constructs classified under the dimension of positive traits, some of which may not be associated with hyper-Mentalism (e.g. perceptual aberrations), but possibly with underlying apophenia liability (Belayachi et al., 2014; Fyfe et al., 2008; see also Chapter 4 and Chapter 6.2.1.1).

Similarly, ImpNon traits were not designed to account for hyper-Mentalism, although it is likely they did (Chapter 3). Results obtained with ImpNon also suggest that (hypo-)manic features

may account for hyper-Mentalism and its associated over-mentalizing (Chapter 3). Indeed, we observed that ImpNon scores correlated negatively with ComMind scores, and were also featured in PCDF-K, along UnEx scores. We did not retain ImpNon for several psychometric and theoretical reasons. Pickering (2004) and Cochrane et al. (2010) consider this dimension to not measure schizotypic *per se*, but a historical remenant of Psychoticism. Indeed, sO-LIFE's ImpNon correlated with Psychoticism (Chapter 3), because it shares items with Psychoticism (Claridge et al., 1996). To our knowledge, there is no equivalent of ImpNon in either SPQ (Raine, 1991), WSS (Winterstein, Silvia et al., 2011), in clinical instruments such as SANS/SAPS (Andreasen et al. 1983; 1984), or in any other PSp assessment tool.

The theoretical relevance of ImpNon (or similar dimensions) relatively to SSD is unclear. The ImpNon dimension that is part of the Hypomania Personality Scale (Eckblad & Chapman, 1986) did not predict transition to psychosis (Chapman, Chapman, Kwapil, Eckblad, & Zinser, 1994), but did so for bipolar mood disorder (Kwapil et al., 2000; Walsh, DeGeorge, Barrantes-Vidal, & Kwapil, 2015). Mason and Claridge (2006) justified the inclusion of ImpNon to SSD. They argued that ImpNon accounts for additional general features accounting for a "unitary psychosis theory" in which schizotypy would account for psychosis-proneness and not only schizophrenia-proneness (i.e. psychosis-proneness: schizophrenia *and* bipolar disorders; see also: Crow, 1990; Shevlin et al., 2016). In case that ImpNon's reckless/antisocial behaviour shares the same liability than hypo-mania (Claridge & Blakey, 2009), (hypo-)manic traits accounted for by ImpNon may account for hyper-Empathizing along with positive schizotypy. Brosnan et al. (2010) observed that higher Empathizing associated with higher manic and paranoid traits of the Psychosis Screening Questionnaire (PSQ; Bebbington & Nayani, 1995).

Although ImpNon may be relevant for PSp and related to Empathizing, the main problem with this dimension is psychometric (Chapter 3). Recent CFA analyses offered mixed support for ImpNon's inclusion to schizotypy (Cella et al., 2013; Fonseca-Pedrero et al., 2015; Lin et al., 2013). Notably, a recent intercultural study on sO-LIFE further questioned the reliability and factorial validity of ImpNon (Fonseca-Pedrero et al., 2015). A revised ImpNon scale or an analogue one could be useful for schizotypy, as well as for the study of ASp-PSp relationships, as both might oppose with respect to impulsivity (Del Giudice et al., 2014). For ImpNon to be useful, future studies should clarify its nature as a schizotypy construct, probably revise and improve its scale, and examine whether it accounts for hyper-Mentalism.

6.5.1.3 LIMITATIONS RELATIVE TO AUTISTIC MENTALIZING DEFICITS AND HYPO-MENTALISM

Autistic mentalizing deficits dimensions (ComMind, Ima) were not designed to account for hypo-Mentalism, possibly explaining their small correlations with positive schizotypic traits (Chapter 3). Contrary to UnEx, the very definition of ComMind and Ima, and their item content are quite homogenous and resemble what could be expected from scales measuring hypo-Mentalism, or deficit in Empathizing (Baron-Cohen, 2002). This is, however, more convincing for ComMind than for Ima (Chapter 3). Moreover, several different AQ validation studies supported an "empathising" deficit construct underlying the original AQ social deficits traits (SocSki, Comm, Ima; Bishop et al., 2004; see Chapter 6.3.1.), such as "Mindreading" deficits (Lau, Gau et al., 2013) or "Social Cognition" deficits (Lau, Kelly et al., 2013). Yet, as we previously pointed out (Chapter 6.3.1.), we are not aware that a relationship between Empathizing construct and ComMind and Ima[24] has been reported in the published literature. Only indirect evidence is available.

Another problem with ComMind and Ima is their low number of items, the weak reliability of ComMind and the unacceptable reliability of Ima (see Chapter 3). These problems are not specific to our results, but reflect a more general criticism towards the AQ (Hurst, Mitchell et al., 2007; Kloosterman et al., 2011). Future studies should improve the AQ, notably by additing more items and validation studies. These improvements would in turn improve the measurement of, respectively, mindreading deficits and social imagination deficits, and likely hypo-Mentalism.

Improving the Ima dimension, however, implies additional challenges in terms of psychometric and clinical validity (Chapter 3, Chapter 6.3.1.3). First, not all studies found support for the existence of Ima or analogue dimensions (e.g. Lau, Gau et al., 2013; Lau, Kelly et al., 2013). Studies discarded Ima items or transferred items to other dimensions analogue to ComMind (i.e. respectively, "Mindreading" and "Social Cognition" deficits dimensions), casting doubt that Ima is a valid factor (Hurst, Mitchell et al., 2007; Lau, Gau et al., 2013; Lau, Kelly, et al., 2013). Second, imagination deficit traits may be relevant to a subset of individuals with autistic traits. Social imagination deficits are present in autism, but not in indiviuals with an Asperger's syndrome (*DSM-IV*, APA, 1994; Ten Eycke & Müller, 2014). U. Frith (2004) and Attwood (2007) pointed out that individuals with Asperger's syndrome tend to develop fantasy worlds, have imaginary friends, and

[24] Unpublished data show significant negative correlations between EQ's "Cognitive Empathy" dimension (alternative factor structure: Lawrence, Shaw, Baker, Baron-Cohen, & David, 2004) and ComMind (medium size effect) and Ima (small size effect), respectively. Since EQ is probably the best proxy for Empathizing and Mentalism, we can assume that conceptually ComMind and Ima represent inverse concepts of Empathizing and Mentalizing.

may actually develop a rich fantasy life. To our knowledge, we have no research evidence that quantifies the existence of social imagination deficits along the ASD, in particular in adulthood. Accordingly, we cannot be sure that imagination deficits should be included as a specific dimension to autistic traits. Social imagination deficits may not be relevant when assessing hypo-Mentalism, even after psychometric improvements of such a scale.

In sum, we would like to stress that the use of original AQ-BC dimensions is problematic (Chapter 3), and alternative AQ factor structures should be preferred. "Eempathising"/mentalizing items (ComMind) were scattered across several dimensions (SocSki, Comm, Ima). This scattering may have delayed the identification of a mentalizing deficit dimensions in AQ validation studies, as some do not retain such dimensions at all (e.g. Austin, 2005; Hoekstra et al., 2008, 2011). Also, this scattering might have prevented *more robust* and *larger* negative correlations between positive schizotypy and autistic mentalizing deficit dimensions (Hurst, Nelson-Gray et al., 2007; Russell-Smith et al., 2011). Finally, this scattering might have prevented understanding opposite traits as hyper- and hypo-Mentalistic traits (Del Giudice et al., 2014; Dinsdale et al., 2013) or finding opposite traits in the first place (Ford & Crewther, 2014). A reliable and valid AQ factor structure containing autistic mentalizing deficits scores is a prerequisite to properly account for hypo-Mentalism, thereby contributing to clarifying ASp-PSp relationships.

6.5.1.4 GENERAL STRATEGY FOR IMPROVING THE MEASUREMENT OF HYPO- AND HYPER-MENTALISTIC TRAITS

A new questionnaire specifically measuring hyper- and hypo-Mentalism may not be necessary. It could be sufficient to combine existing questionnaires that have been improved incluing the possibility to add theoretically-driven new items. One could improve Hyper-Mentalism measurements by adding items from instruments' dimensions such as O-LIFE's UnEx dimension (Mason et al., 1995), SPQ's Cognitive-Perceptual dimension (SPQ: Raine, 1991), Chapman scales'/WSS "Magical Ideation" scale (Eckblad & Chapman, 1983), PSQ (Bebbington & Nayani, 1995; used in Brosnan et al., 2010), or Community Assessment of Psychic Experiences (CAPE42: Konings, Bak, Hanssen, van Os, & Krabbendam, 2006; used in Abu-Akel, Wood, Hansen, & Apperly, 2015). Since paranormal beliefs correlate positively with positive schizotypy traits (Thalbourne & French, 1995; Bouvet, Djeriouat, Goutaudier, Py, & Chabrol, 2014), certain items from paranormal belief scales might complement positive schizotypy scales. It would be possible to capture extreme forms of mentalizing, provided these scales do not confound with cultural variables (e.g. "Psi" dimension from the Revised Paranormal Belief Scale; Tobacyk, 2004; telepathy-related

items from the Sheep Goat Scale; Thalbourne & French, 1995). Moreover, the PSQ (Bebbington & Nayani, 1995) dimensions or items that associated with Empathizing (Brosnan et al., 2010) may improve measurement of (hypo)manic constructs related to ImpNon.

As for measurement of empathy, adding items accounting for enhanced or reduced empathy might be relevant. EQ items might be useful, as they could be treated like reverse items for hypo-Mentalistic autistic traits (Baron-Cohen & Wheelwright, 2004; Wakabayashi et al., 2006b). Here again, one would have to take the multidimensionality of EQ into account (Lawrence et al., 2004). In the current context, Empathizing items of Cognitive Empathy should be favoured. As for use of AQ dimensions, improved autistic mentalizing dimensions would imply improved measurement of hypo-Mentalism. Possibly, other autistic trait questionnaires may contain interesting items with regard to autistic mentalizing deficits (e.g. BAPQ, Hurley et al., 2007; SRS, Constantino, Davis et al., 2003). Moreover, generalizing the use of CFAs in validation studies, and if possible, transitioning to methods inspired from item response theory may help in improving the psychometric measurement of hypo- and hyper-Mentalistic traits (Chapter 3).

6.5.2 BEHAVIOURALLY

6.5.2.1 PAREIDOLIA-PRONENESS AND FACE/FLO/OBJ PROCESSING

Our results of Chapter 4 feature several types of limitations, some pertaining to questionnaires, others to our experimental protocols. First, we already pointed to the psychometric limitations of the sO-LIFE and AQ (Chapter 6.5.1.). These limitations may have impeded the detection of diametrical relationships in pareidolia-proneness and face/FLO/object processing (Chapter 4). Moreover, the use of PC scores offered weak or questionable evidence for relationships of the questionnaire scores with behavioural variables. A general problem with PC scores was that they confounded various questionnaire dimensions, potentially masking some dimensions' specific relationship with behaviour (e.g. PCDF-K: AttDet, RRBeh, UnEx, and ImpNon). As a result, we conducted regression models using the sO-LIFE and AQ-K subscale scores. Future study should not solely rely on such PC scores.

Second, we had aimed to account for pareidolia-proneness as a behavioural expression of hyper-Mentalism, but failed to do so (Chapter 4). We postulated that a smaller difference between IE:FACE and IE:FLO would index a tendency to process ambiguous FLO stimuli rather as faces than objects. This smaller difference should represent a behavioural index of hyper-Mentalism. Yet, the IE was unstable for non-Face categories. The homogeneization of stimuli did not improve its

stability (see also: Pallett & MacLeod, 2011). Because we did not find a stable IE, we cannot expect to find stable associations between personality traits and a face-like processing bias for FLOs. We propose several causes and solutions to respectively explain and solve this problem: (i) our assumption that pareidolia proneness can be accounted for by the IE magnitude may be false, (ii) our stimuli may not have been adequate to elicit a pareidolia effect (e.g. not standardized enough; but see Hadjikhani et al., 2009), (iii) pareidolia proneness may not be captured behaviourally, and require alternate techniques (e.g. EEG: Caharel et al., 2013; eyetracking; Windhager et al., 2010), and (iv) pareidolia-proneness may not be measured independently from basic face/FLO/object processing effects (Chapter 4).

Third, alternative protocols or adjustments could be more efficient to investigate ASp-PSp relationships (Crespi & Badcock, 2008). For instance, studies could manipulate the encoding duration and encoding-retrieval delay to assess target detection as a function of, respectively, SSD/schizotypy (Butler et al., 2008; Chambon et al., 2006) and ASD/autistic (Weigelt et al., 2012) traits. Also, instead of old-new recognition judgements (memory component), protocols could measure stimuli discrimination (e.g. Wallace et al., 2010) when assessing encoding mechanisms (e.g. a matching task). Finally, eyetracking and EEG and other imaging techniques could visualize face/FLO/object processing, in particular when searching for weak mechanisms in healthy individuals (Caharel et al., 2013; Falck-Ytter, 2008).

Fourth, some of our regression models might have been underpowered, and/or our predictions were not accurate enough. Indeed, the sizes of our significant effects were small, making them difficult to detect. Power analyses (Chapter 4) and general multiple regression models *F*-tests (Appendix Table 29) and specific *t*-tests on slopes (Appendix Table 30) indicated that our models lacked power. If we assume that all significant effects are true, we should have tested a minimum of n=85 participant per study (considering *F*-tests on general models) or n=80 participant per study (considering *t*-tests on regression slopes) to reach a minimally acceptable power (.80). Other studies featured more participants (e.g. 150-240 participants; Rhodes et al., 2013; see also Halliday et al., 2014) with some up to two times larger samples when performing sex-specific analyses (e.g. 30 women in Rhodes et al., 2013).

Fifth, future studies could use Signal Detection Theory to allow a better measurement of the performance of participants. Distinguishing between false-alarm-prone vs. miss-prone strategies might correspond to opposite cognitive styles (e.g. internal vs. external encoding styles : Belayachi et al., 2007; Lewicki, 2005 ; intuitive vs. deliberative cognitive styles : Brosnan et al., 2013, 2014). Creating an adaptive memory task, suitable for both patients and nonaffected controls, would be

ideal to account for such biases across the whole range of ASp and PSp, and their probable different performance levels (e.g. Cappe et al., 2012).

6.5.2.2 GAZE DIRECTION AND GAZE CUEING

Among the limitations of our study (Chapter 5), some pertain to instruments we used, others to the protocol we replicated. We evoked several limitations for each of these categories in addition to some potential solutions and perspectives. We highlighted that psychometric limitations regarding sO-LIFE and AQ (Chapter 6.5.1.) may have rendered it impossible to detect diametrical relationships with GD and GC behavioural variables (Chapter 5). Moreover, using PC scores was not very informative and we would not advice using these scores (Chapter 6.5.2.1.). Finally, we question results of the GD experiment and suggest to use adaptive protocols to better account for difficulty level of the experiment.

We found a ceiling effect in the GD experiment that likely flawed our regressions models. We did not expect this ceiling effect (see Wallace et al., 2006, 2010). To deal with differences in performances between studies, researchers could use adaptive procedures (e.g. Cappe et al., 2012). Adjustments would be performed by varying the interval between the stimulus (e.g. gaze) and the mask. The resulting dependent variable would be the amount of time between the stimulus onset and the mask for the participant to accurately process GD in 75% of trials. When using social visual stimuli (e.g. schematic stimuli mimicking gaze as sclera/pupil contrast and shape), researchers could test the diametrical model. We would expect worse performance with higher schizotypy (as seen with CogDis in a non-social task; Cappe et al., 2012), and better performance with higher autistic traits (see Mottron et al., 2006).

Fourth, we did not replicate performance in the GD task for additional reasons (Chapter 5). We did not use a group design (see Wallace et al, 2006, 2010), but computed performance indices. These indices confounded main behavioural variables and may have masked specific effects. For instance, the centre Arrow condition was not ecological and might have biased our results (see also Chapter 5). We computed indices summarizing results for different conditions. It is possible that the RT difference between “Centre Arrow” and “Averted Arrow” conditions accounted for most of the “Centre vs. Averted” RT index, driving its relationship with personality traits. The “Centre Arrow” condition stimuli could be modified to more ecologically valid representatons by presenting upward or downward arrows, so that differences with “Averted Arrow” would be minimized.

In GD (Chapter 5), we presented stimuli at the shortest display duration (40 ms; Wallace et al., 2006, 2010) to shorten the duration of the experiment and avoid a geometric strategy (Wallace et al., 2006). These short display durations may not be long enough for activating the mentalizing network (e.g. STS, TPJ), as performed by higher-level mentalizing tasks (Abu-Akel, Apperly et al., 2015). Hence, the effects we found may owe little to mentalizing deficits, but to low level perceptual abnormalities when processing social cues (e.g. Tanaka & Sung, 2013). Future studies could use a range of display durations (40, 70, 100 ms; Wallace et al., 2006, 2010). Possibly, other deficits may be detected. The drawback would be a three times longer protocol.

