Behaviour is the response of an animal to stimuli in its internal or external environment, ranging from simple reflexive behaviours to those that are more complex and goal directed, such as foraging, finding a mate, or engaging in aggressive interactions. However, even reflexive behaviours can be modified by experience. For example, in the zebrafish, *Danio rerio*, the decision to escape or swim is influenced by social status, achieved through a shift in the excitability of neural circuits (Miller et al. 2017). Therefore, a behavioural act requires an individual not only to process sensory information and respond with motor output, but also to integrate its current internal motivational state and memory of past experiences (Bendesky and Bargmann 2011; O’Connell and Hofmann 2011). As such, the genes that affect behaviour can act to influence many different layers of the nervous system, ranging from sensory perception to the connectivity and modulation of neural circuits (Marder 2012; McGrath 2013). This feature of behaviour, the ability to be modified at many different levels, may contribute to the high evolvability of behavioural traits (Blomberg et al. 2003).

To gain an understanding of how animal behaviour evolves requires an integrative approach that examines how behavioural traits are inherited and also characterizes the genetic variants underlying behaviour and their specific effects on neural processing. In this chapter, we present a current understanding of the relationship between genes (of large effect) and behaviour. We first outline how most phenotypic traits, including behaviour, are controlled by many variants of small effect (see also Chapters 1 and 2). We then describe several well-studied examples of single genes that mediate behaviour, as well as ‘supergenes’ that can control behavioural divergence within species. Next, we discuss how certain classes of genes may be more likely to influence the evolution of behaviour. Finally, we consider whether the genetic architecture of behavioural traits is unique in relation to other phenotypic traits. We conclude the chapter by suggesting that an integrative approach to the study of genes and behaviour will lend the most insight into the forces underlying behavioural and genetic diversity.
5.1 Genetic Architecture of Phenotypic Traits

Evolutionary change requires variation in phenotypic traits to have a genetic component. The proportion of variance of a phenotypic trait in a population that is due to genetic factors can be described by the degree of heritability (Visscher et al. 2008). Heritability has been best quantified in humans. A meta-analysis of twin studies identified no traits with heritability estimates of zero, suggesting that all traits are heritable (Polderman et al. 2015). The narrow-sense heritability for one of the most well-studied traits, human height, is around 80%, meaning that 80% of the variance is due to additive genetic factors (Silventoinen et al. 2003). Many behavioural human diseases are also highly heritable, with heritability for schizophrenia and autism spectrum disorder at 80% and 50%, respectively (Purcell et al. 2009a; Gaugler et al. 2014). There is great interest in identifying the causal genetic variants that underlie the heritable variation in phenotypic traits; finding these variants has important implications for identifying disease risk, developing drug treatments, improving efficiency in agriculture, and appreciating the effect of selection on maintaining genetic and phenotypic diversity (Robinson et al. 2014).

Quantitative trait locus (QTL) mapping in inbred laboratory model organisms was the first approach commonly used to examine the genetic architecture of traits. The results from early QTL studies, often biased by low statistical power, suggested that a few large-effect QTL could explain a high proportion of trait variation, fuelling hope that large-effect variants underlying disease could be identified in humans (Flint and Mackay 2009). However, subsequent fine-mapping studies revealed that multiple QTLs, often with opposite effects, were contained within the originally identified single QTLs (Flint and Mackay 2009). Furthermore, even a well-defined QTL contains some 300–500 genes (Mackay 2004; Mott and Flint 2008). The picture that eventually emerged suggests that allelic effects follow an exponential distribution, with few loci of large effect and many loci of small effect (Orr 1998; Flint and Mackay 2009; Rockman 2012).

A genetic architecture with many variants of small effect has also been found using the more recently developed tool of genome-wide association studies (GWAS) (Manolio et al. 2009). Using outbred populations, this approach identifies single nucleotide polymorphisms associated with phenotypic traits and overcomes many of the challenges associated with QTL analyses that typically only identify regions containing tens of hundreds of genes. GWAS with large sample sizes (250 000 individuals) have shown that the genetic architecture for human height is characterized by a very large number of causal variants (Wood et al. 2014). The effect sizes of the thousands of loci implicated in human height are typically small, with any given allele typically accounting for less than 1 mm difference in height (Wood et al. 2014). Behavioural human diseases appear to have a similar genetic architecture with many genes of small effect. For example, both schizophrenia and autism spectrum disorder are associated with a large number of common genetic variants (Purcell et al. 2009a; Gaugler et al. 2014).

