Received: 10 November 2021 | Revised: 17 February 2022 | Accepted: 27 February 2022

DOI: 10.1002/ajp.23374

RESEARCH ARTICLE

AMERICAN JOURNAL OF PRIMATOLOGY WILEY

The relationship of maternal rank, *5-HTTLPR* genotype, and *MAOA-LPR* genotype to temperament in infant rhesus monkeys (*Macaca mulatta*)

John P. Capitanio[1,2] | Nicolas Sommet[3] | Laura Del Rosso[1]

[1]Neuroscience and Behavior Unit, California National Primate Research Center, University of California, Davis, California, USA

[2]Department of Psychology, University of California, Davis, California, USA

[3]LIVES Center, Faculty of Social and Political Sciences, University of Lausanne, Lausanne, Switzerland

Correspondence

John P. Capitanio, California National Primate Research Center, University of California, One Shields Ave, Davis, CA 95616, USA.
Email: jpcapitanio@ucdavis.edu

Funding information

National Institutes of Health, Grant/Award Numbers: P51OD011107, R24OD010962

Abstract

Temperament is a construct whose manifestations are quantifiable from an early age, and whose origins have been proposed as "biological." Our goal was to determine whether maternal rank and infant genotype are associated with five measures of temperament in 3- to 4-month old rhesus monkeys (*Macaca mulatta*), all of whom were born and reared by their mothers in large, outdoor, half-acre cages. Maternal rank was defined as the proportion of animals outranked by each female, and the two genes of interest to us were monoamine oxidase and serotonin transporter, both of which are polymorphic in their promoter regions (*MAOA-LPR* and *5-HTTLPR*, respectively), with one allele of each gene considered a "plasticity" allele, conferring increased sensitivity to environmental events. Our large sample size ($n = 2014–3140$) enabled us to examine the effects of individual genotypes rather than combining genotypes as is often done. Rank was positively associated with Confident temperament, but only for animals with the 5-repeat allele for *MAOA-LPR*. Rank had no other effect on temperament. In contrast, genotype had many different effects, with *5-HTTLPR* associated with behavioral inhibition, and *MAOA-LPR* associated with ratings-based measures of temperament. We also examined the joint effect of the two genotypes and found some evidence for a dose-response: animals with the plasticity alleles for both genes were more likely to be behaviorally inhibited. Our results suggest phenotypic differences between animals possessing alleles for *MAOA-LPR* that show functional equivalence based on in vitro tests, and our data for *5-HTTLPR* revealed differences between short/short homozygotes and long/short heterozygotes, strongly suggesting that combining genotypes for statistical analysis should be avoided if possible. Our analysis also provides evidence of sex differences in temperament, and, to our knowledge, the

Abbreviations: AIC, Akaike's Information Criterion; BBA, biobehavioral assessment program; BI, behavioral inhibition; EDTA, ethylenediaminetetraacetic acid; ICC, Intraclass correlation coefficient; LR, likelihood ratio; MAOA-LPR, monoamine oxidase-A linked polymorphic region; MCMC, Monte Carlo with Markov Chain method; SLC6A4, gene that codes for the serotonin transporter (5-HT); SPSS, statistical package for the social sciences; 2LL, Deviance−2 *log-likelihood; 5-HTTLPR, serotonin-transporter-linked promoter region.

only evidence of differences in temperament based on specific pathogen-free status. We suggest several directions for future research.

KEYWORDS
behavior genetics, behavioral inhibition, monoamine oxidase-A promoter, plasticity alleles, serotonin transporter promoter

1 | INTRODUCTION

Temperament is a multidimensional concept with a long history (Capitanio, 2008; Kagan, 1994), but an overarching commonality has been the linking of behavioral traits with some aspect of the individual's "constitution." That is, temperament is typically considered as having a strong biological basis (Rothbart & Derryberry, 1981). The nature of this biological basis, however, remains unclear, though there has been considerable interest in prenatal contributions (and particularly stress experienced by the pregnant female) and genetics (for reviews, see Huizink, 2012; Saudino & Wang, 2012, respectively).

In studying influences of both prenatal and genetic factors on temperament, research with animal, and particularly primate, models have been a useful adjunct to studies done with humans, with animal studies providing a degree of experimental control and precision that is often lacking in human studies. For example, in examining the role of prenatal influences, human studies have focused either on naturally-occurring anxiety in the pre-partum period (e.g., Vaughn et al., 1987) or on the experience of "naturally-occurring" stressors, such as the 9/11 World Trade Center attacks (Brand et al., 2006) or the aftermath of Hurricane Katrina (Tees et al., 2010). Primate studies, on the other hand, have been able to use experimental methods to study the effects of stress in the pre-partum period. One of the more productive paradigms has been use of an acoustic startle stimulus, delivered at precise times during gestation (Coe et al., 2003). This procedure has been found, for example, to increase animals' emotional reactivity to stress (Clarke & Schneider, 1993; Schneider, 1992; Worlein & Sackett, 1995), and there is evidence that when, during gestation, the stimulus is experienced affects outcomes (Rendina et al., 2016).

Primate studies have also been useful in examining the effect on temperament of allelic variation in specific candidate genes. One of the first genes to be examined is the gene that codes for the serotonin transporter (*SLC6A4*, also known as *5-HTT*), the molecule responsible for reuptake of serotonin from the synaptic cleft following its release. A length polymorphism in a promoter region of this gene (*5-HTTLPR*) affects the transcription of the transporter protein, with a short allele showing reduced transcriptional efficiency compared to the long allele (Lesch et al., 1997). Animals with at least one short allele showed greater emotionality compared to long/long homozygotes (Champoux et al., 2002). A second gene of interest is the X-linked monoamine oxidase-A (*MAOA*) gene, the protein product of which deaminates the monoamine neurotransmitters. This gene also has a polymorphism in its promoter region (*MAOA-LPR*), consisting of a variable number of repeat sequences, and this polymorphism has been related to variation in transcriptional efficiency of the gene in both humans (Sabol et al., 1998) and rhesus monkeys (Newman et al., 2005). The low transcriptional activity alleles have been associated with poor affect regulation (e.g., depression: Fan et al., 2010). Barr (2012) provides a recent review of this literature in primates.

Importantly, both *5-HTTLPR* and *MAOA-LPR* often exert their effects as moderators for experimental interventions or experiences of some type, either pharmacological or behavioral. For both genes, the alleles that promote reduced transcriptional efficiency were initially considered "risk" alleles, insofar as individuals that experience some form of adversity showed poorer behavioral outcomes if they also had the low-transcriptional variants of the genes. The classic result demonstrating this showed poor outcomes for maltreated children, but those that also had the low-activity alleles were more likely to show these poor outcomes (Caspi et al., 2002). Similarly, in rhesus monkey infants, we (Karere et al., 2009) have shown interactions of *MAOA-LPR* genotype and the rearing environment. Growing evidence, however, suggests these genes may be viewed more productively as "sensitivity" or "plasticity" alleles: under adverse conditions, they typically confer greater risk for poor outcomes, but data also suggest that under extremely beneficial conditions they promote more positive outcomes (Belsky & Pluess, 2009; Ellis et al., 2011; Taylor et al., 2006).

As described above, human research has often focused on naturally-occurring events during pregnancy, while primate research has typically focused on the use of tightly controlled experimental paradigms to understand how prenatal exposure to stress might impact infant temperament (although exceptions exist: Herrington et al., 2016, showed that prenatal exposure to a matrilineal overthrow influenced emotionality and anxiety in infant rhesus monkeys, and Walker et al., 2018, showing that naturally-occurring variation in maternal adiposity before pregnancy was associated with lower Confident temperament among infant rhesus). One reason for these different approaches may be related to sample size—studies of naturally-occurring events in humans typically involve large sample sizes, which is impractical at many primate facilities.

One component of social life that might influence variation in temperament is social rank, which is an important organizing feature of rhesus macaque social life. Recent studies with macaques have shown that subordinate animals in small social groups can show a variety of adverse behavioral and physiological effects (Shively, 1998; Snyder-Mackler et al., 2016, 2019; Tamashiro et al., 2011) including

effects on personality (Kohn et al., 2016). These consequences are typically conceptualized as resulting from the chronic stress associated with social subordination, some consequences of which are likely to influence development during gestation. Rather surprisingly, only a single paper exists to our knowledge that examined the effects of maternal rank on offspring temperament: Suarez-Jimenez et al. (2013) reported that infants of high-ranked mothers showed greater affective reactivity in response to adverse events, such as during the presence of a human intruder. This was a relatively small study ($N = 26$ infants), where rank was operationalized as high versus low/middle.

The goal of the present study was to determine whether maternal social rank is associated with infant temperament and whether *5-HTTLPR* or *MAOA-LPR* contribute independently to temperament or moderate the effects of rank. Specifically, we were interested in whether high social rank conferred any positive benefits, and low social rank conferred negative consequences, for infant temperament. Because there is some evidence in the literature that middle-ranked animals might show differences from low- and high-ranked animals (see references and discussion in Weinstein et al., 2014), we also were interested in whether the relationship between rank and temperament might be nonlinear.

We elected to look at the effects of all of the major genotypes for these two genes. Often, for *MAOA-LPR*, the low transcriptional genotype(s) are combined (there are more than one in humans, but only one in rhesus) as are the high transcriptional genotypes. Similarly, for *5-HTTLPR*, the heterozygotes (short/long) are often grouped with the homozygotes (short/short) to examine the effects of possessing at least one short allele. Groupings of this type are common in human studies, but especially so in nonhuman primate studies because sample sizes are typically so low, particularly in the more rare subgroups (e.g., for *5-HTTLPR*, short/short). Because we had more than 2000 subjects in our sample, we have sufficient statistical power to identify small-sized lower-level effects of individual genotypes on measures of infant temperament (Arend & Schäfer, 2019). Finally, because Belsky and Beaver (2011) reported a dose-response effect for plasticity alleles from multiple genes, and because we had a large sample size, we also examined whether both genes contributed to differences in temperament.

Our five measures of temperament were obtained from the data archive for the BioBehavioral Assessment (BBA) program at the California National Primate Research Center (CNPRC). Four of the measures (Confident, Gentle, Nervous, Vigilant) were obtained from ratings conducted at the end of the 25-h assessment period and were identified using exploratory and confirmatory factor analyses with oblique rotations, as described by Golub et al. (2009). The fifth measure, behavioral inhibition (BI), was derived from focal animal observations taken on the animals at the beginning and the end of the 25-h period. Importantly, all five measures have been shown to be related to behavioral and/or health outcomes assessed many years after the initial assessments: these include diarrhea (Elfenbein et al., 2016; Gottlieb et al., 2018), asthma (Capitanio, Miller, et al., 2011; Chun et al., 2013), and various behavioral outcomes (Capitanio, 2019; Capitanio et al., 2017; Chun & Capitanio, 2016; Fox et al., 2021; Gottlieb et al., 2013; Vandeleest et al., 2011). The relationships between infant-assessed temperament and later behavioral and health outcomes provide evidence that the temperament measures are in fact assessments of traits, and not simply transient states. Moreover, because genetic variation has been related to variation in these temperament measures (heritability estimates ranged from 0.2 to 0.4, a range similar to that seen in human studies [Blomquist et al., 2021; Fox et al., 2021]), it seems reasonable that our two candidate genes of interest might influence temperament.