Fifth, gaze self-attribution protocols might be more promising to assess GD processing when testing ASp-PSp relationships (e.g. Hooker & Park, 2005; Matsuyoshi et al., 2014). Instead of investigating purely GD perception (Wallace et al., 2010), several studies assessed gaze self-directedness judgements integrating intention attribution, a higher level of social cognition (Hooker & Park, 2005). Face stimuli were displayed with gaze directed at various angles. Participants had to judge whether each gaze stimulus was directed at them or not. In line with the diametrical model (Crespi & Badcock, 2008), ASp individuals under-attributed gaze as self-directed (Matsuyoshi et al., 2014), whereas individuals with schizophrenia over-attributed it (Hooker & Park, 2005; Tso et al., 2012). Gaze self-attribution biases ought to be tested in ASp and PSp populations jointly, their relatives, but also in healthy individuals differing in their positive schizotypy and autistic mentalizing deficit traits. In this respect, coupling such experiments of gaze self-attribution with the gaze perception might be valuable to disentangle purely perceptual from attributional biases. Thereby, it would be possible to disentangle the contributions of lower-level perceptual gaze processing, and higher-level social cognition/attributional gaze processing, as a function of ASp and PSp.

Sixth, our GC task has several limitations, e.g. small GC magnitude, the absence of variable SOAs, and the absence of non-social cueing as a control condition (Chapter 5). In our GC task, we used one short stimulus display duration instead of a range of different durations. This might have lowered our GC magnitude. Although our GC magnitude was similar to Jones et al. (2011), it was smaller than those in other studies (Lachat et al., 2012, for a summary; Senju et al., 2004), and non-significant in men, as previously reported (e.g. average 2 ms GC effect in men in Bayliss, Di Pellegrino et al., 2005). Driver et al. (1999) stressed that GC magnitude depends on the SOA (i.e. the delay between the cue, i.e. gaze, and the target, i.e. star/circle). Using a range of SOAs may increase the chance of getting a larger overall GC magnitude, notably in smaller SOAs. Possibly, GC magnitude in men could be raised overall or at least in some SOAs. Possibly, a larger GC effect and a range of different SOAs may increase the chances to detect associations with personality,

notably at shorter SOAs (Langdon et al., 2006, 2016). Again, the drawback would be a longer protocol.

Alternatively, non-social stimuli should be used along gaze stimuli, as a control condition of GC (Chapter 5). Deficits in social (gaze) and non-social cueing would not have the same implications as sole deficits in GC. The former would point to broad attentional causes (Landry & Parker, 2013), whereas the latter would point to causes related to social cue processing, such as gaze aversion (Tanaka & Sung, 2013). Bayliss, Di Pellegrino et al. (2005) showed that cueing deficits in individuals as a function of autistic traits also extended to non-social stimuli. These direction and cueing deficits may not be limited to social stimuli but relate to more fundamental attentional deficits (Landry & Parker, 2013). More research is required to ascertain these results with autistic and schizotypic traits. Systematically comparing social and non-social conditions might be useful, although this approach would double the duration of the experiment. Comparing ASD and other populations, such as ADHD, might be instructive to tease apart purely attentional and social deficits (Tye et al., 2013).

Other improvements to the GC protocol (Chapter 5) could consist in making the stimuli either more ecological, or more schematic. Including movements (several images or video clips) or more ecological conditions would likely improve the cueing effect, and possibly provide a better activation of STS (see Lachat et al., 2012). On the contrary, it would be interesting to investigate whether schematic gaze stimuli elicit a cueing effect or not, also for GD experiments. Schematic gaze is less likely to be correctly ascribed as salient (Senju & Johnson, 2009), or elicit gaze avoidance (Tanaka & Sung, 2013). In particular with respect to gaze avoidance, one could suppose that the contrast between dark pupil/iris and white sclera is a potent signal, activating amygdala very quickly (Dalton et al., 2005), then STS. Possibly, the sensitivity to gaze owes much to this feature of eyes (Kylliäinen et al., 2012). In line with the hyper-arousal model (Dalton et al., 2005; Tanaka & Sung, 2013; but see Chapter 6.3.2.1. for a discussion of explanations), Kylliäinen et al. (2012) reported that children with ASD but not neurotypicals showed autonomic arousal proportional to the amount of visible sclera of stimuli. Possibly, manipulating ecological and schematic stimuli might reveal distinct or opposite gaze processing between ASp and PSp, thereby improving the understanding of their relationships.

In summary, comparing the performances for arrows, schematic gaze and real gaze, we would make it possible to distinguish whether ASp and PSp groups feature an attentional orientation deficit (i) pertaining to all kinds of directional stimuli (arrows, pointing gestures, real gaze, schematic gaze), (ii) specific to eye-like stimuli (real gaze, schematic gaze), or (iii) specific to real eye gaze. Respectively, these deficits would point to (i) broad attentional deficits (Landry &

Parker, 2013), (ii) amygdala over-reactivity to gaze defined as low-level visual properties of sclera/pupil configurations (Dalton et al., 2005; Kylliäinen et al., 2012), and (iii) gaze-specific processing deficits, possibly related to a lack of reward value of gaze or an aversion (Dawson et al., 2005; Tanaka & Sung, 2013). We would predict for ASp that direction processing and attention orienting would be impaired for non-social stimuli, but in particular for schematic and real gaze. In contrast, in PSp, we suggest that direction processing and attention orienting might be intact or enhanced, possibly slower, for schematic and real gaze.

Finally, some of our regression models might have been underpowered, and/or our predictions were not accurate enough. The sizes of our significant effects were mostly small, in particular when interactions between personality variables and sex were added (Chapter 5). Additional power analyses for behavioural experiments (Chapter 5) considering general multiple regression models *F*-tests (Appendix Table 29) and specific *t*-tests on slopes (Appendix Table 30) showed a lack of power. If we assume that all significant *F*-tests effects are true, GD study should have tested a minimum of 68 participant (considering *F*-tests on general models) or 81 participants (considering *t*-tests on slopes) to reach a minimally acceptable power (.80). If we assume that all significant *F*-tests effects are true, GC study should have tested a minimum of 119 participants (considering *F*-tests on general models, including Sex × personality interaction effects) or 77 participants (considering *t*-tests on slopes) to reach a minimally acceptable power (.80). Although some regression models had adequate power (e.g., GC models and ComMind), larger samples would be necessary to increase the power of these regression models, partly because of the necessity of sex-specific predictions (Bayliss, Di Pellegrino et al., 2005; Scheeren & Stauder, 2008). Hence, collapsing both sexes should be avoided as it may blur sex-specific effects, and reduce the overall effect. Instead, sex-specific predictions should be made, and tested with sample sizes permitting adequate power.

6.5.3 SEX DIFFERENCES IN GAZE CUEING LIABILITY

In line with the literature, our data suggest that GC deficits might be specific to men, yet it remains unclear whether this effect is sex or gender-specific (Chapter 5). Indeed, hypo-Mentalistic trait associations with gaze processing were prominent in men (see also Alwall et al., 2010; Bayliss, Di Pellegrino et al., 2005; Bayliss & Tipper, 2005; Deaner et al., 2007; Matsuyoshi et al., 2014; Scheeren & Stauder, 2008). Irrespective of autistic traits and associated biases, Scheeren and Stauder (2008) showed that GC was abnormal and smaller in unaffected fathers of children with ASD, as compared to fathers of neurotypicals. At the psychometric level, autistic traits show higher

scores in men than women (Baron-Cohen et al., 2001), which may reflect a female advantage in affective empathy, and possibly cognitive empathy or mentalizing (Christov-Moore et al., 2014; Wakabayashi et al., 2006b).

Although several theoretical models predicted such sex differences, it does not follow that gender effects are to be ruled out. Baron-Cohen (2002) proposed the autistic brain would show exaggerated male features. Conversely, Brosnan et al. (2010) proposed that psychotic brains would be characterized by exaggerated female features. Differently, Crespi and Badcock's (2008) model involves paternal and maternal genetic imprinting in offspring. Autism would be caused by an over-expression of paternal genes (i.e. silencing of maternal ones), whereas psychosis would be caused by an over-expression of maternal genes (i.e. silencing of paternal ones). The "extreme male brain theory" was criticized suggesting that sex differences might stem from stereotypes (Halpern, 2000). Gender differences should be considered before concluding on sex differences notably expressed in Empathizing and Systemizing (Baron-Cohen, 2002). Although the diametrical model is not relying on the same assumptions and was not subjected to the same criticism, it is nonetheless possible that gender differences may account for differences in psychometric Mentalism and Mechanism.

Instead of sex-differences, gender differences in questionnaire responding may explain the men-specific personality-behaviour associations. Higher autistic scores in men and higher Empathizing scores in women (Baron-Cohen et al., 2001; Wakabayashi et al., 2006b) may rather reflect gender-specific roles than genuine trait differences reflecting biological roles (e.g. "women should be empathic"). Influence of gender roles may particularly apply to self-report questionnaires (Eisenberg & Lennon, 1983). Technically, the influence of demographic (e.g. culture, gender) factors on questionnaire responding may be accounted for by differential item functioning. Differential item functioning implies that some items are endorsed as a function of different cultures, genders or other social groups. Differential item functioning was evoked to explain sex differences in AQ (Broadbent et al., 2013), in EQ (Allison et al., 2011), and in negative schizotypy (Winterstein, Ackerman et al., 2011). In our case, genuine trait (biological) differences, gender-specific (social) response biases, or both might have resulted in lower scores in women, to the point of masking any association with gaze task performance.

Influence of socio-demographic variables on the relationships between face recognition and personality should be ruled out before concluding on sex-specific face recognition endophenotypes (but see Matsuyoshi et al., 2014). As we could see in the paragraphs above (Chapter 6.5.3.), some evidence and theories support sex-specific mentalizing impairments in relationship to autistic traits. Further research is necessary to prove that these impairments reflect sex differences, implying to first rule out possible gender differences. Globally, future studies might use multi-group CFAs to

test whether sO-LIFE and AQ traits behave in a similar way (i.e. same factor structure, comparable scores) across different cultural backgrounds (Fonseca-Pedrero et al., 2015), between the sexes, and between healthy and clinical populations (Murray et al., 2014). If possible, scales should be revised so to include only items that do not elicit cultural- or gender-specific responses.

6.5.4 LIMITATIONS OF THE DIAMETRICAL MODEL

Throughout this work, we could notice limitations pertaining to the diametrical model (Chapters 3, 4 and 5). Notably, the diametrical model does not address overlaps in negative and positive traits, their possible influence on cognition, the uncertainties and the dearth of studies regarding mechanisms, and the emphasis on hyper-Mentalism as a PSp feature. These and other limitations of the diametrical model encouraged us to imagine an alternative model of ASp-PSp relationships. In what follows, we summarize the limitations, and propose alternatives.

6.5.4.1 PSYCHOMETRICALLY

6.5.4.1.1 OVERLAP AND OPPOSITION BETWEEN AUTISTIC AND SCHIZOTYPIC TRAITS

The diametrical model does not theorize or conceptualize much overlaps in sets of symptoms and traits between ASp and PSp (Crespi & Badcock, 2008). We replicated both overlap and opposition of particular autistic and schizotypic traits (Dinsdale et al., 2013; Hurst, Nelson-Gray et al., 2007; Russell-Smith et al., 2011). Obviously, the "diametrical" but not "shared" traits are the most relevant when it comes to distinguish between ASp and PSp, for instance with behavioural measures. Yet, this does not mean that "shared"/overlapping traits are unimportant. On the contrary, they should be accounted for by ASp-PSp relationship models. We briefly evoke overlapping negative features, and overlapping positive ones[25].

6.5.4.1.1.1 OVERLAP IN NEGATIVE TRAITS

Overlap in negative autistic and schizotypic traits has already been reported, and suggests at least partly shared underlying aetiological factors. In Chapter 3, we replicated overlap between

[25] Note we use the terms "negative" and "positive" in their broad meaning, respectively characterizing *deficits* and *additional eccentric* features, as compared to healthy population (Malaspina et al., 2014)

negative schizotypic traits (IntAn), and autistic lack of social interest and motivation (SocSki), as previously reported (Claridge & McDonald, 2009; Dinsdale et al., 2013; Ford & Crewther, 2014; Hurst, Nelson-Gray, et al., 2007; Russell-Smith et al., 2011). Despite their label, AQ-K's SocSki items refer to lack of pleasure, interest or motivation in social interactions (Kloosterman et al., 2011), as does the IntAn's Social Anhedonia component (Mason et al., 2005). The only conceptual difference between IntAn and AQ-K's SocSki is the absence of physical anhedonia items in the latter. This can explain the imperfect correlation between them. The overlap between autistic and schizotypy "negative" traits was interpreted as a shared schizoid or "schizoidness" dimension (Ford & Crewther, 2014; Hurst, Nelson-Gray et al., 2007). The question arises whether this overlap in negative traits reflects shared aetiological factors between ASp and PSp (De Lacy & King, 2013), or a psychometric bias (Del Giudice et al., 2010).

Del Giudice et al. (2010) argued for a psychometric bias proposing that the negative trait overlap could be explained by a loose item formulation which would confound different constructs. Yet, speaking against the loose item formulation suggestion, clinical data support an overlap in negative *symptoms*, because of what is observed in diagnostic clinical interviews. Comorbidity exists between schizoid PD and Asperger's syndrome and schizoid PD (Lugnegård et al., 2012; Strunz et al., 2014). ASD individuals score higher in all negative schizotypic dimensions (Barneveld et al., 2011; Wakabayashi, Baron-Cohen, & Ashwin, 2012). Furthermore, correlations with broader personality dimensions suggest Introversion as a common underlying construct, in both general and clinical populations (Austin, 2005; Kanai et al., 2011; Strunz et al., 2014; Wakabayashi et al., 2006a). We think that it is unlikely that both personality questionnaires *and* clinical instruments suffer from loose item formulation, since the latter are designed for differential diagnosis. As a result, the overlap may be genuine, suggesting *transdiagnostically* common underlying mechanisms and aetiological pathways for negative features. Yet, similarity cannot be proven (Chapter 1.1.5.1), putting the burden of proof on the shoulders of those claiming that negative features are reliably different between ASp and PSp, and possibly other disorders (e.g. as we are doing here).

Globally, shared negative traits were poor at predicting behaviour (Chapters 4 and 5). If negative traits would be important to explain aetiological pathways of ASp and PSp's social cognition deficits, we should have obtained more convincing results from relationships between face/gaze processing and negative features in our data. Instead, autistic mentalizing deficits and positive schizotypy better explained our behavioural results. As for our GC task (Chapter 5), we observed that larger PCSF-K in men tended to associate with a smaller GC effect. These results resemble those on Introversion obtained by Ponari et al. (2013). We can interpret our and Ponari et

al. results in two ways. A first possibility is that the influence of introversion and social anhedonic shared traits (IntAn, SocSki) may owe to their association with autistic mentalizing deficits (ComMind, Ima), because negative traits relate to and might be *wrongly* confounded with mentalizing deficits (Bishop et al., 2004; Lau, Gau et al., 2013; Lau, Kelly et al., 2013; Sergi et al., 2007; Chapter 5). Possibly, the correlation between mentalizing deficits and negative trait dimensions could be due to the former causing the latter (Lau, Gau et al., 2013), in line with the "mindblindness theory" (Baron-Cohen et al., 1985), possibly manifesting in abnormal gaze processing (hyper-, hypo-arousal, gaze aversion or abnormal gaze saliency; see Chapter 6.3.2.1.).

A second possibility is that the smaller autistic liability to GC effects does in addition relate to an abnormal reward value, salience attributed to social stimuli (notably gaze), itself reflected in psychometric social anhedonia (Senju & Johnson, 2009). Another possibility is that ASp associate with an abnormal specialization of the gaze processing brain network (Senju & Johnson, 2009; see also Senju, Johnson, & Tomalski, 2015). It is possible that a smaller GC liability as a function of autistic traits is not solely due to autistic mentalizing deficit traits, but also to social anhedonic, negative or introvertive traits. However, these shared negative traits did neither explain GD perception, nor face/FLO/object processing (Chapters 4 and 5). Despite their saliency, the clear overlap between ASp and PSp, negative social anhedonic and introverted personality traits may not be as important as previously thought with regard to social cognition. At the very least, the scales measuring these traits should be questioned. We believe that research on disorders with social cognition would gain from a better and clearer distinction between social anhedonia (e.g. "I don't like social interactions") and mentalizing deficits (e.g. "I can't understand social situations"), although both may be linked (e.g. one can dislike social situations, *because* one cannot understand them properly; one cannot understand social situations properly, *because* one does not like them and failed to acquire social expertise).