Somewhat surprisingly, even very large-scale GWAS still often explain only a limited proportion of the heritability of a given trait. This has led to the suggestion that non-additive genetic variance contributes to the ‘missing heritability’ (Eichler et al. 2010; Zuk et al. 2012a). However, most evidence suggests that the missing heritability probably lies in unidentified variants of very small effect which studies lack the power to detect (Rockman 2012). The proportion of accounted-for heritability has
been shown to increase with ever larger sample sizes and inclusion of more single nucleotide polymorphisms (SNPs). For example, the common variants identified across independent studies of human height now account for 60% of the heritability (Wood et al. 2014). Similarly, a meta-analysis based on over 14.5 million pairs of human twins found that for two-thirds of all traits, twin resemblance fit a simple model of additive genetic variance (Polderman et al. 2015). Deviation from the model for the remaining one-third of traits apparently stems from the effect of shared environmental factors, rather than a prevalence of non-additive genetic variance (Polderman et al. 2015). It is also possible that trait heritability is overestimated due to the presence of epistasis, so the concern over the problem of ‘missing heritability’ may have been overstated (Zuk et al. 2012b). Unfortunately, the statistical power to detect non-additive genetic effects via genome-wide scans is very low given the nearly infinite number of models that can be fit. On smaller scales, the importance of non-additive genetic effects is well documented (Greenspan 2001; Meffert et al. 2002). For example, gene knockout studies have shown that the genetic background influences the resulting phenotype (Holmes et al. 2003; Dowell et al. 2010).

While it appears that most phenotypic traits are characterized by a genetic architecture composed of many variants of small effect acting additively, as described above, there are also many cases where traits are under the control of variants of large effect. For example, two variants with large effects on human personality are due to inversions (Giglio et al. 2001; Stefansson et al. 2005; Huddleston and Eichler 2016). The 17q21.31 inversion in humans contains several neurological-related genes, including microtubule-associated protein tau (MAPT) and corticotropin-releasing hormone receptor 1 (CRHR1) (Stefansson et al. 2005). Two haplotypes exist within human populations, H1 (direct) and H2 (inverted), and the two do not recombine over 1.5 Mb (Steinberg et al. 2012). A historical double recombination event may have occurred between the two haplotypes (Steinberg et al. 2012). Eight structural subtypes, five in the H1 lineage and three in the H2 lineage, have also been identified as a result of duplications and complex rearrangements within the inverted region (Steinberg et al. 2012). The primary H1 haplotype has been implicated in many neurodegenerative diseases (de Jong et al. 2012; Puig et al. 2015). One subtype of the H2 haplotype, H2D, increases the risk of microdeletion syndrome and has greatly increased in frequency in individuals of European descent, mostly likely because women carrying this subtype tend to have more children (Stefansson et al. 2005). It has been suggested that inversions and other structural variants may be an important and unappreciated cause underlying variation in human phenotypes (Huddleston and Eichler 2016).

Below, we discuss further examples of single genes and large non-recombining chromosomal regions with large effects on behavioural phenotypes.