2 | METHOD

All procedures reported in this paper were approved by the Institutional Animal Care and Use Committee of the University of California, Davis, and complied with all local, state, and federal regulations. The research also adhered to the American Society of Primatologists' Principles for the Ethical Treatment of Non-Human Primates.

2.1 | Open science statement

Raw data and SPSS and Stata script files to reproduce the findings are available on the OSF: https://osf.io/untrc/?view_only=7469c7618d5840f0a68533f57269e837

2.2 | Subjects

The initial sample comprised $N = 3424$ infant rhesus monkeys (mean age = 106.6 days, range = 88–134 days; 46.2% male). This included all animals (a) assessed in CNPRC's BBA program between 2001 and 2019; (b) born into one of 24 half-acre outdoor field corrals, containing up to 200 animals of all ages and both sexes; (c) socially-reared in the field corrals by their biological mothers (i.e., not fostered) as verified by a microsatellite panel designed to identify maternity and paternity (Andrade et al., 2004; Kanthaswamy et al., 2006); and (d) for whom complete rank data were available.

From this initial sample, six subsamples were drawn, one for each of the analyses presented below. Samples 1 and 2 were drawn for the *5-HTTLPR* analyses and involved eliminating animals (a) for whom *5-HTTLPR* data were missing (due to failure to obtain a blood sample, or genotyping results that were ambiguous: $n = 217$) and (b) animals that possessed the rare XL allele ($n = 58$). In addition, 13 animals were missing data on one or more of the four temperament ratings variables, resulting in $n = 3136$ for Sample 1, and nine animals were missing data for the BI measure, resulting in $n = 3140$ for Sample 2.

Samples 3 and 4 were drawn for the *MAOA-LPR* analyses. Animals from the initial sample were eliminated if they had missing genotype data ($n = 179$), and if they had genotypes other than 5/-, 6/-, and 7/- for the hemizygous males, and 5/5, 6/6, and 7/7 for the

homozygous females (recall that *MAOA-LPR* is X-linked) ($n = 1083$ were eliminated). Ten animals were missing data on one or more of our four ratings-derived measures, leaving $n = 2152$ for Sample 3, and six animals were missing data for BI, leaving $n = 2156$ for Sample 4.

Samples 5 and 6 were drawn for the two-gene analysis and involved the same criteria as above for the two genes; after eliminating animals that had valid data for both genes but were missing data on our outcome measures, our final sample sizes were $n = 2014$ for the two-gene analysis of the four ratings measures (Sample 5) and $n = 2017$ for the two-gene analysis of BI (Sample 6). Table 1A describes the demographic characteristics of the six samples.

2.3 | Procedure

2.3.1 | BBA program

The BBA program at the California National Primate Research Center comprises a series of tests conducted over a 25-h period designed to assess various aspects of behavior and physiology (for details see Capitanio, 2017, 2022; Golub et al., 2009). Mothers and infants were net captured from their field corrals, separated from each other, and delivered to holding cages in the testing room (infants) or to an area (mothers) that was outside of sensory range of the infants. Infants, which were always tested in cohorts of five to eight animals, arrived at 0900 h, and were housed individually in standard-sized cages (0.61 × 0.69 × 0.81 m, Lab Products) indoors. Infants were returned to their mothers at 1000 h the following day, where they were allowed an hour to nurse before return to their natal cages with their mothers. Each infant holding cage contained a stuffed furry toy duck, a towel, and a novel object that the infants could manipulate. Infants were provided with water ad libitum, orange-flavored drink, fresh fruit, and commercial monkey chow. Three assessments from the BBA program are considered in this report.

Holding cage behavior

Each animal was observed by a technician for two 5-min periods in a predetermined random order: once approximately 15 min after relocation to the holding cage (0915 h) on Day 1 of the BBA and again at 0700 on Day 2. An observer sat approximately 2.4 m in front of the holding cage while avoiding eye contact with the subject. All behaviors were scored according to the ethogram in Golub et al. (2009), using The Observer software program (Noldus, 1991), by a trained observer whose interobserver reliabilities exceeded 85% agreement. Frequencies of behaviors were converted to a rate per 60 s, and durations were converted to a proportion of total time observed. Data from the larger BBA program (>1400 animals, including animals from other rearing conditions) were treated to exploratory and confirmatory factor analyses (Golub et al., 2009), and two factors were identified: "Emotionality" comprised the rate of vocalizations (barks and coos); and whether the subject displayed lipsmacks, threats, and self-scratching behaviors; and "Activity" that comprised the proportion of time locomoting; proportion of time an animal was not in a hanging position on the front, sides, or top of the cage; rate of environmental exploration; and whether or not the animal drank, ate food, or was in a crouched posture. For each year of BBA testing, the Day 1 and Day 2 data were summarized by *z*-scoring each item, then creating the factor analysis-derived scales, which were then re-*z*-scored to have a mean of zero and SD = 1. Animals were considered behaviorally inhibited if their scores on all four scales (Day 1 Emotionality, Day 1 Activity, Day 2 Emotionality, Day 2 Activity) were less than zero. Animals not satisfying these criteria were considered not inhibited. More information about this measure of BI can be found in Capitanio (2019) and Fox et al. (2021).

Temperament ratings

At the conclusion of the 25-h assessment, the technician who performed the testing rated the overall temperament of each animal during the 25-h test period using a listing of 16 adjectives and a

TABLE 1 Demographic characteristics

Sample	1	2	3	4	5	6
A: Demographic characteristics and analyses examined for the six subsamples						
Analysis— genotype	5-HTTLPR	5-HTTLPR	MAOA-LPR	MAOA-LPR	Both	Both
Analysis— outcomes	Ratings	BI	Ratings	BI	Ratings	BI
Sample size	3136	3140	2152	2156	2014	2017
Age (mean and range)	106.7 (88–134)	106.7 (88–134)	106.5 (88–134)	106.5 (88–134)	106.5 (88–134)	106.5 (88–134)
% male	46.6%	46.6%	69.7%	69.5%	69.8%	69.7%
B: Cluster information (numbers of dams and sires, and cluster size [i.e., number of offspring] of each) for each sample						
Number of unique dams	1763	1766	1421	1423	1348	1350
Mean size and range of dam cluster	1.78 (1–9)	1.78 (1–9)	1.51 (1–7)	1.52 (1–7)	1.49 (1–7)	1.49 (1–7)
Number of unique sires	759	759	662	662	644	645
Mean size and range of sire cluster	4.13 (1–62)	4.14 (1–62)	3.25 (1–46)	3.26 (1–46)	3.13 (1–42)	3.13 (1–42)

Abbreviation: BI, behavioral inhibition.

seven-point Likert-type scale, with "1" reflecting a total absence of the behavior and "7" reflecting an extremely large amount of the behavior. Exploratory and confirmatory factor analyses suggested a four-factor structure to the data: Confident (confident, bold, active, curious, playful), Gentle (gentle, calm, flexible, curious), Nervous (nervous, fearful, timid, not calm, not confident), and Vigilant (vigilant, not depressed, not tense, not timid; note, the adjectives preceded by "not" indicate the item was reverse-scored; e.g., a high score on the Vigilant scale indicated that animals tended to have high scores on the individual vigilant item, and low scores on the depressed, tense, and timid items). As with the holding cage behavior scales, factor scores were *z*-scored within each assessment year. Trait definitions and details of the factor analyses and scale construction can be found in Golub et al. (2009).

Genotyping

Genotyping was performed on buffy coats obtained from a blood sample drawn on the animals at 1100 h on Day 1. One ml whole blood was obtained via femoral venipuncture using manual restraint. Blood was collected in an un-heparinized syringe and was immediately transferred to two ½ ml tubes containing ethylenediaminetetraacetic acid (EDTA) as an anti-coagulant. One tube was spun in a refrigerated centrifuge (4°C) at 1277 g for 10 min, after which the plasma was removed. The remaining buffy coat was refrigerated for later genotyping. The multiplex methods for genotyping have been described in Karere et al. (2012); the same methods were used across years. *MAOA-LPR* alleles were classified according to repeat number of 5 (240 base pairs), 6 (258 bp), and 7 (276 bp) (small numbers of animals had other repeat numbers; they are not considered here). As the *MAOA* gene resides on the X-chromosome, males are hemizygous and females can be homo- or heterozygous. But because of X-inactivation, it is unclear which allele is being expressed in any given cell of a heterozygote, so those animals were dropped as described above, leaving 5/-, 6/-, and 7/- allele male hemizygotes, and 5/5, 6/6, and 7/7 homozygous females (Below, we also refer to the *MAOA-LPR* genotypes for both males and females as the 5-repeat, 6-repeat, and 7-repeat genotypes, respectively). Two alleles were found in the serotonin transporter promoter (*5-HTTLPR*), consisting of 402 bp (long allele) and 381 bp (short allele), resulting in three genotypes: L/L, L/S, and S/S (the small number of animals with an XL allele were dropped as described above). Table 2 shows the genotype distribution for each sample.

TABLE 2 Genotype distribution

Sample	1	2	3	4	5	6
Sample size	3136	3140	2152	2156	2014	2017
5-HTTLPR: S/S	172	171	-	-	100	100
5-HTTLPR: S/L	1096	1095	-	-	690	688
5-HTTLPR: L/L	1868	1874	-	-	1224	1229
MAOALPR: 5/-, 5/5	-	-	654	660	605	610
MAOALPR: 6/-, 6/6	-	-	622	620	582	581
MAOALPR: 7/-, 7/7	-	-	876	876	827	826

Note: Genotype frequencies for the four subsamples.

2.3.2 | Rank

Rank data were obtained from CNPRC's Behavioral Management unit. Data are collected approximately monthly on animals in each corral. The staff conducts observations after throwing several handfuls of sunflower seeds into the corral to bring the animals to the front of the corral. While the monkeys forage, observers monitored the group for aggressive and submissive interactions as the monkeys compete for the limited resource, recording wins and losses. Monkeys were ranked higher if they threatened, displaced, chased, or attacked another monkey; monkeys were ranked lower if they surrendered resources or their location, or showed submissive behaviors like fear grimacing, or moving out of proximity of another monkey. Subjects were ranked separately by sex, with the alpha male and alpha female ranked as 1. Because young monkeys often share the dominance rank of their mother before puberty (Bernstein & Williams, 1983), the observers do not rank subjects younger than 1-year old; consequently, we used maternal rank in our analyses. For each mother, rank data were converted to the proportion of animals outranked by using the formula:

Proportion outranked =
(number of females ranked minus the rank of the specific animal)
/(number of females ranked minus 1).