Theoretically, ASp-PSp relationship models interpreted this overlap in negative symptoms in different ways. Rausch and Johnson (2008) elaborated on this overlap proposing ASD and SSD would be part of a "negative symptom spectrum". In contrast, Crespi and Badcock (2008) considered overlapping negative symptoms between ASp and PSp as an epiphenomenon, resulting from different underlying causes in each spectrum. Recent evidence supports the nonspecific and transdiagnostic presence of schizophrenia-like negative symptoms in mood-psychotic disorders, as well as other disorders such as autism and OCD (Cath et al., 2008; Foussias et al., 2014; Kaiser et al., 2011; Malaspina et al., 2014). Our results replicated an overlap in negative and social anhedonic traits, without them being mirrored by "shared" face and gaze processing deficits. We doubt that these "shared" traits are crucial to explain face and gaze processing behaviours, and possibly social

cognition, at least with regard to ASp-PSp relationships. Instead, our results support the diametrical model (Crespi & Badcock, 2008) emphasising opposite traits *in distinguishing social cognition features of ASp and PSp relationships* (see also: Dinsdale et al., 2013).

Social anhedonia should, however, remain included in future studies. Social anhedonia may be important for other reasons than looking for direct causation. We cannot rule out that negative symptoms impact social cognition in ASp and/or PSp (though not supported by our data). Given the literature on symptoms and traits, plus our results, we propose that the shared negative traits (a.o. social anhedonia), like corresponding symptoms, might be nonspecific, transdiagnostic (Cath et al., 2008; Foussias et al., 2014; Kaiser et al., 2011; Malaspina et al., 2014). Hence, shared negative traits and social anhedonia may act as vulnerability and aggravating factors, as part of "Internalizing" symptoms (Krueger & Eaton, 2015). If shared negative traits and social anhedonia do not differentiate ASp and PSp, we could speculate that social anhedonia and negative traits might act as (i) a basic *early or late risk factor* for developing several disorders (e.g. ASD, SSD), (ii) an *aggravating factor* interacting with other deficits or developmental insults (e.g. mentalizing deficits), (iii) and eventually a factor blocking access to *protective factors* stemming from social interactions.

We would like to enrich the last speculative conjecture with some concrete examples. Negative social anhedonic traits may put individuals at risk for developing several disorders. Early learning or mindreading deficits may be aggravated by social anhedonic traits/symptoms. Likewise, later positive features (e.g. odd behaviour, magical beliefs) may be also aggravated by social anhedonia. Moreover, social anhedonia may prevent individuals from getting attention, support and care from caregivers and peers when facing challenging developmental periods (e.g. childhood, adolescence). As such, social anhedonia may deprive individuals from protective or resilience factors obtained through social interactions. Importantly, social anhedonia would be heavily heritable, hence genetically grounded (Barrantes-Vidal, Grant, & Kwapil, 2015).

Yet, social anhedonia might also be secondary, i.e. resulting from previous developmental insults, social stressors, or traumatic experiences. As such, secondary social anhedonia may aggravate other existing troubles, and further impair the access to protective factors. As a result, although prominent in and characteristic of schizophrenia, negative symptoms are not specific to SSD, nor to PSp (Foussias et al., 2014). If true, the corresponding *negative personality traits* may not be specific to autistic, schizotypy or any other trait. Again, it does not make them less interesting, less predictive of transition to illness or irrelevant to understand the mechanisms of a disorder. Indeed, negative symptoms or traits might be bridging different disorders, possibly explaining transitions and co-occurences (see Chapter 6.6.8.). Hence, social anhedonic negative

traits could be considered as detailing broad personality features, similar to Introversion and more generally "Internalizing" features (Krueger & Eaton, 2015). Alternatively, social anhedonic negative traits could be part of a general "psychopathology" factor (Caspi et al., 2016; see Chapter 6.6.7.), extending in various PDs and disorders' phenotypes, and possibly acting as broad risk, or aggravating factors.

We also highlight that neuroticism and associated disorganized schizotypic traits should not be discarded from future studies. Despite their inability to predict behaviour in our studies (Chapers 4 and 5), Neuroticism shows many relationships with ASp and PSp. Neuroticism and disorganized schizotypic traits were part of shared traits (Chapter 3). Neuroticism is associated with autistic dimensions and, in particular, disorganized schizotypy in healthy controls (Austin, 2005; Burch et al., 2006; Claridge et al., 1997; Lin et al., 2013; Wakabayashi et al., 2006a). Neuroticism is higher in ASD patients than in control subjects (Kanai et al., 2011; Strunz et al., 2014), in schizophrenia and associated PDs (Camisa et al., 2005). Neuroticism associates with a higher risk for developing schizophrenia (van Os & Jones, 2001). Wakabayashi et al. (2012) also showed relationships between total AQ scores and disorganized SPQ dimension scores.

Neuroticism is relevant for ASp-PSp relationships as a broad vulnerability factor. Neuroticism lacks specificity but not sensitivity. Neuroticism is a non-informative (i.e. nonspecific) marker of vulnerability for psychopathology (e.g. affective disorders; Ormel et al., 2004), notably schizophrenia (van Os & Jones, 2001). Barrantes-Vidal et al. (2009) showed the importance of Neuroticism as a moderating factor between positive schizotypy and psychopathology and functioning measures. These authors argued in favour of Neuroticism's primary and nonspecific relevance not only for SSD but more generally for affective and psychotic disorders. Neuroticism plays a direct role in ASD, mood disorders, or is comorbid with ASD (Hofvander et al., 2009; Lugnegård et al., 2011; Rydén & Bejerot, 2008), and SSD (Ritsner & Gottesman, 2011). At the very least, Neuroticism might play an indirect role in ASD and SSD, via comorbid mood disorders. Like negative symptoms, Neuroticism is not uninteresting regarding ASp-PSp relationships, or any other disorder, not *despite* but *because* of its nonspecificity, as a symptom/trait that bridges different disorders (see Chapter 6.6.8.). Therefore, its role in the aetiological pathways, its underlying mechanisms, and its genetic liabilities remain unclear and should be further examined (Ormel et al., 2004), notably when being interested in ASp-PSp relationships.

6.5.4.1.1.2 OVERLAP IN POSITIVE TRAITS

More than the overlap in negative traits, the overlap in positive traits potentially questions the diametrical model (Chapter 3). AttDet and RRBeh represent "shared" positive traits together with positive schizotypy (UnEx), and also ImpNon (for AttDet; Dinsdale et al., 2013; Hurst, Nelson-Gray, et al., 2007; Russell-Smith et al., 2011). Uncertainty remains as to which aspects AttDet and RRBeh measure, so that these dimensions and their overlap may or may not be a problem for the diametrical model. Important issues for ASD-SSD relationships are (i) whether AttDet and RRBeh measure hyper-Mentalistic features (Chapter 3), (ii) hyper-Mechanistic features (Baron-Cohen, 2002), (iii) if (ii), how can the association with positive schizotypy hyper-Mentalistic features be explained while theories predict the opposite (Brosnan et al., 2010; Crespi & Badcock, 2008) or their overlap (Rausch & Johnson, 2008), and (iv) whether the overlap in positive traits can be explained by psychometric artefacts. Below we discuss these issues.

The overlap in positive traits may stem from a psychometric artefact, i.e. loose item formulation (Del Giudice et al., 2010). Concretely, AttDet may correlate with UnEx, because of loose item formulation, as hypothesized for negative traits (Del Giudice et al., 2010; Chapter 6.5.4.1.1.1). For instance, participants may have answered similarly to AttDet's autistic sensory abnormalities items (Leekam et al., 2007; Rogers & Ozonoff, 2005) and positive schizotypy items (UnEx). Alternatively, AttDet and RRBeh items may refer to abnormal, odd, eccentric features, shared with positive schizotypy, i.e. strange interests noted in both spectra (Esterberg et al., 2008; Kanne et al., 2012; Wolff, 1991). A similar explanation could apply for an underlying shared local bias, tapping into attention to details (Feigenson et al., 2014; Happé & Frith, 2006; Silverstein & Keane, 2011). In either case of artefactual overlap, it would not challenge Crespi and Badcock (2008) theory, nor support Rausch and Johnson (2008)'s theory. However, the existence of shared *clinical* symptoms would require that clinical symptom criteria are loosely formulated and unreliable. Since we doubt that *both* clinical symptoms criteria *and* questionnaires items are loosely formulated, we consider that the overlap in positive symptoms is genuine.

We consider it very problematic for the diametrical model that AQ-K's AttDet and RRBeh would genuinely overlap with positive schizotypy (UnEx) and hyper-Mentalism as suggested in Chapter 3. AttDet and RRBeh are autistic traits, and should accordingly relate to hypo-Mentalism/Empathizing and hyper-Mechanism/Systemizing (Baron-Cohen, 2002; Crespi & Badcock, 2008). AttDet was explicitely considered to be a precursor of Systemizing (Auyeung et al., 2009; Baron-Cohen, 2002). Despite lack of literature on RRBeh or similar traits, routines and repetitive behaviours may represent a Systemizing strategy used to make the close environment more predictable, more like a system, diminishing anxiety, as described in Asperger's syndrome (Attwood, 2007). As Systemizing resembles Mechanism on a conceptual level, and AttDet and

RRBeh should reflect Systemizing, it follows that AttDet and RRBeh would reflect Mechanism. Importantly, Systemizing was claimed to be either opposing (Brosnan et al., 2010) or orthogonal to Empathizing (Baron-Cohen, 2002), but never overlapping. Hence, an overlap of AttDet and RRBeh with hyper-Mentalistic positive schizotypy is incompatible with predictions of both E-S theory (Baron-Cohen, 2002) and, by proxy, with those of the diametrical model (Crespi and Badcock, 2008).

The problem that AttDet and RRBeh overlap with hyper-Mentalism is not helped by scarce empirical insight as to what these traits measure and how they relate with other constructs. We found only one report (e.g. correlation) showing that AttDet and RRBeh or analogue AQ dimensions positively associate with hyper-Mentalism/Empathizing or Systemizing. Walter, Dassonville and Bochsler (2008) reported a moderate positive correlation between SQ and AQ-BC's AttDet[26]. Claims that AttDet would be a precursor of Systemizing are, to our knowledge, poorly substantiated, and mostly theoretical (Ayeung et al., 2009; Baron-Cohen, 2002). Such a dreadth of empirical evidence prevents any firm conclusion about where AttDet and RRBeh should be placed in either the diametrical model (Crespi & Badcock 2008) or E-S theory (Baron-Cohen, 2002). Since AttDet and RRBeh dimensions originate from an alternative factor structure of AQ-K (Kloosterman et al., 2011), they have been poorly investigated. We are not aware of studies examining the relationships between these scales and other relevant constructs. To conclude, we are not sure what these dimensions truly measure, and observe that their association with hyper-Mentalistic positive schizotypy (UnEx) is unaccounted for by the diametrical model (Crespi & Badcock, 2008).

Taking the data and the literature as they are, we feel that the overlap between autistic and schizotypy positive features can be explained by a (i) common tendency to stereotypy (Rausch & Johnson, 2008) or (ii) genuine comorbidity of ASp for positive symptoms/traits. Firstly, positive traits would results from an underlying tendency to stereotypy, manifested either cognitively (e.g. positive symptoms, positive traits, UnEx) or behaviourally (e.g. repetitive behaviours, restricted interests; RRBeh). Clinically too, routines/Repetitive behaviours are a core ASD symptom (*DSM-5*, APA, 2013; Steer et al., 2010), reflected in some questionnaire dimensions (e.g. RRBeh; Kloosterman et al., 2011). Repetitive behaviours such as obsessive traits, obsessive PD, and OCD can be observed in ASD (Hofvander et al., 2009; Lugnegård et al., 2011, 2012; Wakabayashi et al.,

[26] Unpublished data show significant positive correlation between SQ Short score and AQ-K's AttDet (small effect size), but not RRBeh. Although SQ is probably the best proxy for Systemizing and Mechanism, such a weak correlation just confirms that AttDet associates with these constructs (Auyeung et al., 2009), rather than truly represent them.

2012) and SSD (Bottas, Cooke, Richter, 2005; Frías-Ibáñez, Palma-Sevillano, & Farriols-Hernando, 2014; Hadi, Greenberg, & Sirotta, 2012). Secondly, positive traits such as "Suspiciousness" (Wakabayashi et al., 2012), SSD positive symptoms (Blackshaw, Kinderman, Hare, & Hatton, 2001; Craig et al., 2004; Rydén & Bejerot, 2008), psychotic disorders and schizoid, paranoid and schizotypal PDs (Barneveld et al., 2011; Hofvander et al., 2009; see also Lugnegård et al., 2011) have been observed in ASD. Also, early autistic traits associated with later psychotic experiences (Jones et al., 2012). Unsurprisingly, positive traits could not reliably distinguish ASD and SSD patients (Naito et al., 2010; Spek & Wouters, 2010; Wakabayashi et al., 2012; Wouters & Spek, 2011). As for negative symptoms (Chapter 6.5.4.1.1.1), positive symptoms may therefore extend transdiagnostically, as it is now established for delusions (Bebbington & Freeman, 2017).

Despite the discussed overlap, we do not want to claim the nonexistence of clinical differences: these may just not be measured by current psychometric instruments. Attwood (2007) remarks that paranoia in Asperger's syndrome and schizophrenia feature subtle qualitative differences. They may stem from qualitatively distinct mechanisms, i.e. ToM deficits with or without attribution abnormalities (Blackshaw et al., 2001; Craig, Hatton, Craig, & Bentall, 2004; Pinkham et al., 2012). The literature remains uncertain whether this overlap in positive symptoms reflects similar underlying causal mechanisms resulting in subtle differences (multifinal outcomes) or distinct underlying causal mechanisms resulting in globally shared features (equifinal outcomes; Cicchetti & Rogosch, 2002). Current questionnaires are not able to capture these subtle differences in patients, since positive traits could not distinguish ASD from SSD (Naito et al., 2010; Spek & Wouters, 2010; Wakabayashi et al., 2012; Wouters & Spek, 2011).

To conclude, although the overlap in positive features is small, it is consistent (Wakabayashi et al., 2012). The diametrical model cannot explain this overlap (Crespi & Badcock, 2008). Only Rausch and Johnson's (2008) model could explain this overlap. As for negative traits/symptoms (Chapter 6.5.4.1.1.1), the overlap in positive traits/symptoms is a problem that vastly exceeds the scope of ASp-PSp relationships and will require more research. Another implication is more crucial for ASp-PSp relationships. As measured with the instruments considered, positive traits alone cannot distinguish between ASD and SSD (see also Appendices Table 28) contradicting previous claims (Konstantareas & Hewitt, 2001; Spek & Wouters, 2010). Further research should be conducted to know whether AttDet and RRBeh reflect hyper-Mechanistic features. Revision of AttDet, and RRBeh scales, adding other items, and parallel use with other instruments (e.g. Systemizing Quotient; Baron-Cohen & Wheelwright, 2004) might help us in improving these scales. Assessment of clinical and healthy populations with improved instruments may indicate whether this overlap in positive traits is artefactual or genuine, and which model best explains it.

6.5.4.2 FOCUS ON HYPER-MENTALISM IS PARTLY SATISFACTORY

The big problem with the diametrical model is that its hyper-Mentalism component raises as many questions as hopes. Hyper-Mentalism is one part of the Mentalistic distinction between ASp and PSp. Much more so than autistic hypo-Mentalism, PSp hyper-Mentalism raises several questions: (i) independent social cognition deficits in PSp is a relatively new idea (Ritsner & Gottesman, 2011), (ii) hyper-Mentalism in schizophrenia solely relies on one model of social cognition impairment in schizophrenia (Abu-Akel & Shamay-Tsoory, 2013), backed by growing evidence but flawed by enduring uncertainties, (iii) hyper-Mentalism applies only to a subset of schizophrenia and schizotypy features (Fyfe et al., 2008; Montag et al., 2011), while also involving the psychotic features of other disorders (Usnich et al., 2015), and (iv) these positive psychotic features may not tap the biological vulnerability to schizophrenia, but rather to healthy, protective or advantageous traits associated with psychosis (Grant, 2015). We discuss each of these points below, before discussing the implication for the diametrical model.