5.2 Effects of Single Genes on Behaviour

Most genes with a large effect on behavioural phenotypes have been identified through mutagenesis experiments and association mapping in classic model laboratory organisms (Greenspan 2009). Another successful approach has been to conduct interspecific QTL mapping, which involves crosses between closely related species that differ in characteristic behavioural phenotypes (Schielzeth and Husby 2014). While powerful, an
issue with this method is that effect size is often overestimated when traits are mapped in two related species, probably as a by-product of differences in the structure of the mapping populations (Flint et al. 2005). QTL mapping in *Peromyscus* mice revealed that a simple genetic architecture may underlie tunnel burrowing behaviour (Weber et al. 2013). Oldfield mice, *Peromyscus polionotus*, build complex burrows with long entrances and escape tunnels. In contrast, the sister species, the deer mouse, *Peromyscus maniculatus*, build simple burrows with no escape tunnel. Genetic crosses between the two species resulted in offspring that build tunnels similar to oldfield mice, suggesting that the alleles that affect burrowing behaviour segregate in a dominant manner (Weber et al. 2013). QTL mapping on a recombinant backcross generation revealed that three additive genetic variants (QTLs) associated with the length of the entrance tunnel explain more than half of the genetic variation for the trait. A single variant was found to be associated with whether or not an individual builds an escape tunnel (Weber et al. 2013).

The same approach can be used on within-species crosses when there is strong variation among populations. For example, two independent studies using QTL mapping have examined the genetic architecture associated with schooling behaviour in fishes. By crossing strongly schooling marine and weakly schooling benthic populations of the threespine stickleback, *Gasterosteus aculeatus*, a genetic variant associated with schooling position was identified (Greenwood et al. 2013). Interestingly, this variant is also associated with lateral line anatomy, a peripheral neurosensory system important to positioning during social group formation in fishes (Greenwood et al. 2013). Specifically, the identified region contains genes that underlie variation in the number and pattern of neuromasts, as well as other candidate genes implicated in lateral line development or social behaviour. Similarly, in the Mexican tetra, *Astyanax mexicanus*, crosses between a sighted surface-dwelling form that schools and a blind cave-dwelling form that does not revealed that the genetic variants related to loss of schooling behaviour are, in part, associated with loss of vision, rather than changes in lateral line anatomy, as was found for benthic stickleback (Kowalko et al. 2013).

While QTL mapping studies have uncovered many genetic variants associated with behaviour, the causal gene(s) and functional changes in DNA sequence that contribute to behavioural variation have only rarely been identified. One exception comes from a study of parental care in *Peromyscus* mice (Bendesky et al. 2017). A large-effect QTL associated with nest building, a component of parental care, was identified by conducting an interspecies cross between two *Peromyscus* species that differ in parental care behaviour. By examining expression differences in the hypothalamus for the approximately 500 genes identified within the QTL, the neuromodulator arginine vasopressin (AVP) was identified as a main candidate. Increased expression of AVP was associated with less nest building. Pharmacology and chemogenetic experiments showed that vasopressin neurons within the hypothalamus are critical to parental nest-building behaviour, suggesting that differences in this gene contribute to the evolution of parental care in mice (Bendesky et al. 2017).

A comparative approach that examines variation in RNA or protein expression across species that differ in behavioural phenotypes is another way in which specific large-effect genes have been identified. For example, variation in the distribution of AVP 1a receptors in the male brain have been associated with differences in pair-bonding behaviour between vole species (Insel et al. 1994). This gene was originally examined as a candidate because central administration of vasopressin was demonstrated to
have wide-ranging effects on reproductive and parental care behaviours across species (Winslow et al. 1993). Findings such as these suggested that there may be many genes underlying behaviour with evolutionarily conserved functions, prompting the idea of a ‘genetic toolkit’ for behaviour, in which the same gene is either conserved or repeatedly co-opted in the evolution of shared behavioural phenotypes (Fitzpatrick et al. 2005; Toth et al. 2007; Rittschof and Robinson 2016). Recent comparative genomics approaches, however, have largely failed to identify new single genetic variants associated with behaviour across taxa (Rittschof et al. 2014; Kapheim et al. 2015). Given the large number of loci found which contribute to phenotypic variation, as well as the general conservation of physiological processes, there remain relatively few examples of the same genes contributing to shared behaviours in different species (Flint and Mackay 2009). Nevertheless, there are several striking examples of single genes with large effects on behaviour both between and within species; examples are described below (see also Figure 5.1).