Using this formula, the alpha animals had values of 1.0, and the lowest-ranked females had a value of 0.0. In any given year, the specific hierarchies used to determine maternal rank were chosen to be within ±1 month of the infants' BBA test date. Maternal ranks are extremely stable in our colony; for cages in which ranks are unstable (due, e.g., to a matrilineal overthrow, a new group formation, or a cage in which Behavioral Management staff are aware of social instabilities), no BBA testing would be conducted on animals from that cage that year.

Assessing whether there is a nonlinear influence of rank involved adding a quadratic term ($rank^2$) to the analyses. Because proportions have the unusual characteristic whereby a squared value is less than the original value, we added the constant 1 to each animal's rank proportion. The only impact of this transformation is to change the intercept term for the models. The quadratic term was created by squaring the now-transformed rank variable.

3 | DATA ANALYSIS

3.1 | Overview of the cross-classified multilevel linear and logistic analysis

We built a series of multilevel models in which infant rhesus monkeys (level-1 units) were nested in dam (level-2a units) and cross-classified

by sire (level 2b-units). The units are said to be cross-classified because infant rhesus monkeys nested in a given dam are not necessarily subclassified by the same sire (i.e., infants from the same dam may have a different sire or vice versa). Cross-classified multilevel, rather than single-level, modeling was chosen because evidence suggests that our measures of temperament are heritable, and we wanted to disentangle the variance due to between-infant differences from the variance due to the principal genetic relationships, parentage. Table 1B shows the numbers of unique dams and sires in each of the samples, as well as the mean number (and range) of offspring per parent.

Specifically, we built a series of cross-classified multilevel *linear* models to predict the four ratings-derived, continuous measures of temperament (Sommet & Morselli, 2021). For these analyses, we used the MIXED procedure in IBM SPSS, version 26 (we used maximum likelihood to estimate parameters). Moreover, we built a series of cross-classified multilevel *logistic* models to predict the behavior-derived binary measure of BI (Sommet & Morselli, 2017). For these analyses, we used the xtmixed command in Stata 16.1 (we used maximum likelihood to estimate parameters, except for the more complex two-gene analysis for we used Monte Carlo with Markov Chain [MCMC] method; see Dunn et al., 2015). For the results presented below, we provide measures of effect sizes for the logistic models only (i.e., odds ratios), inasmuch as there does not yet appear to be unbiased methods of calculating effect sizes for multilevel linear regression models (LaHuis et al., 2014).

3.2 | Analytical strategy

3.2.1 | Preliminary step

For each outcome, we first examined null models (i.e., with no predictors) and calculated the intraclass correlation coefficients (ICCs), which reflect the proportion of total variance in the outcome measures due to between-cluster differences (i.e., the sire cluster on the one hand and the dam cluster on the other hand). Some authors have suggested that an ICC below 0.05 indicates so little between-cluster variation that multilevel models are unnecessary (Heck et al., 2014), but this perspective is not universally shared (Huang, 2018; Musca et al., 2011).

3.2.2 | Single-gene analysis

Next, we ran a series of four nested cross-classified multilevel models (one series for the *5-HTTLPR* genotype and one series for the *MAOA-LPR* genotype) and compared model fits. We performed a likelihood ratio (LR) test to compare each pair of models; the LR test is calculated as the difference between deviance (−2 × the log-likelihood [−2LL]) for successive models, and the difference is distributed as chi-square with *df* equal to the number of parameters added to the previous model. Table S16 provides the deviance for each model and each analysis and also includes Akaike's Information Criterion (AIC) for readers more interested in an information-theoretic approach. We note that both the LR test and the AIC approach (utilizing the common two-point difference in AIC for identifying models: Burnham et al., 2011) pointed to the same final models in every case.

Model 1

The first cross-classified multilevel model (M1) included five covariates that were not especially of interest, but were included because prior analyses had indicated they might explain variance in one or more of our outcome measures. These measures included sex (coded −0.5 for females and +0.5 for males), firstborn (−0.5 for animals that were not the firstborn live offspring, +0.5 if they were the firstborn), and two continuous measures of age and weight at BBA testing, both of which were grand-mean centered before analysis. We also included specific pathogen-free (SPF) status. SPF animals live in identical circumstances as do non-SPF animals, but such animals had progenitors that were nursery-reared, as that was the method used to derive SPF animals at our facility. We coded SPF status as −0.5 if the animals were "conventionally" reared, that is, not free of specific pathogens such as B-virus, simian T-lymphotropic virus, and so forth, and +0.5 if they were raised in cages free of those pathogens (see Yee et al., 2016). Below are the regression equations for the cross-classified multilevel linear (Equation 1) and logistic (Equation 1′) models.

$$Y_{ijk} = B_{000} + B_{100} \times \text{Sex}_{ijk} + B_{200} \times \text{Firstborn}_{ijk} + B_{300} \times \text{Age}_{ijk} + B_{400} \times \text{Weight}_{ijk} + B_{500} \times \text{SPF\_Status}_{ijk} + v_k + u_j + e_{ijk}, \quad (1)$$

where Y_{ijk} is the continuous temperament rating, v_k represents the level-2a residuals ($k = 1, 2, \ldots I$ dams), u_j represents the level-2b residuals ($j = 1, 2, \ldots, k$ sires), and e_{ijk} represents the level-1 residuals ($i = 1, 2, \ldots, n$ infant rhesus monkeys). As for the regression coefficients, B_{n00} designates the n^{th} level-1 predictor, B_{0n0} designates the n^{th} level-2a predictor, B_{00n} designate the n^{th} level-2b predictor (for a similar nomenclature, see Hox et al., 2017).

$$\textit{Logit}(P_{ijk}) = B_{000} + B_{100} \times \text{Sex}_{ijk} + B_{200} \times \text{Firstborn}_{ijk} + B_{300} \times \text{Age}_{ijk} + B_{400} \times \text{Weight}_{ijk} + B_{500} \times \text{SPF\_Status}_{ijk} + v_k + u_j, \quad (1')$$

where P_{ijk} represents the probability that the animal is behaviorally inhibited.

Model 2

The second model (M2) added the main effects of rank and genotype. Rank, defined above as proportion of animals out-ranked, with one added, was grand-mean centered. Genotype was treated as a three-category variable and broken down into two dummies: For the analysis of *5-HTTLPR*, we used the L/L genotype as the reference category, whereas for the *MAOA-LPR* analysis, we used the 7/7 (or

for males, 7/-) genotype as the reference category. Individual contrasts for either gene were examined only if the omnibus test for that gene was statistically significant. Below are the regression equations for the cross-classified multilevel linear (Equation 2) and logistic (Equation 2′) models.

$$Y_{ijk} = B_{000} + B_{i00} \times \mathrm{M1}_{ijk} + B_{600} \times \mathrm{Rank}_{ijk} + B_{700} \times \mathrm{Dummy\_1}_{ijk} + B_{700} \times \mathrm{Dummy\_2}_{ijk} + v_k + u_j + e_{ijk}, \quad (2)$$

where $B_{i00} \times \mathrm{M1}_{ijk}$ represents a vector of the predictor variables used in M1.

$$Logit\,(P_{ijk}) = B_{000} + B_{i00} \times \mathrm{M1}_{ijk} + B_{600} \times \mathrm{Rank}_{ijk} + B_{700} \times \mathrm{Dummy\_1}_{ijk} + B_{700} \times \mathrm{Dummy\_2}_{ijk} + v_k + u_j. \quad (2')$$

Model 3

The third model (M3) included the M2 variables plus the two first-order interaction terms between rank and genotype, which were constructed by multiplying the rank and each of the dummy-coded genotype variables. Below are the regression equations for the cross-classified multilevel linear (Equation 3) and logistic (Equation 3′) models.

$$Y_{ijk} = B_{000} + B_{i00} \times \mathrm{M2}_{ijk} + B_{800} \times \mathrm{Rank}_{ijk} \times \mathrm{Dummy\_1}_{ijk} + B_{900} \times \mathrm{Rank}_{ijk} \times \mathrm{Dummy\_2}_{ijk} + v_k + u_j + e_{ijk}, \quad (3)$$

where $B_{i00} \times \mathrm{M2}_{ijk}$ represents a vector of the predictor variables used in M2.

$$Logit\,(P_{ijk}) = B_{000} + B_{i00} \times \mathrm{M2}_{ijk} + B_{800} \times \mathrm{Rank}_{ijk} \times \mathrm{Dummy\_1}_{ijk} + B_{900} \times \mathrm{Rank}_{ijk} \times \mathrm{Dummy\_2}_{ijk} + v_k + u_j. \quad (3')$$

Model 4

The final model (M4) included the M3 variables as well as a quadratic term for rank and the quadratic interaction terms, which were constructed by multiplying rank^2 and each of the dummy-coded genotype variables. Below are the regression equations for the cross-classified multilevel linear (Equation 4) and logistic (Equation 4′) models.

$$Y_{ijk} = B_{000} + B_{i00} \times \mathrm{M3}_{ijk} + B_{1100} \times \mathrm{Rank}^2_{ijk} + B_{1200} \times \mathrm{Rank}^2_{ijk} \times \mathrm{Dummy\_1}_{ijk} + B_{900} \times \mathrm{Rank}^2_{ijk} \times \mathrm{Dummy\_2}_{ijk} + v_k + u_j + e_{ijk}, \quad (4)$$

where $B_{i00} \times \mathrm{M2}_{ijk}$ represents a vector of the predictor variables used in M3.

$$Logit\,(P_{ijk}) = B_{000} + B_{i00} \times \mathrm{M3}_{ijk} + B_{1100} \times \mathrm{Rank}^2_{ijk} + B_{1200} \times \mathrm{Rank}^2_{ijk} \times \mathrm{Dummy\_1}_{ijk} + B_{900} \times \mathrm{Rank}^2_{ijk} \times \mathrm{Dummy\_2}_{ijk} + v_k + u_j. \quad (4')$$

3.2.3 | Follow-up two-gene analysis

For the follow-up two-gene analyses, the same analytic strategy was followed: M1 comprised the five covariates; M2 added to Model 1 the rank main effect, and the two dummy variables for *5-HTTLPR* and the two dummies for *MAOA-LPR*; M3 added to Model 2 the interactions between rank and *5-HTTLPR*, rank and *MAOA-LPR*, and the interaction between the two genotypes. However, we did not test for a quadratic effect since we found no evidence of such an effect in our single-gene analyses; moreover, our model-building did not include the rank ×5-HTTLPR × MAOA-LPR interaction because we did not think that our sample size would allow us to estimate such complex second-order two-gene interactions.