Independent social cognition deficits in PSp is a relatively new idea. Indeed, study of independent social cognition deficits has long been obscured by study of non-social fundamental cognitive deficits (Ritsner & Gottesman, 2011). Social cognition is a relatively new study field, marginal to the study of PSp. Social cognition is less studied than more general cognitive deficits (e.g. attention, episodic memory or executive functions; Braff & Freedman, 2002; Goldberg et al., 2003; O'Flynn et al., 2003; Ritsner & Gottesman, 2011). Social cognition deficits are, however, relevant to schizophrenia (Burns, 2004; Green, Horan, & Lee, 2015). As of now, they are not explicitly mentioned among *DSM-5* diagnostic criteria (APA, 2013) or as being relevant to the broader set of SSDs or other "functional psychoses", such as bipolar disorder (Ritsner & Gottesman, 2011; Shevlin et al., 2016).

The concept of hyper-Mentalism in schizophrenia relies on one model, i.e. the diametrical model (Crespi & Badcock, 2008). Brüne (2005) reviewed evidence of social cognition deficits in schizophrenia, mostly ToM deficits, and relied importantly on studies by Frith (1992), Hardy-Baylé (1994; see also Hardy-Baylé, Sarfati, & Passérieux, 2003), and Abu-Akel (1999). The diametrical model (Crespi & Badcock, 2008) essentially builds on the model by Abu-Akel (1999) and C. Frith (2004). Abu-Akel (1999) and Abu-Akel and Bailey (2000) proposed the existence of a hyper-theory-of-mind in schizophrenia, based on an over-attribution of mental states to self and others (for a recent review see Abu-Akel & Shamay-Tsoory, 2013). Unconstrained generation of numerous and/or faulty hypotheses on one's or other's mental states would result in impaired ToM. This

suggestion contrasts with the difficulty in generating hypotheses in negative schizophrenia or autism under-mentalizing (Fretland et al., 2015; Montag et al., 2011; see also: Chung, Barch, and Strube, 2013). Crespi and Badcock (2008) included this idea in their diametrical model (see also Badcock, 2004). The extended E-S model relies on a similar idea (Brosnan et al., 2010). The idea of hyper-Mentalism/over-mentalizing in the PSp continues to receive attention and partial support in recent years (e.g. Fretland et al., 2015; Montag et al., 2011; see also Chapter 3).

It has been suggested that hyper-Mentalism applies only to the "positive" subset of schizophrenia and schizotypy profiles; extending, however, to other disorders with psychotic features (Abu-Akel & Shamay-Tsoory, 2013 for a review). Positive features represent the most typical feature of schizophrenia and schizotypy, although both are multidimensional constructs including negative and disorganized dimensions (Arndt et al., 1991; Kwapil & Barrantes-Vidal, 2015; Liddle, 1987). Over-mentalizing in ToM task associated exclusively with positive features in schizophrenia patients, e.g. paranoid symptoms (Montag et al., 2011) and delusion proneness in healthy individuals (Fyfe et al., 2008). In contrast, under-mentalizing associated consistently with negative or disorganized symptoms in patients (Fretland et al., 2015; Montag et al., 2011), and mentalizing was dysfunctional in healthy relatives of patients with schizophrenia (Montag et al., 2012). Additionally, hyper-Mentalism/over-mentalizing is not specific only to the "positive" subset of schizophrenia, but extends to other disorders: bipolar disorder and borderline PD (Andreou et al., 2015; Usnich et al., 2015). Thus, over-mentalizing seems nonspecific to schizophrenia, pointing to a transdiagnostic mechanism possibly stemming from a shared aetiological pathway, understood as "psychotic" *lato sensu*.

At this point, we would like to consider that positive symptoms and traits manifesting hyper-Mentalism/over-mentalizing may not only account for the biological vulnerability to schizophrenia, but also for advantageous, protective or healthy traits associated with positive traits. Mohr and Claridge (2015) recently reviewed the idea that schizotypy, in particular positive schizotypy, is not problematic but reflects healthy, adaptive and possibly beneficial personality traits. Several authors distinguished between healthy schizotypic traits, and schizotypic traits representing schizophrenia liability: pseudo-schizotypy and neuro-schizotypy (Raine, 2006), healthy schizotypy and schizotypy *per se* (McCreery, 1997), and benign schizotypy and schizotypy *per se* (Claridge, 1997). Grant (2015) distinguished between psychosis-in-schizophrenia and schizophrenia-in-psychosis. The former element of this dichotomy refers to a benign, healthy or psychotic part of schizotypy, i.e. positive traits, not carrying the liability to schizophrenia, yet a potential endophenotype of psychosis. The latter would refer to the more pathological and biologically-rooted schizophrenia

liability. For Grant (2015), schizophrenia would result from the conjunction of both heightened positive and negative traits.

These distinctions between schizotypy as a schizophrenia (biological) liability, on the one hand, and schizotypy as healthy personality features, or liability to psychosis, on the other hand, are empirically supported (Cella et al., 2013; Grant, 2015). A cluster analytic study showed that adolescents presenting only heightened schizotypic traits did not show worse quality of life and distress than those low in all dimensions, while those high in positive, negative and disorganized did (Cella et al., 2013). As a result, the diametrical model's hyper-Mentalism may account for the healthier "positive" part of schizotypy and psychosis liability, whereas the overlapping models' "shared" disorganized/negative traits may account for the more detrimental schizophrenia liability. While the latter may be more informative regarding ASp and PSp aetiology and mechanisms, the former may be informative regarding healthy compensatory adjustments (Mohr & Claridge, 2015), and possibly cognitive styles (Brosnan et al., 2013, 2014).

In sum, the use of hyper-Mentalism is partly problematic, partly satisfactory in distinguishing between ASp and PSp, suggesting a revision of the diametrical model's Mentalism. Hyper-Mentalism is satisfactory to characterize PSp because further theoretical elaboration and recent empirical results support the existence of over-mentalizing in schizophrenia. Yet, hyper-Mentalism remains problematic in that over-mentalizing applies *only to the "positive" subset* of schizophrenia patients, including the extension to other disorders entailing psychotic features. Also, hyper-Mentalism only applies to the *cognitive* and not the affective mental states, since both are dissociable (Lavoie et al., 2014; Shamay-Tsoory & Aharon-Peretz, 2007), leaving aside the literature dealing with emotion processing deficits. In light of these recent results, the diametrical model's Mentalism should not be conceived as opposing spectra (ASp vs. PSp). Instead, the diametrical model should be reformulated as opposing transdiagnostic symptoms or syndromes (i.e. positive symptoms/traits vs. mentalizing deficits), and possibly *some* shared underlying mechanisms and liabilities on shared aetiological pathways (e.g. gaze sensitivity; Chapter 5; Hooker & Park, 2005; Matsuyoshi et al., 2014). This shift of distinction from diagnostic categories to syndroms/symptoms/traits will have implications regarding the model to be supported regarding ASp-PSp relationships.

6.5.4.3 ABSENCE OF PSYCHOMETRIC ACCOUNT FOR MECHANISM OR SYSTEMIZING

A weakness of the diametrical model is the uncertainty regarding the opposition between Mechanism/Systemizing and Mentalism/Empathizing (Crespi & Badcock, 2008). With the extension of the E-S model, Brosnan et al. (2010) not only demonstrated the association of psychotic and positive schizotypy with Empathizing but also their diametrical opposition to Systemizing. Since Systemizing is conceptually the closest dimension to Badcock's (2004) Mechanism, Brosnan et al. (2010) results offer a psychometric support to the diametrical model (Crespi & Badcock, 2008).

However, autistic traits did not show a corresponding opposition between analogues of Systemizing and Empathizing (f.i. Chapter 3). We previously discussed (Chapter 6.5.4.1.1.2.) the surprising overlap in positive autistic and schizotypic traits, contradicting the diametrical model. As possible explanations, we evoked (i) reliability and validity issues of AttDet and RRBeh, (ii) their somewhat poorly substantiated association with Systemizing, and (iii) the fact that the diametrical model cannot account for them (Crespi & Badcock, 2008). Still, before properly refuting the diametrical model, further studies should be performed to improve these AQ scales, thereby clarifying their relationships with Systemizing/Mechanism (Chapter 6.5.4.1.1.2.).

We are aware of arguments against the diametrical model; we refer to literature that reports on conflicting results on the dimensionality of autistic, schizotypy, Empathizing and Systemizing traits, notably AttDet (see Chapter 3). Obviously, this literature complicates the debate around Mechanism, Mentalism and the diametrical model. Some studies about ASp and PSp personality traits reported 2-dimension models, with "shared" and "diametrical" dimensions (Brosnan et al., 2010; Dinsdale et al., 2013; Chapter 3), as reported for the diametrical model (Crespi & Badcock, 2008). In contrast, another study supported a 3-dimension model with one "shared" and two "distinct" orthogonal dimensions (Ford & Crewther, 2014; Ford, Apputhurai, Meyer & Crewther, 2017), paralleling the E-S theory (Baron-Cohen, 2002; see also Abu-Akel, Wood et al., 2015). As for cognitive styles, Empathizing and Systemizing relationships themselves do not support or refute the diametrical model (Crespi & Badcock, 2008). Indeed, Empathizing and Systemizing showed inconsistent correlations across studies (negative: Brosnan et al., 2010; positive Wright & Skagerberg, 2012; null: Russell-Smith, Bayliss, Maybery & Tomkinson, 2013). Further work is need to know whether ASp-PSp relationships are best represent by a "shared" dimension accompanied by (i) one "opposite" dimension, as we argued (Chapter 3), or (ii) two "distinct" ones.

Other variables, such as sex, might influence dimensionality of Empathizing-Systemizing. Valla et al. (2010) observed that Systemizing and Empathizing were inversely related in men but independent in women. Men would experience a trade-off between Systemizing and Empathizing,

whereas women would not. This stresses the importance of controlling for socio-demographic variables, and the possible environmental sensitivity of these measures.

Further research is required to evidence psychometrically the opposition between Mentalism/Empathizing and Mechanism/Systemizing. This task is important since the Mechanism continuum complements Crespi and Badcock's (2008) diametrical model as well as the extended E-S model by Brosnan et al. (2010). The AQ has to be improved, and probably completed by the joint use of instruments such as the SQ and EQ. Gathering psychometric as well as behavioural evidence on the influence of a Mechanistic/Systemizing tendency as different cognitive styles in neurocognitive tasks seems promising to our understanding of aetiological pathways in ASD. Not being specific to social cognition, enhanced perceptual functioning in ASD (Mottron et al., 2006) could be tested with tasks taping functions related to hyper-Mechanistic features (e.g. Reed & Dassonville, 2013). Also, accounting for Mechanism/Mentalism duality may allow testing for ASD over-development of posterior perceptual brain areas, and their underconnectivity with frontal areas (Just, Keller, & Kana, 2013). Moreover, hypo-Mechanistic traits may be related to other personality traits such as disorganized schizotypy, and suggest diametrical phenotypes[27]. Indeed, larger disorganized schizotypy associated with worse perceptual organization in schizophrenia patients and healthy controls (Feigenson et al., 2014; Silverstein & Keane, 2011), as well as worse performance at visual backward masking (Cappe et al., 2012), an endophenotype candidate of SSD (Herzog, Roinishvili, Chkonia, & Brand, 2013).

6.5.4.4 THE DIAMETRICAL MODEL IS BACKED BY LITTLE, DIRECT EVIDENCE

Direct psychometric, behavioural or neuroimaging evidence for the diametrical model exists, but is scarce (Chapter 6.4.). Brosnan et al. (2010) and Dinsdale et al. (2013) support Crespi and Badcock (2008) diametrical model using personality traits, while other reports did not (Ford & Crewther, 2014; Ford et al., 2017). With regard to behavioural performances, autistic and schizotypic traits related to local biases in opposite ways (Embedded Figure Task, Russell-Smith et al., 2012). In social cognition tasks (mentalizing), Ciaramidaro et al., (2014) and Abu-Akel, Apperly et al. (2015) showed diametrical brain activations between ASD and SSD. Opposite brain activation patterns in the *same* areas are suggestive of a diametrical bias of the *same* mechanisms,

[27] Unpublished data confirm a significant negative correlation between AQ-K's AttDet and CogDis (Sierro, unpublished data).

and possibly on the *same* underlying aetiological pathway. Abu-Akel, Wood et al. (2015) reported on a diametrical performance in a perspective taking task in healthy population. These results are encouraging.

6.5.5 SUMMARY

We observed limitations of our psychometric instruments, our cognitive tasks, and the diametrical model onto which we based our investigations. Yet, we also suggested potential alternative methods and approaches that could solve certain problems we encountered. Improvements of questionnaires' reliability and validity should ensure that future studies profit from a better ability to account for hyper-Mentalistic/positive schizotypy and, in particular, hypo-Mentalism/autistic mentalizing deficit traits. Combining these with analogue instruments together with the use of modern validation techniques (e.g. item response theory's Rasch modelling) would further help in understanding the relationships between ASp and PSp via the personality traits. These improvements might help in identifying possible gender differences in questionnaire responding. We also mentioned numerous limitations regarding our pareidolia-proneness and gaze sensitivity measurement. Apart from many different solutions, we outlined the interest of using different imaging techniques to complement a behavioural measure of pareidolia-proneness (e.g. EEG; eyetracker).

The investigation of face/FLO/object processing as a function of autistic and schizotypic traits is promising, in particular if encoding duration and encoding-retrieval duration are manipulated. Likewise, we noted several limitations relative to our GD (e.g. ceiling effect, non-ecological stimuli), and our GC experiment (e.g. SOA, absence of non-social control condition). For GD, the most promising solutions might be the use of adaptive protocols measuring gaze self-attribution. For GC, we proposed using a range of SOA, adding a control condition with non-social stimuli, and adding schematic and ecological stimuli to better tease apart the underlying mechanisms behind GC deficits.

Finally, we observed limitations to the diametrical model, i.e. inability to explain psychometric overlaps, showing weak empirical support regarding hyper-Mentalism/over-mentalizing, the opposition between hyper- and hypo-Mentalism, as well as the difficulty to account for Mechanism/Systemizing. Along with psychometric solutions and the continued empirical testing of the diametrical model, our results and the literature support a revision of the diametrical model. Below, we will review an alernative model of ASp-PSp relationships, and praise for a broad

consideration reagarding underlying concepts (e.g. dimensional approaches, personality traits, cognitive styles, and neurocognitive endophenotypes).

6.6 CHALLENGES AND FUTURE PERSPECTIVES OF ASP-PSP RELATIONSHIPS RESEARCH

6.6.1 NEED FOR AN ALTERNATIVE MODEL OF ASP-PSP RELATIONSHIPS

Research needs an alternative model of ASp-PSp relationships, because no current model is sufficient to account for them (Chapter 6.4.2.; see Chisholm et al., 2015). Our results and the literature support the co-occurrence of "shared", "opposite", and "distinct" features between ASp and PSp (Chapter 6.4.2.). However, no current model of ASp-PSp relationships can encompass all 3 kinds of relationships (Chapter 6.4.3.). Obviously, several models of ASp-PSp relationships were proposed, departing from total distinction (Rutter, 1972), among which, some emphasized similarities (De Lacy & King, 2013; King & Lord, 2011), and others proposed a diametrical opposition in social cognition (Badcock, 2004; Crespi & Badcock, 2008). Extending and detailing this issue, Chisholm et al. (2015) recently reviewed, and examined 8 possible models of ASD-SSD relationships, potentially clarifying the issue of ASD-SSD comorbidity. We briefly present and discuss these models in the perspective of ASp-PSp relationships:

1. the **multiformity model**: ASD and SSD represent the same underlying disorder, yet featuring different manifestations,
2. the **increased vulnerability model**: one disorder may predispose to another but separate disorder,
3. the **chance model**: disorders such as autism and schizophrenia co-occur due to chance, misdiagnosis, pheno-mimicry, or superficial similarities,
4. the **stages model**: ASD and SSD are the same disorder but ASD can develop into SSD at a later stage,
5. the **associated liabilities model**: two disorders are separate but share underlying genetic risks or environmental ones,
6. the **independence model**: two disorders co-occur causing a third, distinct disorder,
7. the **diametrical model**: opposite alterations of common imprinted genes, in which co-occurrence would actually normalize or balance the cognitive phenotype,
8. and finally, **the multiple overlapping aetiology model**: disorders share *some* aetiological pathways, but not others.