5.2.1 The Foraging Gene and Food-Search Behaviour

The foraging (for) gene, which underlies a naturally occurring polymorphism in the food-searching strategy of Drosophila melanogaster larvae and adults, was one of the first large-effect behavioural genes to be identified (de Belle et al. 1989). The gene for encodes for a cyclical guanosine monophosphate (cGMP)-dependent protein kinase G (PKG) and has two naturally occurring variants (Osborne et al. 1997). The rover phenotype moves further distances in search of food compared to the sitter phenotype. Rover individuals have higher PKG activity and higher for gene expression relative to sitters.

![Figure 5.1](image-url)  
*Figure 5.1* Examples of single genes with large effects on behavioural phenotypes. Information listed for each gene includes the product which it encodes, its effect on behaviour, and the species in which it has been well studied. GPCR, G-protein coupled receptor; PKG, cGMP-dependent protein kinase; TF, transcription factor.
(Osborne et al. 1997). The for gene also has pleiotropic effects on other food-related traits, including metabolism and insulin signalling (Kent et al. 2009).

The for ortholog also affects food-searching strategies in other invertebrates. In the honey bee, Apis mellifera, gene expression of for increases in association with the age-related transition from in-hive activities to foraging behaviour (Ben-Shahar et al. 2002). The opposite pattern appears to be true for ants, whereby foraging behaviour is associated with lower levels of for gene expression (Ingram et al. 2005; Lucas et al. 2015), although the relationship between age, foraging behaviour, and for activity may be complex (Oettler et al. 2015). The specific tissues, cells, and gene networks within which for exerts its behavioural effects remain to be elucidated (Allen et al. 2017).

5.2.2 Arginine Vasopressin Receptor and Pair-Bonding Behaviour

The nonapeptides oxytocin and AVP have wide-ranging effects on social behaviour across species (Goodson 2013). They function as hormones in the periphery and as neuromodulators in the central nervous system. In the brain, differences in receptor expression, ligand binding, and microsatellite length have been shown to predict differences in pair bonding, social flocking, parental care, and various other social behaviours (Goodson 2013). The vasopressin 1a receptor (V1aR, encoded by avpr1a) has been associated with pair bonding in several species, including humans (Walum et al. 2008), but has been particularly well studied in the socially monogamous prairie vole Microtus ochrogaster (Yount et al. 2011). V1aR exhibits high intraspecific variation in this species. The effects of V1aR on behaviour depend on the brain region/neural circuit in which it is acting. For example, administration of a V1aR antagonist in the lateral septum or ventral pallidum, regions involved in the reward circuit, prevents partner preference formation in males (Lim and Young 2004). Avpr1a expression in a spatial memory circuit, but not in the lateral septum or ventral pallidum, is associated with male sexual fidelity (Ophir et al. 2008).

While prairie voles are socially monogamous, nearly one-fourth of offspring are sired by males that engage in extra-pair fertilizations. These males have larger home ranges and more frequently intrude in other territories compared to the majority of males that adopt a ‘resident’ strategy (Okhovat et al. 2015). Interestingly, levels of V1aR in spatial memory-related brain regions are associated with space use and site fidelity. Also in these brain regions, single nucleotide polymorphisms in avpr1a were found to predict individual differences in V1aR abundance. These genetic markers appear to be under balancing selection, reflecting the fitness trade-offs associated with either closely maintaining a pair bond or engaging in extra-pair mating (Okhovat et al. 2015).