4 | RESULTS

4.1 | ICCs

Inspection of the null models indicated that parentage had an impact on all of our outcome measures, as indicated by ICCs, which are presented in Table 3. Values ranged from 0.048 to 0.161 for dams and 0.072 to 0.129 for sires.

4.2 | Single-gene analysis

4.2.1 | Analysis 1: Influence of rank and 5-HTTLPR genotype on temperament ratings

Summary

We found no evidence that rank or *5-HTTLPR* genotype was associated with the four ratings-derived temperament measures.

TABLE 3 Intraclass correlations coefficients

	Confident	Gentle	Nervous	Vigilant	BI
Sample	*1*	*1*	*1*	*1*	*2*
Dams	0.112	0.140	0.111	0.117	0.065
Sires	0.109	0.087	0.072	0.095	0.093
Sample	*3*	*3*	*3*	*3*	*4*
Dams	0.151	0.157	0.124	0.131	0.052
Sires	0.124	0.095	0.075	0.112	0.100
Sample	*5*	*5*	*5*	*5*	*6*
Dams	0.140	0.161	0.120	0.143	0.048
Sires	0.129	0.100	0.084	0.121	0.104

Note: Intraclass correlations for dams and sires. The number of the sample from which the ICCs were calculated is indicated.

Abbreviations: BI, behavioral inhibition; ICCs, intraclass correlation coefficients.

TABLE 4 Analysis 1, 5-*HTTLPR*, and temperament rating

	Confident (Model 1)		Gentle (Model 1)		Nervous (Model 1)		Vigilant (Model 1)	
	B	95% CI	*B*	95% CI	*B*	95% CI	*B*	95% CI
Intercept	−0.09**	(−0.14, −0.04)	−0.11*	(−0.17, −0.06)	0.01	(−0.04, 0.05)	−0.09*	(−0.15, −0.04)
Sex	−0.14*	(−0.21, −0.07)	0.1**	(0.03, 0.17)	−0.07***	(−0.14, 0)	−0.1**	(−0.17, −0.03)
Age (in days)	0	(0, 0)	0	(−0.01, 0)	0	(0, 0)	0	(−0.01, 0)
Weight (in kg)	0.2	(−0.05, 0.45)	−0.1	(−0.35, 0.15)	−0.13	(−0.37, 0.11)	−0.21	(−0.47, 0.05)
Firstborn	0.07****	(−0.01, 0.16)	0.04	(−0.04, 0.12)	−0.04	(−0.12, 0.04)	0.09***	(0.01, 0.18)
SPF status	−0.17*	(−0.27, −0.08)	−0.14**	(−0.23, −0.05)	0.16**	(0.07, 0.24)	−0.19*	(−0.28, −0.09)
Variance parameters								
Var (level 1)	0.77*	(0.72, 0.83)	0.78*	(0.72, 0.84)	0.78*	(0.73, 0.84)	0.85*	(0.79, 0.91)
Var (dam ID)	0.12*	(0.08, 0.17)	0.14*	(0.1, 0.19)	0.1*	(0.06, 0.15)	0.13*	(0.09, 0.18)
Var (sire ID)	0.1*	(0.07, 0.14)	0.08*	(0.06, 0.12)	0.06*	(0.04, 0.1)	0.09*	(0.06, 0.13)

Note: Coefficient estimates and 95% confidence intervals (CIs) from the cross-classified multilevel models (best models) testing the influence of rank and 5-HTTLPR genotype on the temperament ratings.
*$p < 0.001$.; **$p < 0.01$.; ***$p < 0.05$.; ****$p < 0.10$.

Model fits

For all measures, M1, which included only the covariates, fit the data better than the null model (Confident: $\chi^2(5) = 33.10$, $p < 0.001$; Gentle: $\chi^2(5) = 21.39$, $p < 0.001$; Nervous: $\chi^2(5) = 19.47$, $p = 0.002$; Vigilant: $\chi^2(5) = 35.76$, $p < 0.001$). M2, which added the rank and genotype main effects did not fit the data better than M1 (all ps > 0.400). Similarly, M3, which added the rank by genotype interactions, and M4, which added the quadratic rank term and interactions did not provide better fits (M3: all $p > 0.463$; M4: all $p > 0.309$). Table 4 presents the best models, and Tables S1–S4 present the full results of all models.

Best model

Inspection of M1 for the four measures showed that females had higher scores for Confident, Nervous, and Vigilant, and lower scores for Gentle compared to males. SPF animals had higher values for Nervous, and lower values for Confident, Gentle, and Vigilant. Finally, firstborn animals had higher scores for Vigilant.

4.2.2 | Analysis 2: Influence of rank and 5-HTTLPR genotype on BI

Summary

We found evidence that the *5-HTTLPR* genotype, but not rank, was associated with BI.

Model fits

M1, which included only the covariates, fit the data better than the null model, $\chi^2(5) = 17.94$, $p = 0.003$. M2, which added the rank and genotype main effects, fit the data better than did M1, $\chi^2(3) = 8.03$, $p = 0.046$. M3, which added the rank by genotype interactions, and M4, which added the quadratic rank term and interactions did not provide better fits (M3: $p = 0.994$; M4: $p = 0.331$). Table 5 presents the best model, and Table S5 presents the full results of all models.

TABLE 5 Analysis 2, 5-*HTTLPR*, and behavioral inhibition

	Model 2	
	B	95% CI
Intercept	−1.39*	(−1.56, −1.22)
Sex	0.26**	(0.07, 0.45)
Age (in days)	0	(−0.01, 0.01)
Weight (in kg)	−0.03	(−0.68, 0.62)
Firstborn	−0.13	(−0.36, 0.09)
SPF status	0.32**	(0.09, 0.54)
Rank	0.22	(−0.11, 0.55)
S/S versus L/S & L/L	−0.54***	(−1.02, −0.05)
L/S versus S/S & L/L	0.06	(−0.14, 0.26)
Variance parameters		
Var (dam ID)	0.26	(0.09, 0.73)
Var (sire ID)	0.32	(0.18, 0.58)

Note: Coefficient estimates and 95% confidence intervals (CIs) for cross-classified multilevel logistic models (best model) testing the influence of rank and 5-HTTLPR genotype on behavioral inhibition.
*$p < 0.001$.; **$p < 0.01$.; ***$p < 0.05$.; ****$p < 0.10$.

Best model

Inspection of M2 showed that the omnibus test for genotype was marginally significant, $\chi^2(5) = 5.82$, $p = 0.054$. Specifically, animals with the S/S genotype were 1.72 times less likely to be BI (a 42% lower odds) compared to animals with the L/S or L/L genotypes ($B = -0.54$, $Z = -2.17$, $p = 0.030$, OR = 0.58; Figure 1). In addition, males and SPF animals were more likely to be BI.

4.2.3 | Analysis 3: Influence of rank and MAOA-LPR genotype on temperament ratings

Summary

We found evidence that the *MAOA-LPR* genotype was associated with the four ratings-derived temperament measures. We additionally found evidence that the *MAOA-LPR* genotype interacted with rank for Confident temperament.

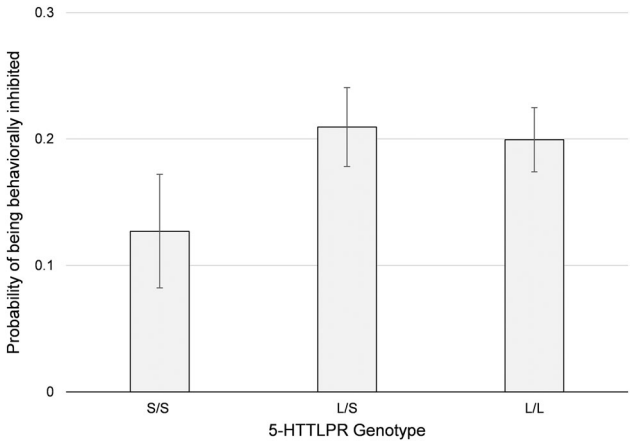

FIGURE 1 Analysis 2. Probability of being behaviorally inhibited as a function of the 5-HTTLPR genotype (S/S vs. L/S vs. L/L). Error bars represent 95% confidence intervals; the estimates come from the best model (M2)

Model fits (Confident)

For Confident temperament, M1, which included only the covariates, fit the data better than the null model, $\chi^2(5) = 34.75$, $p < 0.001$. M2, which added the rank and genotype main effects, fit the data better than did M1, $\chi^2(3) = 13.743$, $p = 0.003$. M3, which added the rank by genotype interactions, fit the data better than did M2, $\chi^2(2) = 7.14$, $p = 0.028$. M4, which added the quadratic rank term and interactions did not provide better fit ($p = 0.760$). Table 6 presents the final models, and Table S6 presents the full results of all models.

Model fits (Gentle, Nervous, and Vigilant)

For Gentle, Nervous, and Vigilant temperament, M1, which included only the covariates, fit the data better than the null model, χ^2s$(5) \geq 11.231$, $p \leq 0.047$. M2, which added the rank and the genotype and rank main effects, had a significantly better fit than did M1 (Gentle: $\chi^2(3) = 10.29$, $p = 0.016$; Nervous: $\chi^2(3) = 11.34$, $p = 0.010$; Vigilant: $\chi^2(3) = 12.98$, $p = 0.005$). M3, which added the rank by genotype interactions, and M4, which added the quadratic rank term and interactions did not provide better fits (M3: ps > 0.467; M4: ps > 0.550). Table 6 presents the best models, and Tables S7–S9 present the full results of all models.