In the light of a critical analysis of the literature, Chisholm and colleagues retained 4 plausible models among these 8 ones: (2) the "increased vulnerability model", (5) the "associated liabilities model", (7) the "diametrical model", and (8) the "multiple overlapping aetiologies" one. When we account for our own data, we would support the "multiple overlapping aetiology model", the "diametrical model", and the "associated liability model". Yet, none of these models alone is exact or sufficient to represent ASp-PSp relationships. No model accounts *at the same time* for shared, opposite and distinct features for ASp and PSp. We need a model that can achieve this.

6.6.2 DELINEATING AN ALTERNATIVE MODEL OF ASP-PSP RELATIONSHIPS

An alternative *integrative* model is necessary to account for all ASp-PSp relationships reported in the literature and in the present work. As presented in Chisholm et al. (2015), the different proposed models are not necessarily mutually exclusive. The idea of (8) "multiple aetiological pathways" could provide a basic structure integrating different ASp-PSp relationships' models, not only overlap, but also opposition and distinction.

This integrative model would have to account for overlapping relationships: shared features (e.g. negative symptoms/traits), suggestive of shared aetiological pathways, as proposed by the (8) "multiple overlapping aetiological pathways" and (5) "associated liabilities" models. For instance, these overlapping features would comprise negative symptoms/traits, such as social anhedonia (Craddock & Owen, 2011; Malaspina et al., 2014), impaired social cues processing, possibly cascading in ToM deficits (Chung et al., 2014; Roux, d'Arc, Passerieux, & Ramus, 2014; Senju & Johnson, 2009), and overlapping brain anatomic (Cheung et al., 2010) and functional abnormalities, in particular of the social brain (Pelphrey et al., 2011; Sugranyes, Kyriakopoulos, Corrigall, Taylor, & Frangou, 2011).

This integrative model would have to *also* account for opposite relationships, suggestive of opposition on the same aetiological pathways, as proposed by the (7) "diametrical model" (e.g. Mentalism). Diametrical biases of the same underlying mechanisms would comprise diametrical biases between PSp and ASp relatively to positive symptoms/traits vs. autistic mentalizing deficits (Dinsdale et al., 2013; C. Frith, 2004; see Chapter 3), possibly gaze perception (Chapter 5), gaze self-attribution judgement (over- vs. under-attribution; Hooker & Park, 2005; Matsuyoshi et al., 2014), brain activation in perspective taking, and intention attribution tasks (over- vs. under-activation; Abu-Akel, Apperly et al., 2015; Ciaramidaro et al., 2014), and possibly of encoding

styles during a non-social decision making task (i.e. the "jumping to conclusion bias" vs. "circumspect reasoning bias"; Brosnan et al., 2013, 2014; Lewicki, 2005).

In addition, this integrative model would have to account for distinct yet independent (i.e. not diametrically opposite) relationships, suggestive of distinct aetiological pathways (Chapters 6.4.2. and 6.4.3.). Distinct biases affecting distinct underlying mechanisms would comprise possible distinct personality traits (e.g. Ford & Crewther, 2014; Ford et al., 2017; see also later E-S theory, Baron-Cohen, 2002), PSp specific deficits in perceptual integration (Silverstein & Keane, 2011), and face configural processing deficits (Butler et al., 2008; Chambon et al., 2006; Shin et al., 2008; see Chapter 4), ASD specific deficits in gaze/social orienting (Sasson et al., 2007; Weigelt et al., 2012; see Chapter 4), and ASp-PSp differences in brain anatomy (Cheung et al., 2010; Radeloff et al., 2014), and brain activation patterns (Sugranyes et al., 2011). Looking at recent research, many current endophenotype candidates seem to represent distinct features (Snitz et al., 2006; Sucksmith et al., 2011; Chapter 6.6.5.)[28].

Further research is required to determine (i) whether these features are just apparently or genuinely shared, opposite or distinct, (ii) to identify the underlying mechanisms of each, (iii) their roles on a given aetiological pathway, from gene to behaviour, and (iv) to understand how they interrelate and interact. In this research approach, personality, dimensional models, cognitive styles, and endophenotypes will remain useful concepts, as far as they can be correctly defined, operationalized and articulated. Hence, we finally address the limitations and perspectives pertaining to these concepts, in light of our results, reflexions and the literature.

6.6.3 ISSUES USING PERSONALITY TRAITS AS PROXY FOR PSYCHIATRIC DISORDERS' LIABILITY

Personality traits remain limited in reflecting on an innate and biological liability to develop a psychiatric disorder. These limitations necessarily impact on (i) the comparison between liabilities between several disorders, (ii) their underlying mechanisms, and notably but not only (iii) the comparison between ASp and PSp. We discuss them in turn.

A first problem is that personality is at best an indirect and partial indicator of the biological liability to develop a psychiatric disorder. Recently, schizotypic personality was proposed to satisfy

[28] Obviously, it is not absolutely certain that endophenotype candidates are distinct for different disorders or specific to certain disorders. Most research focused on endophenotype candidates within disorder categories and not between disorder categories (i.e. transdiagnostically). Hence, it is not possible to conclude on distinctiveness and specificity, until they have been tested in other disorders.

criteria for a schizophrenia or psychosis endophenotype (Grant, 2015; Grant et al., 2014). The situation for autistic traits is comparable, given their presence in healthy relatives and general population, their heritability, and their common genetic underpinnings with ASp (Constantino & Todd, 2003; Lundström et al., 2012; Sucksmith et al., 2011). By definition, personality encompasses biological factors, *and* social/environmental factors, referred to as temperament and character, respectively (Millon et al., 2004). While temperament is defined as the innate, biologically-grounded part of personality, character refers to later adjustments as a function of social environment (Millon et al., 2004).

Since personality traits confound both temperament and character, personality traits are *indirect* indicators of the biological liability to develop a psychiatric disorder. This may add variability that complicates empirical replication in ASp-PSp relationships. As a solution, personality should be seen as reflecting the interactive effects of biological, and environmental risk factors. In this regard, efforts could be made to disentangle the biological from the social/environmental risk factors (e.g. traumatism, developmental problems, substance abuse, past psychiatric history, urbanicity, migration), as well as protective factors (e.g. a culture, socio-economic status, social support; Mohr & Claridge, 2015). Possibly, the use of instruments like Temperament and Character Inventory could help in grasping the innate biological ties of ASp and PSp (Cloninger, Przybeck, Svrakic & Wetzel, 1994).

A second problem is that disorder-related personality traits may manifest in a fragmented way in the general populations and patients' relatives (Happé & Ronald, 2008). In particular for ASp, unaffected ASp family members (BAP individuals) inherit a fragmented autistic phenotype (Happé & Ronald, 2008; Losh, Adolphs, & Piven, 2011). In other words, BAP individuals *only* feature *some* autistic traits, behavioural, or neural correlates (Happé & Ronald, 2008). Possibly, the presence of only a subset of autistic traits may only reflect on one ASp aetiological pathway among other possible ones. We are not aware whether a similar fragmented inheritance applies to schizotypy. Differently, it might be that some individuals differ in their expression of either one, the other or both the liability of schizophrenia and the healthy or benign adjustment, as previously discussed (Chapter 6.5.4.2.; see also: Hori et al., 2014). Nevertheless, this fragmented inheritance and/or the presence of adjustments may complicate the replication of ASp-PSp-like neurocognitive features or endophenotypes in the general and unaffected relatives' populations, in particular when total scores are used instead of dimension scores (e.g. Bayliss, Di Pellegrino et al., 2005; Matsuyoshi et al., 2014).

A third problem with personality traits is that illness liability can only be inferred from trait scores rather than being confirmed by familial psychiatric history. Many studies screen the general

population for personality traits and test individuals neurocognitive features or endophenotypes afterwards (e.g. Cappe et al., 2012). Obviously, most of the individuals do not feature heightened traits, and most of them who do will never develop the illness, as certain traits are benign and adaptive (Chapter 6.5.4.2.). Among the various possible research designs (Mason, 2015), a solution we favour would be by testing unaffected healthy relatives, because (i) their familial psychiatric history is known, (ii) their liability to illness is deduced from familial relatedness, and not only inferred from traits, and (iii) their relatedness with affected relatives makes it safer to interpret heightened personality traits as reflecting the genetic/biological liability for this particular investigated disorder. Applying the same logic to screening procedures of healthy student populations would be theoretically possible. Yet, asking for familial psychiatric history shifts the research question from individual differences to clinical disorders. Such a procedure would raise ethical questions (e.g. risk of stigmatization), require comprehensive ethical authorizations, and remain prone to uncertainties (e.g. defensive responding; Mohr & Leonards, 2005).

Finally, several solutions could be considered to solve (i) questionnaire length, (ii) the correct accounting of multidimensional personality profiles, (iii) the ecological validity of personality-related measurement, and (iv) the understanding of the relationships between multiple traits, and variables. As for questionnaire length, adaptive methods could make the testing time shorter (e.g. Fonseca-Pedrero, Menéndez, Paino, Lemos-Giraldéz, & Muñiz, 2013). Adaptive questionnaires refer to questionnaires for which the selection of items is updated as a function of respondents' ongoing responses. Obviously, adaptive questionnaires demand improvement of questionnaires and use of statistical methods stemming from item response theory. The issue of multidimensionality and personality profiles refers to the difficulty of current research to account for different personality profiles, despite acknowledgment of multidimensionality of schizotypy. To put it shortly, there is a risk to consider certain particular traits as sufficient (sensitive) and specific to account for a given phenotype or disorder liability (e.g. positive schizotypy: Grant, 2015; Mohr & Claridge, 2015). Against this view, a given set of traits may, alone, only represent a subset of a multidimensional phenotype, and they may not be specific to the phenotype in question. Technical solutions, such as latent profile analyses, cluster analyses or multiple regressions with interactions (all of these on large samples) may help to further account for the complexity of these multidimensional constructs (Cella et al., 2013; Hori et al., 2014; James, Dubey, Smith, Ropar, & Tunney, 2016).

As for ecological validity, personality questionnaires obviously are limited by desirability biases, insights/metacognitive impairments, and their distance from actual behaviours. Experience sampling methods (e.g. Kwapil, Brown, Silvia, Myin-Germeys, & Barrantes-Vidal, 2012) could be

used to discover the frequency and relationships between concrete behaviours, assumed to correspond to autistic and schizotypic personality traits (e.g. “These last few days, I had the impression that some was laughing at me.”; e.g. “Today, I had a problem in understanding someone else intentions.”). Additional items measuring variables such as stressful life events, or social interactions, may shed light on certain traits (i.e. disorganized traits within a stressful life event may not be interpreted as disorganized traits in a non-stressful period; Cohen et al., 2015). Finally, as for the understanding of multiple variables at multiple levels, network analysis of personality traits might facilitate the visualization and analysis of interrelationships between personality traits (Chapter 6.6.8.; Borsboom, 2017; Borsboom and Cramer, 2013). We further detail this point after discussing cognitive styles and endophenotypes.

6.6.4 ISSUES USING COGNITIVE STYLES AS PROXY FOR PSYCHIATRIC DISORDERS’ LIABILITY

Paralleling the aforementioned personality issue, cognitive styles only indirectly and partially reflect disorders’ liability, and underlying mechanisms. At a cognitive level, Crespi and Badcock (2008) talked about opposite “cognitive styles” for ASp and PSp. The use of this concept has many implications, which should be considered in further research. As recently reviewed by Kozhevnikov et al. (2014), cognitive styles may be defined as stable individual differences in the way people process information, from lower (i.e. perception, memory) to higher cognitive levels (i.e. thought and problem solving). Analogous to personality, cognitive styles do not reflect mere innate, biologically-grounded abilities, but are *sensitive to acquired environmental factors* (e.g. culture, professional, educational, family). At best, they *indirectly* account for an underlying cognitive phenotype, and disorder liability. As a consequence, investigating cognitive styles should be accompanied by an attempt to disentangle environmental factors (e.g. domains of study/work; learning history) from more innate underlying cognitive phenotypes (e.g. gaze processing abnormalities). Cognitive style sensitivity to environment variables limit the expectations to tap disorders’ biological liability or endophenotype for ASp or PSp. Before claiming to account for disorders’ liabilities using cognitive styles, one should first exclude the confounds of environmental variables, vocational choices, and hobbies. Obviously, the task may prove challenging, as it remains unclear how neuroscience, cognitive functions and cognitive styles articulate (Kozhevnikov et al., 2014).

Taking an opposite perspective, cognitive styles’ sensitivity to environmental variables represents an opportunity to account for healthy adjustments, rather than mere deficits. The concept

of cognitive style is rarely explicitly evoked in schizotypy and autistic cognitive biases. For autistic phenotype, Happé (1999) explicitly addressed the question as to whether local processing bias would be a cognitive style rather than a deficit. In schizotypy, Fyfe et al. (2008, pp. 1316, 1324) precisely evoked a "hyper-associative cognitive style" when addressing the fundamental mechanisms behind positive symptoms, over-mentalizing and apophenia. In both cases, the concept of cognitive style underlines that disorder's liability does not necessarily associate with deficits. Instead, disorder's liability may associate with a learnt processing style, possibly resulting in unaffected performances or even advantages. Moreover, since the cognitive style concept emphasizes the *difference in strategies* rather than the *difference in performance*, it is more adapted to individuals from the general populations, typically targeted by our research designs. Also, the concept of cognitive style implies an interaction with environment, and offers the possibility to investigate it. Indeed, a cognitive style in one environment might be beneficial to performance, whereas it might not be in another environment. In this respect, the concept of cognitive style might be crucial to understand protective/resilience factors or advantageous abilities (e.g. creativity, verbal fluency, ease with concepts), associated with positive features, and possibly reflecting a psychotic endophenotype (Grant, 2015; Mohr & Claridge, 2015). Likewise, the cognitive style approach may further improve our understanding of mysterious autistic compensatory strategies ASD use in ToM tasks (U. Frith, 2004). Finally, cognitive style might be useful for ASp cognitive remediation therapies, as well as understanding the origin of enhanced perceptual functioning (Mottron et al., 2006).

Adopting an adapted model of cognitive styles, such as the one proposed by Kozhevnikov et al. (2014), is key for further testing of ASp-PSp relationships using healthy individuals. The cognitive styles' concept suffers from unclear definitions, different operationalizations, and insufficient integration with cognitive psychology and neuroscience fields (Kozhevnikov et al., 2014). Yet, one precise point ties to the number and structure of cognitive styles, and their relationships with different cognitive functions. Departing from traditional dichotomies such as "analytic" vs. "intuitive", Kozhevnikov et al. (2014) propose their own model of cognitive styles that dissociates cognitive styles (e.g. context dependence/independence, internal vs external locus of processing) from cognitive processing levels or functions (e.g. perception, concept formation, metacognition). To put it shortly, cognitive styles used to be considered as unidimensional dichotomies (e.g. analytical-intuitive) affecting all cognitive functions at all levels (i.e. from perception till conceptual level). Against this view, the authors propose that cognitive styles may apply differently at different cognitive levels, for different functions. For instance, an individual may exhibit a local processing style for perceptual functions, but a global processing style for other cognitive functions such as a concept formation. If we apply this model to ASp-PSp-related

cognitive styles, such Mechanism-Mentalism (Crespi & Badcock, 2008), hypotheses regarding ASp-PSp cognitive styles should be revised, and made more precise. For instance, does hyper-/hypo-Mentalism apply to all cognitive levels indiscriminately? We can also ask whether hyper-/hypo-Mentalism only applies to over-/under-mentalizing in high level cognitive functions (e.g. intention attribution tasks), and not to more fundamental automatic social cues perception (e.g. face or gaze processing), as we previously suggested (Chapter 6.4.2.2)?

An important output from this work is the discontinuity of Mentalism across cognitive levels. Our psychometric and behavioural results (Chapters 3, 4 and 5) as well as the literature (see Chapter 6.4) emphasize that a Mentalistic cognitive style opposing ASp and PSp may not manifest identically across all cognitive levels (as initially proposed by Crespi & Badcock, 2008). Indeed, Mentalism best applies to higher level cognitive functions involving self-reported self-representation (Chapter 3), ToM/mentalizing tasks, and possibly gaze direction attribution (see Chapter 6.4 for a summary). In contrast, our social perception tasks associated with Mentalism's components separately, either autistic mentalizing deficits traits or positive schizitypy traits (Chapter 4; Chapter 5:GC). Hence, the diametrical model of ASp-PSp ought not to be interpreted literally: some features will be diametrical, others will be shared, and others may still be distinct (see Chapter 6.6.2.), depending on the cognitive level considered (Kozhevnikov et al., 2014). Further research is required to better understand the relationships between cognitive styles and disorders' liabilities, and protective factors. In this approach, neurocognitive endophenotypes and their relationships also have to be thoroughly investigated, as they may underlie personality, cognitive functions and their expression as cognitive styles.