5.2.3 Neuropeptide Y Homolog, Sensory Neurons, and Social Feeding Behaviour

The nematode Caenorhabditis elegans typically lives within decaying fruit where oxygen levels are low due to microbial respiration (Laurent et al. 2015). If an individual detects an increase in oxygen levels, suggesting that it is approaching the surface, it exhibits avoidance behaviour and reverses direction (McGrath et al. 2009). Failure to find an environment with lower oxygen leads to an aroused state with a suite of
related behaviours, including forming social aggregations on food (Busch et al. 2012; Laurent et al. 2015). A laboratory-cultivated strain, N2, shows only a weak response to increased oxygen and maintains solitary feeding habits in the laboratory (de Bono and Bargmann 1998; Gray et al. 2004). During the early phases of domestication, this strain adapted to the laboratory environment and acquired a single nucleotide substitution in the homolog of the neuropeptide Y gene \( npr-1 \) (McGrath et al. 2009). The high activity version of the allele present in the N2 strain decreases aversion to oxygen levels when consuming bacterial food, leading to modification of aggregation behaviour and differences in adult body size, fecundity, and physiology (Gray et al. 2004; Milward et al. 2011; Andersen et al. 2014). The effect of \( npr-1 \) on these behaviours has been linked to a single pair of inter/motor neurons, called RMG neurons (Macosko et al. 2009). Multiple distributed sensory inputs, including pheromone and oxygen detection, are co-ordinated through gap junctions with the common target neurons (Jang et al. 2017). NPR-1 inhibits RMG activity in the solitary N2 strain, which serves to uncouple the circuit while maintaining the function of the input sensory neurons (Macosko et al. 2009; Bargmann and Marder 2013). Similar neural circuits linking variation in neuropeptide Y homologs with social feeding behaviour may occur in other species, such as \( D. \ melanogaster \) (Wu et al. 2003).

Foraging strategies in \( C. \ elegans \) are also altered by conspecific pheromones. Heritable variation in pheromone sensitivity is linked to a G-protein coupled pheromone receptor, \( srx-43 \), that acts on sensory neurons to suppress exploratory foraging (Greene et al. 2016). The genomic region associated with \( srx-43 \) is under balancing selection as the two different haplotypes confer bidirectional effects on fitness dependent on food distribution and pheromone detection via \( srx-43 \) (Greene et al. 2016).

### 5.3 Effects of Supergenes on Behaviour

Phenotypic associations between multiple traits among individuals within populations occur commonly across taxa and across traits, including behaviour (Saltz et al. 2017). For example, aggression is correlated with colour variation in many species (Ducrest et al. 2008). In the African cichlid fish, \( Astatotilapia burtoni \), yellow male morphs are more aggressive than their blue counterparts (Dijkstra et al. 2017). When such trait correlations are adaptive, recombination can be disruptive and can impose a cost on fitness. This cost is avoided by a genetic architecture that preserves favourable combinations of alleles (Darlington and Mather 1949; Dobzhansky 1970; Thompson and Jiggins 2014). The maintenance of correlated traits can be achieved by genomic rearrangements that are inherited as a single locus. Such solutions have evolved repeatedly and underlie, for example, the evolution of sex chromosomes (Charlesworth 1996).

Supergenes are defined as multiple tightly linked loci that each affect discrete developmental or behavioural phenotypes (Schwander et al. 2014; Thompson and Jiggins 2014). Butterfly mimicry is a classic example of a supergene maintaining a balanced polymorphism, whereby multiple morphs in the same species mimic several different toxic species, functioning to reduce predation (Joron et al. 2011). Any recombination of traits that would reduce phenotype matching would have negative effects on fitness. Recently, supergenes have also been found to maintain several behavioural polymorphisms, described below (Figure 5.2).
5.3.1 Social Organization in Ants

Social organization, defined as the pattern of relationships between individuals within a social group, including the way in which reproduction is partitioned, can vary even within species or populations (Ross and Keller 1995). This phenomenon has been well studied in the fire ant, *Solenopsis invicta*, where there is variation in the number of reproductive queens per colony. *S. invicta* is polymorphic in queen number, as well as in a suite of related traits (Keller 1993). Colony acceptance of multiple queens is directly linked to allelic variation for the gene *Gp-9* (B and b alleles), which encodes an odorant binding protein (Ross and Keller 1998). Colonies in which all workers are homozygous at this locus (BB) will only accept a single BB queen. In contrast, colonies in which at least 10% of workers are heterozygous (Bb) will accept multiple, but only heterozygous queens (Ross and Keller 2002; Gotzek and Ross 2008). It was later shown that *Gp-9* is in fact part of a large non-recombining supergene of approximately 13 Mb with an estimated 616 genes in tight linkage (Wang et al. 2013). Interestingly, the Alpine silver ant, *Formica selysi*, shows a similar polymorphism in the number of queens per colony. This too was found to be under the control of a non-recombining supergene, although the specific location and content of the supergene differ from that of *S. invicta* (Purcell et al. 2014).