TABLE 6 Analysis 3, *MAOA-LPR*, and temperament ratings

	Confident (Model 3)		Gentle (Model 2)		Nervous (Model 2)		Vigilant (Model 2)	
	B	95% CI	*B*	95% CI	*B*	95% CI	*B*	95% CI
Intercept	0.03	(−0.05, 0.11)	−0.02	(−0.1, 0.06)	−0.07****	(−0.15, 0.01)	0.01	(−0.07, 0.1)
Sex	−0.19*	(−0.28, −0.09)	0.09***	(0, 0.19)	−0.08****	(−0.17, 0.01)	−0.14**	(−0.24, −0.05)
Age (in days)	0	(0, 0)	0	(−0.01, 0)	0	(−0.01, 0)	0	(−0.01, 0)
Weight (in kg)	0.37***	(0.08, 0.66)	0.04	(−0.25, 0.34)	−0.2	(−0.49, 0.08)	−0.02	(−0.33, 0.28)
Firstborn	0.07	(−0.03, 0.17)	0.02	(−0.08, 0.12]	0	(−0.1, 0.1)	0.06	(−0.04, 0.16)
SPF status	−0.21*	(−0.32, −0.1)	−0.15**	(−0.25, −0.04)	0.18*	(0.08, 0.28)	−0.22*	(−0.33, −0.11)
Rank	−0.17	(−0.41, 0.06)	−0.03	(−0.18, 0.13)	0	(−0.15, 0.15)	0.06	(−0.1, 0.22)
5/5 or 5/- versus others	−0.12***	(−0.22, −0.01)	−0.15**	(−0.25, −0.05)	0.13***	(0.03, 0.23)	−0.11***	(−0.22, −0.01)
6/6 or 6/- versus others	−0.2*	(−0.3, −0.09)	−0.14***	(−0.24, −0.03)	0.16**	(0.06, 0.26)	−0.19*	(−0.3, −0.08)
Rank × dummy 1	0.47***	(0.11, 0.82)	-	-	-	-	-	-
Rank × dummy 2	0.09	(−0.27, 0.44)	-	-	-	-	-	-
Variance parameters								
Var (level 1)	0.74	(0.67, 0.82)	0.75	(0.68, 0.83)	0.75	(0.68, 0.83)	0.83	(0.75, 0.92)
Var (dam ID)	0.15	(0.09, 0.24)	0.15	(0.1, 0.23)	0.1	(0.06, 0.18)	0.13	(0.07, 0.23)
Var (sire ID)	0.11	(0.08, 0.17)	0.08	(0.05, 0.14)	0.06	(0.03, 0.11)	0.1	(0.07, 0.16)

Note: Coefficient estimates and 95% confidence intervals (CIs) for cross-classified multilevel models (best models) testing the influence of rank and MAOA-LPR genotype on the temperament ratings.

*$p < 0.001$.; **$p < 0.01$.; ***$p < 0.05$.; ****$p < 0.10$.

Best model (Confident)

For Confident temperament, the omnibus tests for genotype and the genotype by rank interaction were both significant (genotype: $F(2, 1974.63) = 6.93$, $p = 0.001$; genotype by rank: $F(2, 1981.24) = 3.59$, $p = 0.028$). The genotype main effect showed that animals with the "sensitivity" genotype (7/- males and 7/7 females) were significantly more likely to show a Confident temperament; this was indicated by significant and negative coefficients for the two dummy variables contrasting animals with the 5-repeat allele versus all others ($B = -0.12$, $p = 0.027$) and animals with the 6-repeat allele versus all others ($B = -0.20$, $p < 0.001$) (Figure 2). Genotype moderated the effect of rank on Confident temperament, however, as indicated by a significant interaction of rank and the 5-repeat dummy measure ($B = 0.47$, $p = 0.010$)—animals that were of higher rank were more likely to demonstrate a Confident temperament compared to animals of lower rank, but only if they had the 5-repeat alleles. Rank was not influential on Confident temperament for animals with the 6- or 7-repeat alleles (Figure 3). Finally, animals were more likely to be Confident if they weighed more at time of BBA testing, were female, and were conventionally (i.e., not SPF) reared.

Best models (Gentle, Nervous, and Vigilant)

For the three measures, inspection of M2 showed that the omnibus tests for the genotype variable were significant: Gentle ($F(2, 1986.86) = 5.19$, $p = 0.006$), Nervous ($F(2, 1938.82) = 5.77$, $p = 0.003$), Vigilant ($F(2, 1934.26) = 6.30$, $p = 0.002$). For each measure, both dummy variables for *MAOA-LPR* were significant: Gentle: 5-repeat ($B = -0.15$, $p = 0.004$), 6-repeat ($B = -0.14$, $p = 0.010$); Nervous: 5-repeat ($B = 0.13$, $p = 0.010$), 6-repeat ($B = 0.16$, $p = 0.002$); Vigilant 5-repeat ($B = -0.11$, $p = 0.039$), 6-repeat ($B = -0.19$, $p < 0.001$). Because both dummies were significant for each measure, and had identical signs, these results suggest that it was members of the reference group,

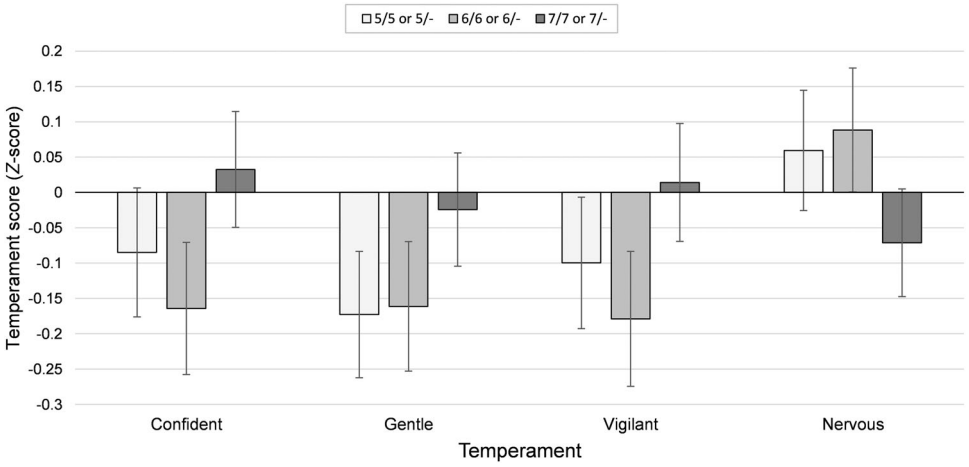

FIGURE 2 Analysis 3. Temperament ratings as a function of the MAOA-LPR genotype (5/5 or 5/- vs. 6/6 or 6/- vs. 7/7 or 7/-). Error bars represent 95% confidence intervals; the estimates come from the best models (for Confident: M3; for the other ratings: M2)

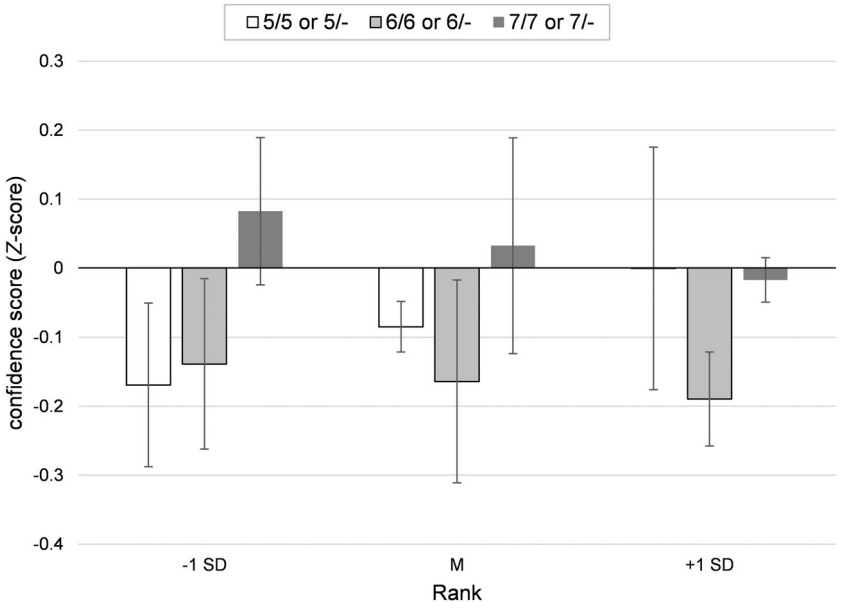

FIGURE 3 Analysis 3. Confidence score as a function of rank and the MAOA-LPR genotype (5-repeat vs. 6-repeat vs. 7-repeat). Error bars represent 95% confident intervals; the estimates come from the best model (M3)

namely the 7/- males and 7/7 females, that were more likely to be Gentle and Vigilant, and less likely to be Nervous (Figure 2). Gentle temperament was also associated with being male and conventionally-reared, Nervous temperament was related to being SPF-reared, and females and conventionally-reared animals showed higher values for Vigilant temperament.

4.2.4 | Analysis 4: Influence of rank and MAOA-LPR genotype on BI

Summary

We found no evidence that rank or *MAOA-LPR* genotype were associated with BI.

Model fits

M1, which included only the covariates, fit the data better than the null, $\chi^2(5) = 20.360$, $p = 0.001$, but the later models did not provide a better fit (M2: $p = 0.107$, M3: $p = 0.327$; M4: $p = 0.108$). Table 7 presents the final model, and Table S10 present the full results of all models.

Best model

Inspection of M1 showed that the odds of being behaviorally inhibited were higher for males and for SPF-reared animals.

4.3 | Two-gene analysis

4.3.1 | Analysis 5: Influence of rank, MAOA-LPR, and 5-HTTLPR genotypes on temperament ratings

Summary

When both genes were considered together as potential influences on the four rating-derived temperament measures, *MAOA-LPR* genotype main effects, but not effects of rank or *5-HTTLPR* (or any interactions) were found for Confident and Gentle temperament; no genotype or rank effects (or interactions) were evident for Nervous and Vigilant temperament.

TABLE 7 Analysis 4, *MAOA-LPR*, and behavioral inhibition

	Model 1	
	B	95% CI
Intercept	−1.51*	(−1.72, −1.3)
Sex	0.45**	(0.19, 0.71)
Age (in days)	0	(−0.01, 0.01)
Weight (in kg)	−0.06	(−0.83, 0.72)
Firstborn	−0.13	(−0.4, 0.15)
SPF status	0.32***	(0.06, 0.59)
Variance parameters		
Var (dam ID)	0.2	(0.03, 1.26)
Var (sire ID)	0.38	(0.19, 0.78)

Note: Coefficient estimates and 95% confidence intervals (CIs) for cross-classified multilevel logistic model (best model) testing the influence of rank and MAOA-LPR genotype on behavioral inhibition.
*$p < 0.001$.; **$p < 0.01$.; ***$p < 0.05$.; ****$p < 0.10$.

Model fits

For all four measures, M1, which included only the covariates, fit the data better than the null model, χ^2s(5) > 11.995, ps < 0.035. M2, which added the rank and genotype main effects, fit better than M1 for Confident, $\chi^2(5) = 11.49$, $p = 0.042$, and Gentle $\chi^2(5) = 11.15$, $p = 0.048$, but not Vigilant, $\chi^2(5) = 11.01$, $p = 0.051$, and Nervous, $\chi^2(5) = 10.60$, $p = 0.061$). M3, which added the rank by genotype interactions, did not fit the data better than M2. Table 8 presents the final models, and Tables S11–S14 present the full results of all models.