6.6.5 NEUROCOGNITIVE ENDOPHENOTYPES

We believe that cognitive style and personality have to be considered along more fundamental neurocognitive endophenotypes. All should be jointly studied and distinguished by measuring environmental as well as biological variables. Yet, as underlined by Lainhart and Lange (2011) biological endophenotypes have a different status than personality or behavioural endophenotypes. Indeed, the former intervene earlier than the latter in the aetiological pathway, linking genes to symptoms, and making them more informative. A crucial question is to be able to correctly select the levels and functions targeting the relevant mechanisms underlying a given aetiological pathway. It should be investigated whether Mentalism distinction correctly describes personality traits, cognitive styles and/or neurocognitive endophenotypes. It is possible that Mentalism only applies to certain domains (e.g. cognitive style), at certain cognitive levels, and

using certain tasks, targeting certain functions, as we remarked in the previous chapter (Chapter 6.6.4.).

To put it briefly, Mentalism may distinguish ASp and PSp profiles only under certain conditions, and not necessarily as an endophenotype. Building on Grant (2015), hyper-Mentalism, as a psychotic feature, may rather reflect healthy adjustment and psychosis-proneness (Mohr & Claridge, 2015), over-mentalizing and hyper-associative cognitive style (Brugger & Graves, 1997; Fyfe et al., 2008). Neurocognitive endophenotypes, such as perceptual integration deficits, or face configural and memory deficits (Calkins et al., 2005; Silverstein & Keane, 2011) may co-occur with the above-mentioned cognitive style, cause opposite social cognition deficits, and reflect schizophrenia-proneness (Grant, 2015) or "neuro-schizotypy" (Raine, 2006). Likewise, the hypo-Mentalistic autistic mentalizing deficit traits, possible mentalizing or ToM deficits, and their behavioural manifestations of abnormal face or gaze processing (e.g. smaller GC liability) may stem from a basic gaze neurocognitive endophenotype (e.g. Matsuyoshi et al., 2014; Scheeren & Stauder, 2008; Tajmirriyahi et al., 2016; Wallace et al., 2010). This basic endophenotype would affect gaze processing due to various possible mechanisms (Chapter 6.3.2.1.). This endophenotype could associate with other biases or compensatory adjustments, such as an external encoding bias (Lewicki, 2005) protecting from over-generation of associations, and a fact-oriented Systemizing style encouraging factual reality-testing (Baron-Cohen, 2002; Brosnan et al., 2014).

We could speculate different origins for hypo- and hyper-developments of Mentalism and Mechanism in ASp and PSp (Chapters 6.6.3. and 6.6.4.). PSp hyper-Mentalism may stem from cognitive style/personality adjustment based on endophenotypes, themselves unrelated to social cognition. In contrast, ASp hypo-Mentalism may stem from fundamental endophenotypes related to social cognition (e.g. altered gaze and face processing), resulting in a cascade of social deficits at higher cognitive and clinical levels. Speculatively, PSp hypo-Mechanism may stem from basic neurocognitive endophenotypes (e.g. deficits in perceptual integration), or others (e.g. deficits in executive functioning and sustained attention), resulting in congruent deficits at higher cognitive and clinical levels, or compensatory development of an associative/internal encoding style. In contrast, ASp hyper-Mechanism may stem from enhanced perceptual functioning at sensory function levels, resulting in or associated with a Systemizing or Mechanistic cognitive style at higher cognitive and phenomenological levels. The understanding of ASp-PSp relationships rests on a better understanding of the relationships between personality, cognitive styles, neurocognitive functions and endophenotypes.

We support the idea to build theories/models starting from the most fundamental levels (e.g. genes) gradually to the higher phenomenological levels (e.g. symptoms/traits). In this respect,

neurocognitive endophenotypes often are more fundamental than cognitive styles, as we learnt from the importance face/FLO/object processing prevailing over pareidolia-proneness (Chapter 4). Hence, before considering solely cognitive styles, we ought to consider the fundamental neurocognitive endophenotypes onto which cognitive styles may have developed (Gur et al., 2015; Lainhart & Lange, 2009; Snitz et al., 2006; Sucksmith et al., 2011). Importantly, the endophenotype level was not taken into account in the diametrical model, nor any existing model (Crespi and Badcock, 2008; Chisholm et al., 2015). Crucially, the same principle holds for research on relationships between other disorders spectra, such as OCD, ADHD, and bipolar spectra.

6.6.6 TRANSDIAGNOSTIC AETIOLOGICAL PATHWAYS INSTEAD OF DISORDER-SPECIFIC ONES

Far from being disorder-specific (i.e. distinct), personality traits, cognitive styles, neurocognitive, and biological endophenotypes should be considered as *transdiagnostic* features, possibly representing overlapping, or opposite aetiological pathways (Krueger & Eaton, 2015; Malaspina et al., 2014; van Os & Kapur, 2009). For instance, despite overlaps between ASp and PSp, neurocognitive endophenotypes still are inferred as disorder-specific. For instance, PSp neurocognitive endophenotype candidates were seldom investigated in ASD, and vice versa (Snitz et al., 2006; Sucksmith et al., 2011). Research traditions tend to be separated between ASp and PSp, reflecting a diagnostic-based specialization. Yet, transdiagnostic examination of endophenotype candidates may help in better understanding the underlying mechanisms, from genes to behaviours. Indeed, we propose that a possible confound of current endophenotype research might stem from reliance on disputable diagnostic categories. We speculate that the heterogeneity *within* disorders' categories and the similarities *between* disorders' categories might add variability to empirical results. As a result, this would diminish the sensitivity of the statistical results, thereby impeding the identification of endophenotype candidates and genes associated with a *syndrome* (about heterogeneity and similarity: Amaral, Dawson & Geschwind, 2011; Ritsner & Gottesman, 2011). For instance, if some studies test schizophrenia patients without distinction of symptoms, this might obscure the relevance of certain endophenotypes. Indeed, certain endophenotypes may be relevant only for a subset of patients featuring *certain symptoms* or a *syndrome* (e.g. disorganized), likely reflecting a given underlying aetiological pathway, and specific mechanisms. Also, ignoring commonalities between schizophrenia and bipolar manic symptoms might lower the power to detect a possible common *transdiagnostically*-relevant PSp endophenotype, and its underlying genes (Grant, 2015; Shevlin et al., 2016). Crucially, this reasoning could be extended to other disorders,

and disorders' spectra. Hence, a transdiagnostic approach would help in explaining not only the relationships between ASp and PSp, but also between other sets of disorders or spectra (e.g. ADHD-ASp, OCD-ASD, PSp-depression/anxiety, and PSp-OCD). Moreover, this approach involves the personality traits, and cognitive styles associated to corresponding disorders and their liabilities. In this respect, a common model is required to be able to account transdiagnostically for all symptoms, traits and other correlates, that is: *syndromes* rather than *disorders*.

6.6.7 NEED FOR A TRANSDIAGNOSTIC MULTIDIMENSIONAL FRAMEWORK

Transdiagnostic multidimensional[29] models may be useful for understanding ASp-PSp reltationships. Indeed, they provide a global representation of disorders' symptoms or traits, useful for understanding their relationships and their aetiological underpinnings. Before the *DSM-5* (APA, 2013) was published, a movement proposed a paradigm shift from a categorical to dimensional nosological system (Krueger, 2005; but see categorical-dimensional model: Maser et al., 2009; categorical supporters: APA, 2013; Lenzenweger, 2010; James et al., 2016). For instance, Krueger (2005) advocated for a continuity between healthy personality, PDs (axis II in *DSM-IV-TR*, APA, 1994), and clinical disorders (axis I in *DSM-IV-TR*, APA, 1994). Also, he advocated finding the minimum number of dimensions to account for most disorders. *In this dimensional perspective, a disorder is not a set of disorder-specific symptoms but a disorder-specific combination of transdiagnostic symptoms.* In this context, comorbidity could result from the interplay of a limited set of dimensions, impacting on the liability to multiple disorders (Krueger & Markon, 2006). In its latest edition, the *DSM-5*, however, conserved the categorical approach, and just mentioned the dimensional approach to diagnosis as an alternative to categorical models limitations (APA, 2013, pp. 12-13). Still, such transdiagnostic models might clarify the issue of ASD-SSD comorbidity, and relationships, supporting one or another ASp-PSp relationship model (Chapter 6.6.2.; Chisholm et al., 2015 for a review).

Several authors proposed speculative transdiagnostic multidimensional models. van Os and Kapur (2009) attempted to fractionate the schizophrenia construct, as part of their "saliency hypothesis". They proposed a transdiagnostic multidimensional model to account for several psychiatric disorders. Indeed, these authors imagined that psychiatric disorders could be modelled using a 5-dimension model comprising: (1) psychotic symptoms, affective dysregulations comprising (2) depressive symptoms and (3) mania, and developmental impairments comprising (4)

[29] We used the term "multidimensional" because it is the most representative of the studies related, although some studies did not use dimensions, i.e. latent variables resulting from FAs, but just PC from PCAs.

negative symptoms, and (5) cognitive impairments. Later, van Os (2009) refined his model and added a sixth dimension (6) disorganisation. Obviously, this model did not feature autistic dimensions. Similarly, Andrews et al. (2009; cited by Krueger & Eaton, 2010) proposed a *DSM-5* meta-structure grouping disorders in 5 clusters: neurocognitive disorders (e.g. dementias), neurodevelopmental disorders (e.g. autism), psychoses (e.g. schizophrenia), emotional or externalizing disorders (e.g. unipolar mood and anxiety disorders), and externalizing disorders (e.g. antisocial behaviour and substance use disorders). Such a model would feature autistic symptoms, yet, as the previous one, it is only theoretical.

Empirical studies began to uncover the transdiagnostic models, and their minimum number of dimensions able to account for psychiatric disorders' diversity, notably ASD and SSD. Krueger and Eaton (2015) recently summarized these new developments. "Externalizing", "Internalizing" and "Thought disorders" dimensions were identified as important transdiagnostic dimensions, for respectively, antisocial/substance use disorders, mood/anxiety disorders, and psychosis. Interestingly, "Thought disorders" was identified as a third dimension, whose addition was necessary to account for psychotic features (Krueger & Eaton, 2015). Importantly, the relevance of transdiagnostic dimension "Thought disorders", to distinguish *between* psychotic and non psychotic *disorders*, parallels the relevance of a disorganized trait/symptom dimension *within PSp* (Arndt et al., 1991; Claridge et al., 1997; Liddle, 1987; Raine, 1991). Lahey et al. (2012) and Caspi et al. (2014) additionally identified a general psychopathology factor, named p-factor, and representing the broad risk for psychopathology *lato sensu*, along with the above mentionned triad (Internalizing, Externalizing and Thought disorder). However, these models did not feature autistic symptoms, and may not account for ASD.

Noordhof, Krueger, Ormel, Oldehinkel and Hartman (2014) attempted to integrate the autistic symptoms within a transdiagnostic multidimensional model. They conducted a longitudinal study on the general population, and confirmed the relevance of the previous dyad of Externalizing, Internalizing, plus the general p-factor. The models with the best fits demanded that autistic symptoms constituted a specific domain, along with a dimension accounting for attention and orientation problems.[30] However, these authors did not investigate a possible role for the psychotic "Thought disorders" dimension. Hence, we are bound to speculate on the empirical relationships

[30] Among the autistic symptoms, we note the presence of "Reduced social contact and interest" correlated with the negative-like Internalizing's "Withdrawn-depressed" symptoms, whereas "Difficulty in understanding social information" rather associated with "Attention problems" dimension. This is in line with our replication of the overlap between "shared" disorganized and negative traits (Chapter 3), and all the reflexion regarding negative symptoms (Chapter 6.5.4.1.1.1.), whereas "opposite" mentalizing deficit traits differ from negative traits and might represent autistic traits and symptoms with a better degree of specificity (Chapter 3; Chapter 6.3.1.).

between clinical transdiagnostic profiles of ASD and SSD symptomatic dimensions. To our knowledge, no complete transdiagnostic model has yet been empirically validated. Obviously, this would be a difficult task, and it may be impossible.

ASp-PSp relationship research would benefit from a complete transdiagnostic multidimensional model. Showing a correspondence between autistic and psychotic clinical symptoms relationships and autistic and schizotypic traits relationships would be an approach (e.g. Chapter 3; Dinsdale et al., 2013). An instrument based on such a transdiagnostic multidimensional model would make it technically possible to examine overlapping, opposite and distinct relationships between autistic and psychotic symptoms, and possibly confirm the ASp-PSp traits relationships (Chapter 3). First, it would be possible to confirm whether autistic and psychotic symptoms would aggregate for a positive symptom dimension (e.g. positive symptoms, attention to details, restricted and repetitive behaviours) and negative symptom dimension (e.g. anhedonia). Second, it would be possible to confirm whether positive symptoms (e.g. paranoia, delusion, magical thinking) oppose to autistic social cognition problems (e.g. mentalizing deficits). Third, it would be possible to test whether other symptoms are simply distinct/independent, and show few or no overlap (e.g. Ford & Crewther, 2014). Finally, it would be possible to judge whether ASD-SSD symptomatic relationships correspond to autistic and schizotypic traits relationships observed on patients or healthy populations (Chapter 3; Dinsdale et al., 2013; Hallerbäck et al., 2012; Konstantareas & Hewitt, 2001; Spek & Wouters, 2010).

A correspondence between clinical and personality trait dimensional models may just confirm what is already known (Chapter 3; Del Giudice et al., 2014; Dinsale et al., 2013; Ford & Crewther, 2014). Indeed, studies about dimensional relationships between ASD and SSD *clinical* symptoms are less developed than ASp-PSp relationships using *healthy* autistic and schizotypic traits (Chapter 3; Dinsdale et al., 2013). The multidimensional studies on autistic and psychotic symptoms we previously cited have not yet combined psychotic and autistic symptoms in the same model (e.g. Caspi et al., 2014; Noordhof et al., 2014). As such, the relationships between autistic and psychotic symptoms within transdiagnostic models need to be investigated. In contrast, studies about the relationships between ASp and PSp via personality traits are numerous, and more informative, since they combined autistic and schizotypic traits (Chapters 1.4.2. and 3). As far as the dimensional approach is valid, ASD-SSD symptom relationships should resemble ASp-PSp trait relationships.

If clinical and personality trait dimensional models correspond, it could justify transdiagnostic research on psychiatric disorders' aetiological pathways for ASp and PSp. Each clinical dimension would be associated with a transdiagnostic aetiological pathway. Some of these

putative pathways would be "shared" or "opposite" concerning both autistic and psychotic symptoms/traits. In contrast, other pathways might be "distinct", or specific to one of the spectra, one autistic or psychotic dimension. Concretely, cognitive styles, neurocognitive and biological endophenotypes could be researched by associations to disorders' dimensions, instead of diagnostic categories. It would emancipate research from current diagnostic categories that confound different syndromes and their associated aetiological pathways. As a result, it would be possible to understand more clearly the causal pathways between genes and certain symptoms, to understand the impact of certain *combinations* of symptoms. However, dimensional models present important psychometric, clinical, as well as ideological challenges, as demonstrated by the discussions surrounding the dimensional approach in *DSM-5* (APA, 2013).

Although our use of dimensional models helped us in clarifying the issue of ASp-PSp relationships, going beyond either categorical or dimensional views might be useful. Our use of a dimensional approach showed limited psychometric properties of autistic and schizotypic traits (Chapters 2 and 3). Dimensions showed limited help for understanding ASp-PSp traits relationships (Chapters 3 and 6.5.1.), and limited ability to clarify the relationships between ASp-PSp traits and cognitive mechanisms (Chapters 4, 5 and 6.5.2.). Dimensional models are too global when summarizing symptoms and traits in dimensions (Goekoop & Goekoop, 2014), which may confound unrelated aetiologies. Further than categorical-dimensional false dichotomy, a third network-based approach might open new perspectives (Borsboom, 2017).

6.6.8 THE ALTERNATIVE OF TRANSDIAGNOSTIC SYNDROMAL NETWORK MODELS

The limitations of the present work and the issue of ASp-PSp relationships showed the advantage of raising a much more global issue: nosology, the structure of psychiatric illness as an explanation for comorbidity (Chapter 6.6.7.). In the previous chapter (6.6.7.), we showed that transdiagnostic multidimensional models attempted to compensate for flaws in categorical model (e.g. Krueger & Eaton, 2015). Yet, it does not follow that multidimensional model are the only and best alternative to categorical ones, such as the *DSM-5* (APA, 2013). Borsboom and Cramer (2013) reversed the usual rationale of disorders as the expression of only few latent dimensions. They proposed to consider personality or disorders as complex networks of multiple causally connected symptoms or traits. Network models of psychiatric disorders, or Psychopathology Webs, may provide a descriptive, explanatory and predictive solution for nosological problems (e.g. nature, boundary and comorbidity; Goekoop & Goekoop, 2014; Borsboom, 2017; Borsboom & Cramer, 2013). This may obviously impact ASp-PSp relationship research.