5.3.2 Alternative Mating Tactics in Birds

The Eurasian sandpiper, *Philomachus pugnax*, is a lek-breeding wading bird with three alternative male morphs. Independent males, the most common morph, defend territories and court females. Satellite males are non-territorial, but co-display and steal matings when independents are distracted. The third morph is the rare female-mimicking sneaker male (called a faeder) (Jukema and Piersma 2006). The three male morphs have been suggested to be under the control of a single Mendelian locus with three alleles (Lank et al. 1995). This was revealed to be a supergene consisting of an estimated 125 genes contained within a 4.5 Mb inversion (Küpper et al. 2015; Lamichhaney et al. 2016). The satellite and faeder morph alleles are dominant to the ancestral independent morph sequence (Lamichhaney et al. 2016). Homozygosity for the inversion is lethal, and heterozygosity reduces survival. Satellite and faeder males have larger testes, suggesting
5.5 Are Behavioural Traits Unique?

Phenotypic traits for which the genetic architecture has been well characterized are often morphological or, in the case of humans, disease related. The extent to which that a higher reproductive success may offset the costs of carrying the inversion (Küpper et al. 2015).

In the white-throated sparrow, *Zonotrichia albicollis*, a supergene controls two alternative morphs in both males and females that differ in plumage colour and social behaviour (Tuttle et al. 2016). Tan morphs are monogamous, while white morphs are promiscuous and invest less in parental care (Tuttle 2003). The supergene is a large inversion over 100 Mb and contains an estimated 1137 genes. The inversion contains several genes that are well known for their role in the neural control of social behaviour and regulation of aggression, including serotonin and oestrogen receptors, as well as vasoactive intestinal peptide (Tuttle et al. 2016). White and tan morphs show strong disassortative mating – there are negative fitness effects for the rare cases of assortative mating. Interestingly, the white morph allele may be degrading, and for genes within the inversion, gene expression is lower compared to the tan morph, suggesting the white allele is similar to a neo-sex chromosome (Tuttle et al. 2016).

5.4 Evolvability of Behaviour-Associated Genes

Certain classes of genes may be more likely to contribute to the evolution of behaviour. Sensory genes, for example, are among the fastest evolving families of genes (McGrath 2013). A genetic change to a sensory receptor provides a simple path to modify a behaviour by changing the perception of a stimulus without negative effects on other aspects of the phenotype (Bendesky and Bargmann 2011). In ants, communication is mainly through pheromone signalling – a large and novel clade of odorant receptor genes that allow pheromone detection evolved in the ancestor of all ants, probably affecting rates of speciation (McKenzie et al. 2016). The visual system, too, is a prime target. For example, changes in visual sensitivity due to genetic modifications of opsins was a major driver of the rapid speciation of African cichlid fishes, in part due to the effects on mate preference behaviour (Kocher 2004; Terai et al. 2006).

Neuromodulators also appear to be highly evolutionarily labile – they can act at a distance from the target cell and are not always essential for neurotransmission (Bendesky and Bargmann 2011; Marder 2012). Interestingly, in a meta-analysis of vertebrates, the sites of ligand production in the brain were found to be less conserved than the spatial distribution of their receptors (O’Connell and Hofmann 2012). It may be that only small developmental changes are necessary to shift the sites of ligand production (Marín and Rubenstein 2003; O’Connell and Hofmann 2012). While the spatial distribution of receptors was found to be highly conserved across taxa, the density of receptors in specific brain regions is well known to influence behaviour. For example, differences in the density of oxytocin, vasopressin, and dopamine receptors may underlie many of the behavioural differences between monogamous prairie voles and promiscuous montane and meadow voles (Smeltzer et al. 2006).
the genetic architecture underlying behavioural traits is unique is not well understood. Studies of outbred mice and rats suggest that the effect sizes for individual QTL may be lower for behavioural compared to physiological traits (Rat Genome Sequencing and Mapping Consortium et al. 2013; Parker et al. 2016). In addition, behavioural phenotypes often have lower heritability estimates compared to morphological and physiological traits (Roff and Mousseau 1987; Meffert et al. 2002; Parker et al. 2016). These low estimates could arise, in part, because measuring behavioural traits is challenging and their repeatability is often not examined (Croston et al. 2015; Greives et al. 2017). Experimental noise in behaviour assays can arise from the effects of age, nutrition, and stress, as well as from abiotic sources, such as temperature (Boake 1994; Meffert et al. 2002). In addition, behavioural traits are often complex, interrelated, and labile across time and development, so it is difficult to dissect behavioural phenotypes into quantifiable components.