Best models

For Confident and Gentle, M2 was the best fit model, and the omnibus tests indicated that the significant effects were attributable to *MAOA-LPR* genotype: Confident ($F(2, 1832.007) = 5.71$, $p = 0.003$); Gentle ($F(2, 1855.537) = 4.16$, $p = 0.016$). Omnibus tests for *5-HTTLPR* were both nonsignificant (Confident: $p = 0.996$, and Gentle: $p = 0.296$), and there was no evidence of a rank effect. For Confident, only the variable contrasting the 6/- and 6/6 genotype with the others was significant (B = −0.19, $p = 0.001$); the 5/- and 5/5 genotype dummy variable also had a negative coefficient, but was nonsignificant ($p = 0.068$), indicating that it was the 6/- and 6/6 animals that had low values for Confidence compared to animals with other genotypes. By contrast, for Gentle temperament, both *MAOA-LPR* dummy variables were significant with negative coefficients (5/- and 5/5 vs. others: $B = -0.14$, $p = 0.011$; 6/- and 6/6 vs. others: $B = -0.13$, $p = 0.021$), suggesting that the reference group, those with 7/- and 7/7 genotypes, showed greater Gentle temperament, as was found in Analysis 3. Significant covariates in the models suggested that animals that weighed more at BBA testing, females, and conventionally-reared (not SPF) animals were more likely to be Confident. Similarly, conventionally-reared animals were also more likely to show Gentle temperament.

As indicated above, for Nervous and Vigilant, the model with covariates alone (M1) was significant. SPF animals were significantly more likely to be Nervous, and females and conventionally-reared animals were more likely to be Vigilant.

4.3.2 | Analysis 6: Influence of rank, MAOA-LPR, and 5-HTTLPR genotypes on BI

Summary

For BI, we found evidence of a genotype by genotype interaction, as well as an interaction of rank by *5-HTTLPR* genotype.

TABLE 8 Analysis 5, both genes, and temperament ratings

	Confident (Model 2)		Gentle (Model 2)		Nervous (Model 1)		Vigilant (Model 1)	
	B	95% CI	*B*	95% CI	*B*	95% CI	*B*	95% CI
Intercept	0.02	(−0.07, 0.11)	−0.03	(−0.12, 0.06)	0	(−0.06, 0.06)	−0.08***	(−0.15, −0.01)
Sex	−0.19*	(−0.28, −0.09)	0.09****	(0, 0.19)	−0.07	(−0.16, 0.03)	−0.16**	(−0.26, −0.06)
Age (in days)	0	(0, 0)	0	(−0.01, 0)	0	(−0.01, 0)	0	(−0.01, 0)
Weight (in kg)	0.35***	(0.04, 0.65)	0.09	(−0.22, 0.39)	−0.22	(−0.51, 0.08)	−0.08	(−0.4, 0.23)
Firstborn	0.08	(−0.03, 0.18)	0.03	(−0.07, 0.14)	−0.02	(−0.12, 0.08)	0.07	(−0.04, 0.17)
SPF status	−0.22*	(−0.33, −0.11)	−0.16**	(−0.27, −0.05)	0.19*	(0.09, 0.3)	−0.24*	(−0.36, −0.12)
Rank	−0.02	(−0.18, 0.14)	−0.04	(−0.2, 0.12)	-	-	-	-
S/S versus L/S & L/L	−0.01	(−0.22, 0.2)	−0.13	(−0.34, 0.07)	-	-	-	-
L/S versus S/S & L/L	0	(−0.1, 0.09)	0.03	(−0.07, 0.12)	-	-	-	-
5/5 or 5/- versus others	−0.1****	(−0.21, 0.01)	−0.14***	(−0.25, −0.03)	-	-	-	-
6/6 or 6/- versus others	−0.19**	(−0.3, −0.08)	−0.13***	(−0.24, −0.02)	-	-	-	-
Variance parameters								
Var (level 1)	0.76	(0.68, 0.84)	0.75	(0.68, 0.83)	0.77	(0.69, 0.85)	0.81	(0.73, 0.9)
Var (dam ID)	0.15	(0.09, 0.24)	0.16	(0.1, 0.24)	0.1	(0.05, 0.19)	0.16	(0.1, 0.26)
Var (sir ID)	0.11	(0, 0.17)	0.09	(0, 0.15)	0.07	(0.04, 0.13)	0.12	(0.08, 0.18)

Note: Coefficient estimates and 95% confidence intervals (CIs) for cross-classified multilevel models (best models) testing the influence of rank, HTTLPR genotype, and MAOA-LPR genotype on the temperament ratings.

*$p < 0.001$.; **$p < 0.01$.; ***$p < 0.05$.; ****$p < 0.10$.

Model fits

M1, which included only the covariates, fit the data better than the null model ($\chi^2(5) = 52.69$, $p < 0.001$), and M2 and M3 both had better model fits than the preceding models (M2: $\chi^2(5) = 37.56$, $p < 0.001$; M3: $\chi^2(8) = 24.30$, $p = 0.002$). Table 9 presents the final model, and Table S15 presents results for all models.

Best model

Inspection of M3 revealed a genotype interaction of the S/S *5-HTTLPR* genotype with both *MAOA-LPR* genotypes (S/S * 5/5 or 5/-: mean = −2.16, $p = 0.011$; S/S * 6/6 or 6/-: mean = −1.58, $p = 0.020$), suggesting the effect was with the 7/7 and 7/- *MAOA-LPR* genotype: animals with an S/S genotype for *5-HTTLPR* and who also had the 7/7 or 7/- genotype for *MAOA-LPR* were more likely to be behaviorally inhibited. We also found a barely-significant interaction of the S/S genotype for *5-HTTLPR* and rank (mean = 1.98, $p = 0.049$), suggesting that for animals with the S/S genotype, animals of higher rank were more likely, and animals of lower rank were less likely, to be behaviorally inhibited. We note, however, that the omnibus test for the *MAOA-LPR/5-HTTLPR* genotype interaction was not significant ($\chi^2(4) = 6.27$, $p = 0.180$), nor was the omnibus test for the interaction of *5-HTTLPR* by rank ($\chi^2(2) = 3.03$, $p = 0.219$); consequently, we present these results with caution. Finally, as we found in Analyses 2 and 4, the odds of being behaviorally inhibited were higher for males and for SPF-reared animals.

5 | DISCUSSION

Our results indicate that infant genotype influences measures of temperament in infant rhesus monkeys, but that maternal rank had limited influence. We found no evidence of nonlinear effects of rank, but did find evidence that, for *MAOA-LPR*, the two alleles with similar transcriptional efficiency had different phenotypic outcomes. Finally, we found limited evidence that the two genes of interest to us in this study had combined effects on our measure of BI.

5.1 | Influence of maternal rank on infant temperament

While many studies have examined the role of maternal rank on infant-mother interactions and infant development (e.g., Arlet et al., 2019; Lee, 1984; I. Tanaka, 1989; Tartabini et al., 1980), very little is known about how rank might affect temperament in infant nonhuman primates. The principal exception is a study published by Suarez-Jimenez et al. (2013), who contrasted infants of high/alpha rank ($n = 8$) with those who were low/middle ranked ($n = 18$). All animals lived in small social groups (1 adult male, 6–8 females plus offspring). Suarez-Jimenez et al. (2013) showed that infants of high-ranked mothers were more affectively reactive in a human intruder test at around 3 months of age, and spent more time away from mothers in the home cage when animals were 4–5 months of age.

TABLE 9 Analysis 6, both genes, and behavioral inhibition

	Model 3	
	B	95% CI
Intercept	−1.66*	(−1.35, −2.02)
Sex	0.49*	(0.78, 0.21)
Age (in days)	0	(0.01, −0.01)
Weight (in kg)	−0.03	(0.84, −0.89)
Firstborn	−0.13	(0.17, −0.43)
SPF status	0.34***	(0.64, 0.05)
Rank	−0.14	(0.65, −0.93)
S/S versus L/S & L/L (dummy A1)	0.56	(1.58, −0.54)
L/S versus S/S & L/L (dummy A2)	0.07	(0.51, −0.35)
5/5 or 5/- versus others (dummy B1)	−0.04	(0.36, −0.43)
6/6 or 6/- versus others (dummy B2)	0.3****	(0.7, −0.09)
Rank × dummy A1	−2.17***	(−0.25, −4.39)
Rank × dummy A2	−1.58***	(−0.06, −3.14)
Rank × dummy B1	0	(0.63, −0.66)
Rank × dummy B2	−0.15	(0.48, −0.79)
Dummy A1 × dummy B1	1.98***	(4.55, −0.38)
Dummy A1 × dummy B2	−0.21	(0.72, −1.15)
Dummy A2 × dummy B1	0.72****	(1.83, −0.36)
Dummy A2 × dummy B2	0.36	(1.4, −0.7)
Variance parameters		
Var (dam ID)	0.3	(0.04, 0.96)
Var (sire ID)	0.45	(0.13, 0.87)

Note: Coefficient estimates and 95% CIs for cross-classified multilevel logistic model (best model) testing the influence of rank, HTTLPR genotype, and MAOA-LPR genotype on behavioral inhibition.
*$p < 0.001$.; **$p < 0.01$.; ***$p < 0.05$.; ****$p < 0.10$.

The authors suggest (p. 72) "that a more engaged or exuberant temperament may be associated with high maternal dominance and a more reserved or inhibited temperament may be associated with lower dominance rank." Our results show a somewhat different picture, with higher rank being associated with Confident temperament (a construct similar to the description offered by Suarez-Jimenez et al., 2013 [p. 70] for their results), but only for animals with particular genotypes. Moreover, our two-gene analysis (Analysis 6) of BI showed an effect of rank, but only for S/S animals, and the effect was opposite to that suggested by Suarez-Jimenez et al. (2013). It's possible that differences in the social conditions, operationalization of rank, measurement of temperament, and/or sampling issues contributed to the different results.

The principal effect of rank (in the sense that our model fit and omnibus tests were all significant) found in our analyses was in Analysis 3, and it was an interaction with *MAOA-LPR* genotype; it was not a main effect. Specifically, rank was positively associated with Confident temperament, but only for animals with a 5/5 or 5/- *MAOA-LPR* genotype. This was unexpected; functionally (based on in vitro work), the 5- and 6-repeat alleles for this gene have been found to have similarly high transcriptional efficiency, compared to the 7-repeat allele (Newman et al., 2005), yet in the present study, the interaction of rank with the 5-repeat genotypes was significant ($p = 0.010$) and the interaction with the 6-repeat genotypes was clearly not ($p = 0.642$). We are aware of no study of nonhuman primates in which behavioral or physiological differences have been found between animals possessing different high-activity alleles. It is becoming increasingly clear, however, that the relationship between *MAOA-LPR* genotype and phenotypic outcomes are more complex than originally thought. For example, attempts to replicate the classic study by Caspi's group (Caspi et al., 2002) that showed that maltreated children with the low activity variant were more likely to develop conduct disorder than were their peers with the high activity alleles, have not always been successful (Haberstick et al., 2005; Young et al., 2006). In addition, when in vivo methods have been used to determine functionality of the *MAOA-LPR* alleles, the low- and high-activity variants have sometimes not been found to differ (Jones et al., 2020; Shumay et al., 2012). Finally, evidence is accumulating that methylation of the *MAOA* promoter may play a greater role in some outcomes than genotype itself (Shumay et al., 2012; Ziegler et al., 2016). Perhaps the 5-repeat and 6-repeat alleles for *MAOA-LPR* in rhesus monkeys provide different opportunities for methylation. Clearly, more work is needed to understand the relationship between *MAOA* genotype and functional activity, and the discrepancies between in vitro and in vivo measures of functionality. Nevertheless, our large sample size (Table 2 indicates that, for Analysis 3, we had $n = 654$ animals in our sample with the 5-repeat genotype) suggests that this is not a sampling issue and may provide the basis for further investigation.