Goekoop and Goekoop (2014) summarized the different nosologic systems that lead to and justified the use of network models or Psychopathology Webs. We present the arguments of these authors. The categorical approach, while reliable and precise, is flawed by insufficient validity, tremendous inflation of diagnostic categories, and impossibility to claim a strict demarcation between them, precisely because of comorbidities. The component approach (PCA) and the multidimensional approach[31] appropriately reduced information. They allowed to redefine disorders as recombinations of a limited number of components or dimensions (around 10 elementary syndromes). Yet, these methods did not solve the issue of comorbidity. They did not provide enough information and lack accuracy. In addition, the (multi)dimensional approach hypothesize an unknown latent dimension, bound to a mathematical abstraction until contradictory evidence. The solution for this nosological puzzle would be to avoid either ultra-precise categorical approach or vague and disputable multidimensional one, and opt for a compromise: the network approach (Goekoop & Goekoop, 2014; see also: Borsboom, 2017; Borsboom & Cramer, 2013).

In the network model approach, a disorder can be defined as a dynamic network of causally related symptoms (Kendler, Zachar, and Carver, 2011). For Goekoop and Goekoop (2014), symptoms can be graphically represented as nodes. The links between these symptoms (i.e. correlations) can be represented by edges[32]. Richly connected nodes are called hubs. Sets of interconnected nodes are called communities, clusters or modules. Rephrased in the psychopathological context, network's clusters of symptoms constitute syndromes. Among a symptoms' cluster or a syndrome, authors describe two kinds of symptoms: (i) core symptoms and (ii) bridge symptoms. Bridge symptoms connect one cluster to another one. In contrast, core symptoms do not bridge with other network clusters. In particular, bridge symptoms might account for comorbidity.

In theory, network models have several advantages over other models (Goekoop & Goekoop, 2014). Network models permit the examination of a multifactorial aetiology in all its complexity. Network models reconcile the categorical idea of segregated (i.e. clustered) disorders with the dimensional one of integrated (i.e. connected) disorders. This last feature allows to account for comorbidity. Network model approaches could be integrated in longitudinal designs and use methods such as experience sampling (e.g. Kwapil et al., 2012). As such, they could additionally

[31] Goekoop and Goekoop (2014) remind the readers that component-based methods (PCA) does not hypothesize the existence of latent variables, whereas dimension methods (FA) hypothesize the existence of latent variables (dimensions).

[32] Relationships between nodes are represented as simple edges when correlations have been computed between the symptoms they represent. Relationships between nodes can be represented as arrows when directional analyses (e.g. regression-based) have been performed between the symptoms they represent.

produce time-series data accounting for the temporal dynamic of disorder development. Crucially, network models clarifiy the specific role of symptoms (e.g. core, bridge, hubs). Global hubs and bridge symptoms might be sources (causing symptoms) rather than sinks (resulting symptoms). Since understanding aetiologies, predicting or treating disorders ought to target the sources rather than the sinks, identifying and studying network's global hubs and bridge symptoms is crucial (Goekoop & Goekoop, 2014).

Empirical studies showed some power for network approaches in delineating Psychopathology Webs, though transversally and only for certain disorders' symptoms. A seminal paper by Borsboom and Cramer (2013) exemplified the power of this approach by creating a network of symptoms for anxiety and depressive disorders. They found that depressive and anxiety disorders clustered similarly as *DSM* categories, together with a tremendous amount of correlations between them. Expanding the issue to 120 *DSM* symptoms, Boschloo et al. (2015) reported a syndromal cluster structure similar to *DSM* categorical diagnostic structure. At the same time, all diagnoses connected via symptoms paired to at least 3 other diagnoses. These symptom connections between syndroms might account for the comorbidity between diagnoses (provided the number of symptoms is sufficient to reach the threshold of diagnosis). Also, they identified strong connections between overlapping symptoms of different diagnoses, and between non-overlaping symptoms of different diagnoses. In line with a conception bridging healthy personality and pathology, similar network models were produced for personality (i.e. Personality Webs; Goekoop, Goekoop & Schulte, 2012). Hence, Psychopathology and Personality Webs satisfied so-called "small world" properties: a balance between local (e.g. *within* cluster) and global connectivity (i.e. *between* cluster).

Since Psychopathology and Personality Webs both exhibit "small world" architecture like other biological networks, they might help in connecting syndromes' phenomenological levels with other explanatory levels (Goekoop et al., 2012; Goekoop & Goekoop, 2014). Goekoop and Goekoop (2014) discussed the fascinating possibility that the architecture of networks at a phenomenological level (i.e. personality, syndromes) could be similar to those at different levels of explanation (e.g. genetic, neurophysiological, neural, pharmacotherapeutic, psychotherapeutic). Such corresponding structures would permit to draw connections between different levels, and delineate transdiagnostic aetiological pathways, and across different explanatory levels, from genes to behavior. Concretely, Goekoop and Goekoop (2014) interpreted the pathology network as representing different domains connecting a syndrome, a neural abnormality, a neurotransmission abnormality, implying a specific pharmacotherapeutic target (e.g. depression, affective regulation issues, serotoninergic abnormality and treatment). By bridging precisely phenomenological levels

(i.e. personality, symptoms) with other explanatory levels (including cognitives styles and endophenotypes), such an approach could help in understanding psychiatric nosological structure, its underlying aetiological structures (causal pathways), thereby providing new intervention targets.

Each of the psychometric, technical and application challenges of network models could improve our understanding of ASp-PSp relationships. A first challenge lays in the development and validation of comprehensive rating scales with all symptoms, as pleaded by Goekoop & Goekoop (2014). Obviously, there is a trade-off between exhaustivity and practicality (i.e. length) of such an instrument. In this regard, combining such a comprehensive instrument with an adaptive method could be very useful (Fonseca-Pedrero et al., 2013). With this method, only the necessary items would be asked to the participant, gradually selected as a function of participant's previous responses. After Goekoop and Goekoop (2014), we could expect that bridge and core symptoms items might play specific roles in this context. Indeed, bridge symptoms may be useful to detect comorbidities, and predict transitions. In contrast, core symptoms may further strengthen the evidence for a given syndrome. A second challenge is the integration between Psychopathology Web to Personality Web (Goekoop & Goekoop, 2014). Indeed, Goekoop et al. (2012) consider the Personality Web based on NEO-PI-R would offer cues about healthy and pathologic personality development. For instance, they consider that if hub traits fail to develop, the individual would suffer from a dysfunctioning personality, possible resulting in a PD. Concretely, traits such as Neuroticism put direct and indirect constraints onto the development of other personality clusters such as Agreeableness and Openness (notably via Extraversion, Conscientiousness and a possible sixth cluster). A third challenge is the application of network models to neurodevelopmental models, which would be very useful for ASp-PSp relationships. No network models we cited (Borsboom & Cramer, 2013; Boschloo et al., 2015; Goekoop & Goekoop, 2012) included autistic symptoms, so that their location and relationships with other symptoms can only be speculated.

We believe that applying network models to ASp and PSp might be profitable for understanding ASp-PSp relationships and aetiological pathways, crossing different explanation levels and time scales. For Borsboom (2017), applying network models to ASD and SSD is challenging, because these disorders are rare and slowly developing ones (i.e. including a temporal aspect). Yet, Borsboom (2017, p. 11) speculated that "in autism, a symptom such as avoiding eye contact, in the long run, [would] limit the ablity of a child to learn the ways of social interaction, leading to a symptoms like problems in maintaining relationships". Such an example illustrated how an early symptom at a certain developmental time (early) at a certain cognitive level (gaze processing abnormality) could interfere with learning and development (gaze/face processing) and

result in another symptom (social interaction deficits) at a certain developmental time (later) and at a certain cognitive level (complex social cognition).

Based on the present work, we could speculate that network models might account for the multiple types of relationships between ASp and PSp. Network models of ASp and PSp may reveal "shared" pathways (e.g. shared negative traits/symptoms, shared cerebral and neurotransmitter abnormalities), "opposite" pathways (e.g. opposite patterns of activations of certain brain areas realted to social cognition), and "distinct" pathways (e.g. distinct genes would cause distinct brain abnormalities or neurotransmitter abnormalities, and distinct neurocognitive endophenotypes). Importantly, network models might shape like forks, towards divergent (multifinal) or convergent (equifinal) outcomes (see Cicchetti & Rogosch, 2002). Different genetic backgrounds could converge to result in similar or equifinal outcomes between ASp and PSp (e.g. negative symptoms/traits). In contrast, similar genetic backgrounds could interact with other abnormalities and diverge resulting in different or multifinal outcomes between ASp and PSp. Applying such network models across several explanation levels, from genes to behaviour, might clarify ASp-PSp relationships. Concretely, this might provide evidence for "shared", "diametrical" and "distinct" aetiological pathways. Crucially, this approach and this reflexion could apply to any transdiagnostic research involving other disorders spectra (e.g. OCD, ADHD, bipolar).

6.6.9 SUMMARY

Both our results and the literature showed that no existing model can fully and accurately account for ASp-PSp relationships as observed by our results and the literature (Chisholm et al., 2015). This prompted a critical reflection about these models, so that we delineated an alternative model. This alternative model would have to provide an integrative account of shared, distinct and opposite relationships between sets of features of ASp and PSp. A key idea is that this model should focus on aetiological pathways pertaining to transdiagnostic clinical manifestations, with the aim of determining their relationships (shared, opposite relationships, or simply distinct). Obviously, such a research direction depends also on underlying constructs we used: personality traits, cognitive styles, endophenotypes, transdiagnostic multidimensional models, or the promising syndrome-based network model approach. Notwithstanding limitations, these concepts and their operationalizations feature promising future perspectives, such as accounting for protective factors, and better understanding of possibly shared endophenotype through transdiagnostic research. Further than ASp-PSp research, our results and their limitations drew attention to the global challenge of nosology, and the necessity of a transdiagnostic comparative research. Transdiagnostic approaches

might help in circumventing the limitations of both categorical and dimensional approaches, and in focusing on networks of observable variables from genes to behavior. Therefore, transdiagnostic approaches might revolutionize research on aetiological mechanisms of psychiatric disorders. beyond the issue of ASp-PSp relationships.

7 CONCLUSION

This work started with the paradox that autism and schizophrenia are related, yet separate disorders (Chapter 1). We then extended our questioning to the healthy personality phenotypes via the dimensional approach, and adopted a comparative strategy focusing on differences. Indeed, focusing on differences, notably using the diametrical model (Crespi & Badcock 2008), seemed to us a promising way to put into question the models focusing on ASp-PSp similarities. Also, we followed the program outlined in the diametrical model and tested social cognition functions in healthy population characterized by variations in autistic and schizotypic traits. Hence, we focused on the construct of Mentalism, representing the social cognition abilities, and searched for psychometric and behavioural biases in Mentalism.

For this to be done, our first goal was to validate the French personality questionnaires measuring autistic and schizotypy traits, and to examine their relationships (Chapters 2 and 3). We replicated the overlap in negative/anhedonic traits, and positive/attention to details traits among autistic and schizotypic traits (Ford & Crewther, 2014; Hurst, Nelson-Gray et al., 2007; Russell-Smith et al., 2011). Crucially, we also replicated the existence of an opposition between positive schizotypy traits and autistic traits (Dinsdale et al., 2013). We interpreted this opposition as a psychometric support for the diametrical model, or analogues (Brosnan et al., 2010; Crespi & Badcock, 2008). To put it shortly, ASp and PSp would oppose on a Mentalism continuum. Autistic mentalizing deficit traits would represent hypo-Mentalism, whereas positive schizotypy traits would represent hyper-Mentalism (Chapter 3; Crespi & Badcock, 2008). Yet, does this psychometric opposition reflect corresponding behavioural biases in social cognition tasks?

As a second goal, we measured face pareidolia-proneness and gaze processing, with the idea that diametrical behavioural and cognitive biases would reflect the psychometric Mentalism continuum (Chapters 4 and 5). Instead of accounting for pareidolia-proneness, our results reflect distinct phenotypes between ASp and PSp (Chapter 4). Positive schizotypy associated with configural deficits in processing face and face-like stimuli (i.e. cars' fronts), and/or a featural processing bias, as noted in previous studies on PSp (Batty et al., 2014; Butler et al., 2008; Chambon et al., 2006; Joshua & Rossell, 2009; Panton et al., 2016; Shin et al., 2008). In contrast, autistic mentalizing deficit traits associated with absence of configural processing bias, yet with an advantage in processing OBJ stimuli (Hedley et al., 2014; Weigelt et al., 2012), suggesting preference for OBJs, but not face(-like) stimuli, as reported in previous studies on ASp (Pallett et al., 2013; Wolf et al., 2008). Hence, Mentalistic traits revealed distinct cognitive phenotypes rather than diametrically opposite ones, in line with previous research separately in ASp and PSp.

Our gaze tasks showed spurious opposite relationships for centre GD, but, for sure, distinct ones for GC between Mentalistic traits (Chapter 5). GD tasks replicated the relative disadvantage in processing centre-directed gaze associated with ASp gaze under-sensitivity (Wallace et al., 2010). In contrast, positive schizotypy associated with better performances in processing centre-directed gaze, in line with previously reported gaze over-sensitivity in SSD (Hooker & Park, 2005). These results could have represented the diametrically opposite performances in social cognition, proposed by the diametrical model. Yet, they are spurious (ceiling effect, regression assumption violations), and cannot be used to unequivocally support the diametrical model. Instead of mirroring these results, GC liability was unrelated to positive schizotypy unlike reports with SSD (Langdon et al., 2006), yet significantly decreased as a function of autistic mentalizing deficits traits, as previously hinted (Alwall et al., 2010; Bayliss, Di Pellegrino et al., 2005). GC liability clearly showed distinct phenotypes between ASp and PSp, rather than diametrically opposite ones posited by the diametrical model. In line with the literature, lower GC liability associated with larger autistic mentalizing deficit scores, but only in men, suggesting a men-specific endophenotype and/or a psychometric gender-specific bias (Scheeren & Stauder, 2008; Tajmirriyahi et al., 2016). These results did not offer more support to the diametrical model.

Nevertheless, we did not conclude that the diametrical model was totally false: this model may hold true for certain cognitive levels and when using certain methods. Still, ASp-PSp relationships may depend on the explanatory level considered (e.g. neuroanatomic, cognitive), thereby casting doubt on a Mentalistic opposition extending throughout explanatory levels, from genes to behaviour, as proposed by the diametrical model. Indeed, at the psychometric level, we showed diametrical relationships, along shared ones between ASp and PSp in line with the literature (Chapter 3; but see distinct relationships in Ford & Crewther, 2014; Ford et al., 2017). In contrast, at a low cognitive/behavioural level of social perception, we found distinct gaze processing, and by inference face processing at social perception's low cognitive/behavioural level, in line with the literature and the possible influence of underlying endophenotypes (Chapters 4 and 5). Our psychometric results reflected on few evidence showing promising hints of opposition at higher social cognition levels, in line with the diametrical model (Chapter 6.4). In contrast, our behavioural results reflected on *a priori* distinct deficits between ASp and PSp at the lower level of endophenotype candidates (Chapter 6.6.6). Hence, along a shared liabilities model, the diametrical model may be necessary to explain ASp-PSp opposite relationships at higher cognitive and social cognition levels, whereas alternative models positing distinct or shared relationships might be necessary to account for ASp-PSp relationships at the lower endophenotypic level. Further research will have to test such a hypothesis, and provide evidence that this work failed to provide.