Behaviours that involve a social component create an additional level of complexity to studying the genetic architecture of behaviour due to the presence of indirect genetic effects (IGEs), which describe how the phenotype of a focal individual can be influenced by the genes expressed by its interacting partners (Moore et al. 1997; Schneider et al. 2016) (reviewed in Chapter 4). These IGEs make the social environment itself heritable and thus also open to the effects of selection (Wolf et al. 1998). IGEs can have additive genetic effects, as well as non-additive effects in the form of epistasis (Wolf 2000). Importantly, because selection can act on traits in the absence of additive genetic variance, these effects can obscure heritability estimates and alter evolutionary trajectories (Meffert 1995; Wolf et al. 1998; Meffert et al. 2002; García-González and Simons 2007). IGEs are prevalent in species with parental care, whereby the phenotype of the offspring depends on the genotype of the parent, often of the mother, beyond the contribution of direct genetic inheritance (i.e. maternal effects) (Mousseau et al. 2009; McAdam et al. 2014) (see Chapter 7). These effects also play a large role in social insect colonies, whereby the genotype of nestmates affects individual and colony-level phenotypes (Pankiw et al. 2002; Linksvayer and Wade 2005; Linksvayer 2006). For example, in the fire ant, *S. invicta*, colonies switch their social organization and a suite of related behaviours when the proportion of colony members with a certain genotype passes above a critical threshold (Ross and Keller 2002) (see below for more detail). IGEs have also been found to affect a wide range of other behavioural phenotypes across species, including courtship (Petfield et al. 2005), aggression (Wilson et al. 2009), mate choice (Bailey and Zuk 2012) (see Chapter 6), and antipredator behaviour (Bleakley and Brodie 2009). Given that social interactions among animals are nearly ubiquitous, the role of IGEs in affecting trait evolution should be carefully considered. These may disproportionately affect the genetic architecture of behavioural traits, in particular.

Interestingly, most of the genes found to have a large effect on behavioural phenotypes come from crosses between different species or populations. These larger effects may be a by-product of differences in the structure of the mapping populations (Flint et al. 2005). Most QTL studies of behaviour conducted within single populations have revealed a complex genetic architecture, similar to that found for other phenotypic traits (Bendesky and Bargmann 2011). Indeed, it appears that variation in behaviour, like other phenotypic traits, is usually modulated by many common variants of small effect (Valldar et al. 2006; Flint and Mackay 2009). For example, studies on the behaviour of inbred
strains of mice and rats revealed a large number of genetic variants of small effect, as well as the presence of complex non-additive genetic effects (Flint 2003).

### 5.6 Conclusion

Animal behaviour is often complex and is affected by genes, experiences, and the environment. In addition, many behavioural phenotypes show adaptive correlations with other phenotypic traits. These beneficial associations can be maintained through pleiotropy or linkage, and supergenes have properties of both (Saltz et al. 2017). Given the frequency of balanced polymorphisms that involve behaviour, it may be that supergenes are a common part of the genetic architecture underlying polymorphic behavioural phenotypes. The extent to which the genetic architecture of behavioural traits is unique, due to either the occurrence of supergenes, the presence of indirect genetic effects, or the way in which the nervous system responds to selection, remains to be determined. Future behavioural studies that are able to draw a link between heritability, fitness, genes, and neural circuits will be crucial to gain a more complete understanding of the genetic basis of behaviour.

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### References


receptors in chemical communication. *Proceedings of the National Academy of Sciences USA* 113 (49): 14091–14096.


References