5.2 | Influence of genotype on infant temperament

Infant genotype was associated with all measures of temperament. Interestingly, genotype for *5-HTTLPR* was related to BI, whereas variation in *MAOA-LPR* predicted the rating-derived measures of temperament. We discuss each set of results in turn.

5.2.1 | *5-HTTLPR*

Analysis 2 indicated that monkeys with the S/S genotype ($n = 171$) were significantly less likely to be classified as behaviorally inhibited compared to animals with the L/S or L/L genotypes. This result contrasts with that found by Bethea et al. (2004) for infant and juvenile rhesus monkeys—in a free play situation in a novel environment, S/S animals showed reduced activity compared to the other genotypes, but showed more affective behavior (fear grimaces and lipsmacks) when challenged. Recall that our definition of BI included scores below the mean for activity and emotionality at the

beginning and end of testing. The differences may be due to sample size (Bethea et al., 2004 had $n = 15$ animals with the S/S genotype), age (animals in our study were younger than many of the animals in the Bethea et al., 2004, study), and/or testing conditions. Our results also differ from a second study, involving $n = 173$ young rhesus monkeys ($n = 13$ animals with S/S) that showed no relationship between genotype and a measure of BI from a human intruder test (Rogers et al., 2008). For humans, a review by Clauss et al. (2015) reports mixed results for whether S/S genotype is associated with BI. While there is a good consensus that *5-HTTLPR* in human and nonhuman primates can interact with adverse or beneficial outcomes to influence BI-associated measures, our study supports the idea that, with sufficient sample size, there is a main effect of this genotype on BI.

One result worth noting in this analysis was that the other *5-HTTLPR* dummy variable, which coded for L/S genotype versus other genotypes, was not a significant predictor of BI. This is important because studies often combine animals with S/S and L/S genotypes and contrast them with L/L animals. This is done because of the relative scarcity of the S/S genotype typically resulting in very small cell sizes. Our analysis does not support this practice, at least for the measure of BI—the S/S animals ($n = 171$) were different from the other genotypes, and despite the larger sample size for L/S animals ($n = 1095$), the L/S versus others contrast was not significant. We have seen in other studies, however, that for some measures, the L/S animals resemble the L/L animals, and for other measures, even within the same study, the L/S animals are statistically intermediate between L/L and S/S animals (Sorenson et al., 2013). We suggest that combining these two genotypes be done with extreme caution, and urge oversampling, if possible, of the S/S genotype to keep the three groups distinct.

5.2.2 | *MAOA-LPR*

Analysis 3 revealed that *MAOA-LPR* was significantly associated with all four ratings-derived measures of temperament: Model 2 was the final model for Gentle, Nervous, and Vigilant, and Model 3 was the final model for Confident (Model 3 showed a significant interaction between rank and the 5-repeat genotype, which was discussed above; here we focus on the main effects of *MAOA-LPR*). For each of the four measures, both dummy variables coding for this genotype were significant and of the same sign, suggesting that it was the reference category, the 7/7 females and 7/- males, that drove this result. Specifically, animals with the 7-repeat allele(s) were more likely to show Confident, Gentle, and Vigilant temperament, and were less likely to be Nervous.

The fact that this genotype was influential for all four measures of temperament suggests that there may be a common, underlying factor to all four measures that is being influenced by the gene. In fact, there is substantial shared variance among these four measures: correlation coefficients in the full BBA sample ($n = 5005$) range from 0.513 to 0.744 in absolute value, with Nervous correlating negatively with the other three measures, all of which correlate positively with each other. During the original exploratory and confirmatory factor analyses of the rating data (Golub et al., 2009), an oblique rotation was used, permitting the derived factors to correlate (we note that human temperament factors are also correlated: Beekman et al., 2015). Our rationale for this analysis of measures in the affective domain paralleled that used in the understanding of intellectual development in humans in the cognitive domain, namely, that development proceeds by differentiating existing competences into more specific characteristics (e.g., Garrett, 1946). In some of our analyses, all four traits have been important contributors to a phenomenon (e.g., diarrhea: Gottlieb et al., 2018) suggesting, as in the present case, that some single overarching factor is being tapped into. In many other analyses, however, only single temperament factors show significant relationships to a phenomenon (e.g., Nervous temperament and pairing success: Capitanio et al., 2017; Vigilant temperament and asthma-related outcomes: Capitanio, Miller, et al., 2011), indicating that, by 3–4 months of age, some differentiation of the single overarching factor had proceeded sufficiently to permit the unique variance associated with a specific trait to become associated with some later behavioral or health measures. Our current results suggest that the 7-repeat allele of *MAOA-LPR* is influential for the overarching factor, whose qualities remain unknown at this time, but likely reflect a broader characteristic such as affective reactivity.

5.2.3 | 5-HTTLPR and MAOA-LPR

Following Belsky and Beaver (2011), we examined the potential joint influence of the two genes to determine if there was an additive effect for individuals with the two "plasticity" alleles – S/S for *5-HTTLPR* and 7/7 or 7/- for *MAOA-LPR*. We found no evidence for the ratings-derived measures, but our analysis of BI provided some support for the idea. Model 3, which included main effects of genes and rank as well as the first-order interactions, was the best fitting model. But we found that, despite a nonsignificant omnibus test, the coefficients for the two-way interactions of S/S with the 5-repeat and the 6-repeat alleles were significant: animals with the S/S genotype for *5-HTTLPR* and who also possessed the 7/7 or 7/- genotype for *MAOA-LPR* were more likely to be behaviorally inhibited. We view this result with some caution. Recall that Analysis 2 showed that *5-HTTLPR* influenced BI, such that animals with the S/S genotype were less likely to be behaviorally inhibited. Analysis 4, focusing on *MAOA-LPR* and BI, showed no significant effects of genotype. The two-gene analysis, however, suggested that, in the presence of the *MAOA-LPR* plasticity genotype, the effect of the S/S genotype reverses: animals with the S/S genotype now are more likely to be behaviorally inhibited. Our concern over this result derives from the small sample size; despite an overall sample size of $n = 2017$ for this analysis, there are only 28 animals that possess both the S/S and the 7-repeat genotypes, only eight of which are behaviorally inhibited. The rate of BI among the 28 animals

(8/28 = 0.286) is higher than, but not significantly different from, the rate for the overall sample (454/2017 = 0.225). Nevertheless, this is a uniquely large data set for such an analysis, and so we conclude, with caution, that this analysis provides some support for the idea of joint influence of two plasticity genes, and Belsky and Beaver's (2011) thesis.

5.3 | Influence of covariates on infant temperament

For every analysis, five covariates were included that prior research had suggested might influence temperament. Of the five, sex and SPF status appeared to be consistently related, across the various analyses, to temperament.

5.3.1 | Sex

We found that males were more likely to show Gentle temperament and to be behaviorally inhibited, whereas females were more likely to be Confident, Nervous, and Vigilant. Gender differences in studies of human personality are common (Costa et al., 2001). For example, females typically show higher levels of Neuroticism than males; elsewhere (Capitanio, Mendoza, et al., 2011), we have argued that Nervous temperament is very similar to the human Neuroticism factor, and our current study shows a parallel sex difference in that measure. In contrast, while we found that males were more likely to be behaviorally inhibited than females, the literature shows a very mixed picture: In humans, gender differences in BI/shyness are rare in young children, but become more evident in later childhood, where girls show a higher prevalence compared to boys (Doey et al., 2014; see also Assari, 2020). In contrast, in rats, males show more inhibition than females (Ray & Hansen, 2004), and Kalin and Shelton (1998) report no sex differences in freezing in response to a human intruder (their measure of BI) in rhesus monkeys a year of age and under.

We also found that females had higher values for Confident and Vigilant temperament, and lower levels of Gentle temperament. Confident, also known as Bold, temperament, is a commonly found dimension of animal temperament (Gosling, 2001), generally reflecting assertiveness. Vigilant temperament is a somewhat unique dimension, focusing on calm, watchful, wariness (i.e., animals score high on the individual item vigilant, and low on the items depressed, tense, timid), and is an important part of a composite measure that is associated with increased risk for hyperresponsive airways, a component of asthma (Capitanio, Miller, et al., 2011; Chun et al., 2013), and persisting affective responses during challenge (Chun & Capitanio, 2016). For Gentle temperament, evidence is accumulating that this dimension reflects a coping style, with high scores reflecting reactive coping, and lower scores reflecting proactive coping (see Koolhaas et al., 1999). For example, when older, animals scoring higher on this factor show more silent bared-teeth displays and withdrawal in their natal field corrals (Beisner & McCowan, 2014) and when relocated indoors have a higher risk of developing motor stereotypies (Gottlieb et al., 2013).

Unfortunately, there is a dearth of information in the literature about sex differences in temperament of young animals in nonhuman primates, with two exceptions, one of which is the research into BI described above. The other exception involves a pair of studies that used a Neonatal Behavioral Assessment Scale. One the one hand, Coe et al. (2010), showed a sex difference at 2 weeks of age in "State Control;" female rhesus monkeys were more reactive than males. Using the same instrument with a much larger sample ($n = 1056$), however, Paukner et al. (2020) found that sex was not a significant predictor of temperament at 7 days of age, nor of the change across the first month of life. Our study, then, with its large sample size of infants reared under relatively naturalistic conditions, suggests new directions to be explored for understanding sex differences in temperament.