Although these results suggested some support for the diametrical model, they remain weak and questionable for the psychometric results, and, at best, spurious for the behavioural results. In this respect, we discussed the limitations pertaining to our psychometric instruments, behavioural tasks, our hypotheses, and the diametrical model itself (Chapter 6). As for solutions, we proposed to improve the self-report questionnaires we used, and to use them along other instruments measuring similar constructs. We also proposed to adjust our protocol to better account for pareidolia-proneness, for instance using eyetracking or neuroimaging. Alternatively, we noted that manipulating encoding duration and encoding-retrieval duration is promising to target the PSp encoding deficits (Butler et al., 2008), and the ASp memory deficits (Weigelt et al., 2012). As for gaze processing, we noted that gaze self-attribution is a promising protocol to oppose ASp and PSp biases (Hooker & Park, 2005; Matsuyoshi et al., 2014). GC liability protocol could be improved, notably by including stimuli that are non-social, depict schematic gaze, or are video clips. Finally, we also noted that the diametrical model was limited in several respects. Notably, the diametrical model showed difficulties accounting for the psychometric overlaps. Also, this model overly relied on hyper-Mentalism a new, controversial, and relevant construct, yet likely only for a subset of individuals with positive features. Yet, after examination, we observed that none of the current ASp-PSp relationship models (Chisholm et al., 2015) can account for the variety and the complexity of ASp-PSp relationships we observed and reviewed.

Therefore, we outlined an alternative model that could integrate shared, diametrical and distinct relationships between ASp and PSp (Chapter 6.6.2.). Crucially, such a model would rest onto the transdiagnostic examination of aetiological pathways of apparently similar features (e.g. negative symptoms), instead of the traditional disorder-specific search for deficits (Krueger & Eaton, 2015). Such investigations would consist in searching whether apparently similar features are caused by either shared, diametrical and/or distinct underlying mechanisms (e.g. face/gaze processing deficits). Such an alternative model would guide the search for candidate endophenotypes in psychiatric research, beyond ASp-PSp comparison.

Other related issues pertain to the significance of personality traits, cognitive styles, endophenotypes, and a multidimensional disorders model. Notably, we stressed that research would gain by considering personality profiles as reflecting specific disorder liability and aetiological pathway, instead of considering nonspecific total scores or discrete dimensions. We outlined the challenges associated with the use of cognitive styles, but also a possible opportunity to account for protective and adaptive mechanisms related to environmental variables (Chapter 6.6.4.). We did the same for endophenotypes, emphasizing the need for research on cognitive styles to be guided by the one about endophenotypes (Chapter 6.6.5.). Finally, we advocated the use of a multidimensional

framework (Chapter 6.6.6.) to represent psychiatric disorders as specific profiles made of nonspecific transdiagnostic dimensions (Chapter 6.6.7.). Combining this framework with healthy personality traits, such a multidimensional model would permit a transdiagnostic examination of the aetiological pathways, testing whether phenomenological similarities refer to underlying shared, diametrical or distinct mechanisms. This approach would help us understanding ASp-PSp relationships and relationships between other spectra featuring phenomenological and cognitive similarities. More provocatively, we proposed network models as an alternative, because of extensively discussed limitations of both categorical and dimensional models. These proposed models are relevant to ASp-PSp relationship issues as well as psychopathological models beyond (Chapter 6.6.8.).

Finally, this work did not bring groundbreaking answers to the question of ASp-PSp relationships. It offered, however, abundant opportunities to question and discuss more broadly research on the causal mechanisms underlying psychiatric disorders, and associated personality liabilities. We believe that the investigation of ASp-PSp relationships as well as their comparison offers interesting opportunities to think about research in such domains more generally. We also highlighted the merit of looking at the interactions between different research fields (e.g. autism vs. psychosis; personality vs. psychiatry), different methods (e.g. categorical vs. dimensional vs. network approaches), and different levels of explanations (e.g. genetic, neurobiological, neuroanatomical / neurofunctional, low cognitive / behavioural endophenotypic, higher cognitive / behavioural and phenomenological levels). As a result, we stress that a transdiagnostic comparative approach, that integrates different domains and explanation levels in an organized way (e.g. from genetics to psychometry), will permit a renewed understanding of clinical and healthy phenotypes, their aetiology and ultimately clarify the psychiatric disorders' classification system.

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9 APPENDICES

Table 27. Principal Component Analysis outcome accounting for the the subscale scores of the sO-LIFE and AQ-BC. The PCA was conducted using Promax rotations. Results are based on data from n=921 participants (from Sierro, Rossier, & Mohr, 2016 dataset).

	Promax Rotation	
	PCSF-BC	PCDF-BC
Explained Variance	28%	20%
UnEx	-.04	**.84**
CogDis	.36	**.60**
IntAn	**.66**	.05
ImpNon	-.02	**.73**
SocSki	**.82**	-.08
AttSwi	**.57**	.13
Comm	**.76**	.10
Ima	**.58**	-.29
AttDet	-.12	**.57**

Loadings < -.40 and > .40 are in bold.
PCSF-BC: PC score of Shared Features with AQ-BC; PCDF-BC: PC score of Diametrical Features with AQ-BC
Global: total sO-LIFE score; UnEx: Unusual Experiences; CogDis: Cognitive Disorganization; IntAn: Introvertive Anhedonia; ImpNon: Impulsive Nonconformity
SocSki: Social Skills deficits; AttSwi: Attention Switching deficits; Comm: Communication deficits; Ima: Imagination deficits; AttDet: Attention to Details

Table 28. Table comparing AQ dimension scores between adult ASD/BAP populations and other adult clinical or healthy comparison populations.

Study	AQ factor structure	Dimensions	Comparison
Baron-Cohen et al. (2001)	AQ-BC	Total	ASD > neurotypicals
Broadbent et al. (2013)	AQ-BC	Total	ASD > neurotypicals
Sonié et al., (2013)	AQ-BC	Total	ASD > neurotypicals
Wouters and Spek (2011)	AQ-BC	SocSki, AttSwi, AttDet, Comm, Ima	ASD > neurotypicals
Lau, Kelly et al. (2013)	other	Sociability, Resistance to change, Interests in patterns, Social cognition, Narrow focus	ASD > neurotypicals
Pisula et al. (2013)	AQ-BC	Total, Comm, SocSki, Ima, AttSwi, AttDet	ASD > neurotypicals
Bishop et al. (2004)	AQ-BC	Total, SocSki, Comm	BAP parents > non BAP parents
Wheelwright et al. (2010)	AQ-BC	SocSki, Comm, AttSwi, Ima, AttDet	BAP parents > non BAP parents
Ruta et al. (2011)	AQ-BC	Comm, Ima	BAP parents > non BAP parents
Kose et al. (2013)	AQ-BC	Comm, SocSki	BAP parents > non BAP parents
Lau, Gau et al. (2013)	other	Socialness, AttSwi	BAP fathers > non BAP fathers
Ketelaars et al. (2008)	AQ-BC	Comm	ASD > general inpatients
Murphy et al. (2011)	AQ-BC	Comm, SocSki	ASD detainees > detainees with various PDs/Mental disorders
Naito et al. (2010)	AQ-BC	Comm, SocSki	ASD > SSD
Spek and Wouters (2010)	AQ-BC	Comm, SocSki	ASD > SSD
Wouters and Spek (2011)	AQ-BC	SocSki, Comm, AttSwi	ASD > SSD
Wouters and Spek (2011)	AQ-BC	SocSki, AttSwi, Comm, Ima	SSD > neurotypicals
Hoekstra et al. (2008)	other	Social Interaction, AttDet	ASD > SAD, OCD, neurotypicals
Cath et al. (2008)	AQ-BC	AttSwi, Comm, Ima	ASD > SAD, OCD
Sizoo et al. (2009)	AQ-BC	Ima, SocSki, Comm, AttSwi	ASD > ADHD

Each dimension present under "Dimensions" column are significantly different in one as compared to the other population in the direction indicated by "Comparison". Dimensions are ordered by decreasing magnitude of the difference. BAP stands for Broader Autism Phenotype (parents of ASD children); SAD stands for Social Anxiety Disorder; OCD stands for Obsessive-Compulsive Disorder; ADHD stands for Attention Deficit and Hyperactivity Disorder.

Total: AQ-BC total score; SocSki: Social Skills deficits; AttDet: Attention to Details; Comm: Communication deficits; Ima: Imagination deficits; AttSwi: Attention Switching deficits

Table 29. Post-hoc (PH) power and a priori (AP) sample size n for a minimal power of 0.80 of significant relationships in multiple regression models F-test between personality predictors and behavioural variables of studies in Chapters 4 and 5.

Study	Type	DV	Pred.	N Pred.	df	n	f^2	Power
PS1	PH	PI Accura	PCDF-BC/PC	2	45	48	0.126	**.49**
PS1	AP	PI Accura	PCDF-BC/PC	2	76.46	**79.46**	0.126	.80
PS1	PH	Face Upr RT	UnEx	2	45	48	0.176	**.71**
PS1	AP	Face Upr RT	UnEx	2	54.70	**57.70**	0.176	.80
PS2	PH	OBJ IN V	PCDF-K	2	45	48	0.250	**.80**
PS2	AP	OBJ IN V	PCDF-K	2	43.70	**46.70**	0.250	.80
PS3	PH	PI Accura	PCDF-K	2	45	48	0.143	**.61**
PS3	AP	PI Accura	PCDF-K	2	67.53	**70.53**	0.143	.80
PS3	PH	PI Accura	ComMind	3	44	48	0.135	**.51**
PS3	AP	PI Accura	ComMind	3	80.73	**84.73**	0.135	.80
GD	PH	Accuracy	UnEx Ima	3	64	68	0.269	**.95**
GD	AP	Accuracy	UnEx Ima	3	40.63	**44.63**	0.269	.80
GC	PH	GC effect	Sex X PCDF-	2	62	68	0.122	**.69**
GC	AP	GC effect	Sex X PCDF-	2	79.08	**85.08**	0.122	.80
GC	PH	GC effect	ComMind	3	63	68	0.205	**.87**
GC	AP	GC effect	ComMind	3	53.21	**58.21**	0.205	.80
GC	PH	GC effect	ComMind x	3	60	68	0.099	**.51**
GC	AP	GC effect	ComMind x	3	110.57	**118.57**	0.099	.80

Study: PS1: Pareidolia-proneness Study 1; PS2: Pareidolia-proneness Study 2; PS3: Pareidolia-proneness Study 3; Type: power test type: AP: a priori; PH: post hoc; DV: dependent variable; Pred.: predictor(s); N pred.: Number of predictors(s); *df*: degrees of freedom; *n*: sample size; β: beta effect size

Post-hoc power (bolded) was computed using f^2 effect sizes of each regression model *F*-test, the effective sample sizes *n* of each regression model *F*-test, and a fixed significance level of .050.

A priori sample sizes *n* (bolded and underlined) were computed using f^2 effect sizes of each regression model *F*-test, a fixed power of .80, and a fixed significance level of .050.

For the pareidolia studies general models (Table 29), Post-hoc power was above .80 in 1/5 tests, and under .80 in 4/5 tests. Mean (±sd) Post-hoc power was 0.62(±0.13), ranging from 0.49 to 0.80. Based on these effect sizes, hypothetical future studies should aim at additional participants: there was on average -19.82(±15.63) missing subjects to achieve a power of .80, ranging from a minimum of -36.73 missing subjects to a maximum of 1.33 excess subjects. Provided all *F*-tests effects measured are true, a minimal sample of n≥85 should have been collected in each pareidolia-proneness study to be able to detect them with appropriate power (≥.80).

For the only significant GD general model (Table 29), Post-hoc power was 0.95, well above 0.80. Theoretically 44.63 participants would have been sufficient to detect this effect at a minimally sufficient power (≥.80).

For the GC general models (Table 29), Post-hoc power was above .80 in 1/3 tests and below in 2/3 tests. Mean(±sd) Post-hoc power was 0.69(±0.18), ranging from 0.51 till 0.87. Based on these effect sizes, hypothetical future studies should aim at additional participants: there was on average -19.53 (±30.22) missing participants to achieve a power of .80, ranging from a minimum of -50.57 missing participants to a maximum of 9.79 participants in excess. The lack of power was only present in regressions with regression models featuring interactions with Sex. Only for models featuring the interactions of personality variables with Sex. Provided all *F*-tests effects measured are true, a minimal sample of *n*≥119 participants should have been collected in GC study to be able to detect all Sex × personality interaction effects with appropriate power (≥.80).

Table 30. Post-hoc (PH) power and a priori (AP) sample size n for a minimal power of 0.80 of significant relationships in regression t-tests on slopes between personality predictors and behavioural variables of studies in Chapters 4 and 5.

Study	Type	DV	Pred.	N pred.	*df*	*n*	β	Power
PS1	PH	PI Accuracy	PCDF-BC/ PCSF-BC	2	45	48	-.39	**.78**
PS1	AP	PI Accuracy	PCDF-BC/ PCSF-BC	2	46.73	**49.73**	-.39	.80
PS1	PH	Face Upr RT	UnEx	2	45	48	.41	**.82**
PS1	AP	Face Upr RT	UnEx	2	42.48	**45.48**	.41	.80
PS2	PH	OBJ IN V Accuracy	PCDF-K	2	45	48	0.42	**.85**
PS2	AP	OBJ IN V Accuracy	PCDF-K	2	39.78	**42.78**	.42	.80
PS3	PH	PI Accuracy	PCDF-K	2	45	48	.31	**.58**
PS3	AP	PI Accuracy	PCDF-K	2	74.98	**77.98**	.31	.80
PS3	PH	PI Accuracy	ComMind	3	44	48	-.31	**.56**
PS3	AP	PI Accuracy	ComMind	3	75.53	**79.53**	-.31	.80
GD	PH	Accuracy center Gaze	UnEx	3	64	68	.31	**.72**
GD	AP	Accuracy center Gaze	UnEx	3	77.49	**81.49**	.31	.80
GD	PH	Accuracy center Gaze	Ima	3	64	68	-.35	**.81**
GD	AP	Accuracy center Gaze	Ima	3	61.67	**65.67**	-.35	.80
GC	PH	GC effect	ComMind	4	63	68	-.37	**.86**
GC	AP	GC effect	ComMind	4	53.19	**58.19**	-.37	.80
GC	PH	GC effect	ComMind (men)	3	32	36	-.50	**.86**
GC	AP	GC effect	ComMind (men)	3	27.25	**31.25**	-.50	.80
GC	PH	GC effect	Ima (men)	3	62	36	-.32	**.45**
GC	AP	GC effect	Ima (men)	3	72.51	**76.51**	-.32	.80

Study: PS1: Pareidolia-proneness Study 1; PS2: Pareidolia-proneness Study 2; PS3: Pareidolia-proneness Study 3; Type: power test type: AP: a priori; PH: post hoc; DV: dependent variable; Pred.: predictor(s); N pred.: Number of predictors(s); *df*: degrees of freedom; *n*: sample size; β: beta effect size

Post-hoc power (bolded) was computed using regressions' β effect sizes of each *t*-test on slopes, the effect sample sizes *n* of each test, and a fixed significance level of .050.

A priori sample sizes *n* (bolded and underlined) were computed using regressions' β effect sizes of each *t*-test on slopes, a fixed power of .80, and a fixed significance level of .050.

For the pareidolia studies specific regression slopes (Table 30), Post-hoc power was above .80 in 2/3 tests, and under .80 in 3/5 tests. Mean (±sd) Post-hoc power was 0.72(±0.14), ranging from 0.56 to 0.85. Based on these β effect sizes, hypothetical future studies should aim at additional participants: there was on average -11.10(±18.12) missing subjects to achieve a power of .80, ranging from a minimum of -33.53 missing subjects to a maximum of 5.22 excess subjects. Provided all pareidolia-proneness Studies *t*-tests effects measured are true, a minimal sample of $n \geq 80$ participants should have been collected to be able to detect them with appropriate power (≥.80).

For the GD study regression slopes (Table 30), post power was above .80 in ½ tests, and below .80 in ½ tests. Mean(±sd) Post-hoc power was 0.76(±0.06), ranging from 0.72 to 0.81. Based on these β effect sizes, hypothetical future studies should aim at additional participants: there was on average -5.58(±11.19) missing subjects to achieve a power of .80, ranging from a minimum of -13.49 missing subject to a maximum of 2.33 subjects in excess. Provided all GD *t*-tests effects measured are true, a minimal sample of $n \geq 81$ participants should have been collected to be able to detect them with appropriate power (≥.80).

For the GC regression slopes (Table 30), Post-hoc power was above .80 in 2/3 tests and below in 1/3 tests. Mean(±sd) Post-hoc power was 0.72(±0.24), ranging from 0.45 till 0.86. Based on these β effect sizes, hypothetical future studies should aime at additional participants: there was on mean -8.65(±27.71) missing participants to achieve a power of .80, ranging from a minimum of -40.51 missing participants to a maximum of 9.81 participants in excess. The lack of power was only present in men-only regression with Ima as a predictor. Provided all GC *t*-tests effects measured are true, a minimal sample of $n \geq 77$ participants (men) should have been collected in GC study to be able to detect Ima effects with appropriate power (≥.80).