5.3.2 | SPF status

At our facility, SPF animals were derived by removing infants born into our half-acre field corrals, typically on the day of birth. Animals were relocated to an indoor nursery, where they were individually housed in incubators until 3 weeks of age, at which point they were given visual access to an infant of the same age with whom they were subsequently paired at 5 weeks of age. Animals then were formed into larger and larger groups, ultimately being relocated with other SPF animals into a field corral. Once an SPF corral was formed, offspring remained with their mothers (but were tested repeatedly to insure their pathogen-free status). We are aware of only two comparisons between SPF and non-SPF animals, and they revealed a number of immunological differences (Oxford et al., 2015), as well as reduced mortality for SPF animals (T. Tanaka et al., 2013) compared to non-SPF animals. Elsewhere, however, we have reported results that are relevant for this discussion. Kinnally et al. (2018) reported that infant monkeys that were born and reared for their first 3–4 months of life in the field corrals, differed depending on whether their fathers, grandfathers, and/or great-grandfathers were nursery-reared (interestingly, there were no effects due to female progenitors). Among the results reported was that infants with a male progenitor that was nursery-reared had significantly higher levels of Nervous temperament. Most of the $n = 340$ animals from the Kinnally et al. (2018) study were part of the current study, so it is no surprise that our analyses, with nearly 10 times the number of subjects, confirmed that SPF animals also had higher levels of Nervous temperament. SPF animals also were likely to be rated lower for Confident, Gentle, and Vigilant temperament, but had higher values for BI. Together, these data suggest that SPF monkeys, derived via nursery-rearing, differ from conventionally reared animals in multiple ways. The mechanism behind this is unclear. It is possible, for example, that the nursery-rearing experience of the male progenitors left some epigenetic marks on their gametes that influenced brain and behavioral development. Alternatively, it's

possible that these outcomes reflect a natural process of development among animals that are free of multiple endemic viruses, with attendant alterations in immune functioning. Understanding the mechanism remains an important direction for future research.

5.4 | Summary and conclusions

Our goal was to determine if maternal rank and infant genotype influenced temperament in infant rhesus monkeys. We found no evidence of a main effect of rank; rather rank was associated with Confident temperament only for those infants with the 5-repeat genotype for *MAOA-LPR*. Several results were found suggesting genetic influences: animals with the S/S genotype for *5-HTTLPR* were less likely to be behaviorally inhibited, unless they also possessed the 7-repeat genotype for *MAOA-LPR*, in which case they were more likely to be inhibited. Importantly, we found two situations in which supposedly functionally-identical genotypes for *MAOA-LPR* (based on in vitro assessments of functionality: Newman et al., 2005) differed phenotypically—the aforementioned interaction for Confident temperament between rank and the 5-repeat allele, and the finding in Analysis 5 that the animals with the 6-repeat allele for *MAOA-LPR* were more likely to be low in Confident temperament. In addition, while we found some influence of the S/S genotype on BI, we found no effects of the L/S genotype. Together, these results do not support the combining of the 5-repeat and 6-repeat genotypes for *MAOA-LPR*, nor the combining of the S/S and L/S genotypes for *5-HTTLPR*. Our analysis also provides some of the first evidence of sex differences in temperament of infant monkeys, and, to our knowledge, the only evidence of differences in temperament based on SPF status. Finally, our analysis provides some support for the idea of a "dose-response" effect of plasticity alleles, as suggested by Belsky and Beaver (2011). The large sample sizes of our analyses provide confidence in our results, and our findings suggest several directions to explore in future research.

ACKNOWLEDGMENTS

The authors thank Laura Calonder for assisting with data collection; Ashley Cameron, Heather Cook, Lindsay Tatum, and Ashleigh Day for providing colony rank data; Jeff Rogers, Shelley Blozis, Tom Famula, and Judy Quan for statistical advice; and the animal care and veterinary staff at CNPRC for excellent care of the animals. The BioBehavioral Assessment program has been funded by R24OD010962 and P51OD011107.

CONFLICTS OF INTEREST

The authors declare no conflicts of interest.

DATA AVAILABILITY STATEMENT

Raw data and SPSS and Stata script files to reproduce the findings are available on the OSF: https://osf.io/untrc/?view_only=7469c7618d5840f0a68533f57269e837

ORCID

John P. Capitanio http://orcid.org/0000-0002-3680-1323
Nicolas Sommet http://orcid.org/0000-0001-8585-1274

REFERENCES

Andrade, M. C., Penedo, M. C., Ward, T., Silva, V. F., Bertolini, L. R., Roberts, J. A., Leite, J. P., & Cabello, P. H. (2004). Determination of genetic status in a closed colony of rhesus monkeys (*Macaca mulatta*). *Primates*, *45*(3), 183–186. https://doi.org/10.1007/s10329-004-0084-x

Arend, M. G., & Schäfer, T. (2019). Statistical power in two-level models: A tutorial based on Monte Carlo simulation. *Psychological Methods*, *24*, 1–19.

Arlet, M., Veromann-Jürgenson, L.-L., Isbell, L., Mänd, R., & Lemasson, A. (2019). Maternal care in free-ranging arboreal grey-cheeked mangabeys (Lophocebus albigena johnstoni) in Kibale National Park, Uganda. *Folia Primatologia*, *90*, 441–455.

Assari, S. (2020). Sex differences in the association between cortical thickness and children's behavioral inhibition. *Journal of Psychological and Behavioral Research*, *2*(2), 49–64. https://doi.org/10.22158/jpbr.v2n2p49

Barr, C. S. (2012). Temperament in animals. In M. Zentner, & R. L. Shiner (Eds.), *Handbook of temperament* (pp. 251–272). Guilford Press.

Beekman, C., Neiderhiser, J. M., Buss, K. A., Loken, E., Moore, G. A., Leve, L. D., Ganiban, J. M., Shaw, D. S., & Reiss, D. (2015). The development of early profiles of temperament: Characterization, continuity, and etiology. *Child Development*, *86*, 1794–1811.

Beisner, B. A., & McCowan, B. (2014). Signaling context modulates social function of silent bared-teeth displays in rhesus macaques (*Macaca mulatta*). *American Journal of Primatology*, *76*(2), 111–121.

Belsky, J., & Beaver, K. M. (2011). Cumulative-genetic plasticity, parenting and adolescent self-regulation. *Journal of Child Psychology and Psychiatry*, *52*(5), 619–626. https://doi.org/10.1111/j.1469-7610.2010.02327.x.

Belsky, J., & Pluess, M. (2009). The nature (and nurture?) of plasticity in early human development. *Perspectives on Psychological Science*, *4*, 345–351.

Bernstein, I. S., & Williams, L. E. (1983). Ontogenetic changes and the stability of rhesus monkey dominance relationships? *Behavioural Processes*, *8*, 379–392.

Bethea, C. L., Streicher, J. M., Coleman, K., Pau, F. K., Moessner, R., & Cameron, J. L. (2004). Anxious behavior and fenfluramine-induced prolactin secretion in young rhesus macaques with different alleles of the serotonin reuptake transporter polymorphism (5HTTLPR). *Behavior Genetics*, *34*(3), 295–307. https://doi.org/10.1023/B:BEGE.0000017873.61607.be

Blomquist, G. E., Hinde, K., & Capitanio, J. P. (2021). Inheritance of hormonal stress response and temperament in infant rhesus macaques (*Macaca mulatta*): Nonadditive and sex-specific effects. *Behavioral Neuroscience*, *136*, 61–71. https://doi.org/10.1037/bne0000493

Brand, S. R., Engel, S. M., Canfield, R. L., & Yehuda, R. (2006). The effect of maternal PTSD following *in utero* trauma exposure on behavior and temperament in the 9-month-old infant. *Annals of the New York Academy of Science*, *1071*, 454–458.

Burnham, K. P., Anderson, D. R., & Huyvaert, K. P. (2011). AIC model selection and multimodel inference in behavioral ecology: Some background, observations, and comparisons. *Behavioral Ecology and Sociobiology*, *65*, 23–35.

Capitanio, J. P. (2008). Personality and disease. *Brain, Behavior, and Immunity*, *22*, 647–650.

Capitanio, J. P. (2017). Variation in biobehavioral organization. In S. Schapiro (Ed.), *Handbook of primate behavioral management* (pp. 55–73). CRC Press.

Capitanio, J. P. (2019). Behavioral inhibition in nonhuman primates: The elephant in the room. In K. Pérez-Edgar, & N. A. Fox (Eds.), *Behavioral inhibition* (pp. 17–33). Springer. https://doi.org/10.1007/978-3-319-98077-5_2

Capitanio, J. P. (2022). Knowledge of biobehavioral organization can facilitate better science: A review of the BioBehavioral Assessment Program at the California National Primate Research Center. *Animals: An Open Access Journal from MDPI*, *11*, 2445. https://doi.org/10.3390/ani11082445

Capitanio, J. P., Blozis, S. A., Snarr, J., Steward, A., & McCowan, B. J. (2017). Do "birds of a feather flock together" or do "opposites attract"? Behavioral responses and temperament predict success in pairings of rhesus monkeys in a laboratory setting. *American Journal of Primatology*, *79*, e22464.

Capitanio, J. P., Mendoza, S. P., & Cole, S. W. (2011). Nervous temperament in infant monkeys is associated with reduced sensitivity of leukocytes to cortisol's influence on trafficking. *Brain, Behavior, and Immunity*, *25*, 151–159.

Capitanio, J. P., Miller, L. A., Schelegle, E. S., Mendoza, S. P., Mason, W. A., & Hyde, D. M. (2011). Behavioral inhibition is associated with airway hyper-responsiveness but not atopy in a monkey model of asthma. *Psychosomatic Medicine*, *73*, 288–294.

Caspi, A., McClay, J., Moffitt, T. E., Mill, J., Martin, J., Craig, I. W., Taylor, A., & Poulton, R. (2002). Role of genotype in the cycle of violence in maltreated children. *Science*, *297*(5582), 851–854.

Champoux, M., Bennett, A., Shannon, C., Higley, J. D., Lesch, K. P., & Suomi, S. J. (2002). Serotonin transporter gene polymorphism, differential early rearing, and behavior in rhesus monkey neonates. *Molecular Psychiatry*, *7*, 1058–1063.

Chun, K., & Capitanio, J. P. (2016). Developmental consequences of behavioral inhibition: A model in rhesus monkeys (*Macaca mulatta*). *Developmental Science*, *19*, 1035–1048.

Chun, K., Miller, L. A., Schelegle, E. S., Hyde, D. M., & Capitanio, J. P. (2013). Behavioral inhibition in rhesus monkeys (*Macaca mulatta*) is related to the airways response, but not immune measures, commonly associated with asthma. *PLoS One*, *8*(8), e71575. https://doi.org/10.1371/journal.pone.0071575.

Clarke, A., & Schneider, M. L. (1993). Prenatal stress has long-term effects on behavioral responses to stress in juvenile rhesus monkeys. *Developmental Psychobiology*, *26*, 293–304.

Clauss, J. A., Avery, S. N., & Blackford, J. U. (2015). The nature of individual differences in inhibited temperament and risk for psychiatric disease: A review and meta-analysis. *Progress in Neurobiology*, *127-128*, 23–45. https://doi.org/10.1016/j.pneurobio.2015.03.001

Coe, C. L., Kramer, M., Czeh, B., Gould, E., Reeves, A. J., Kirschbaum, C., & Fuchs, E. (2003). Prenatal stress diminishes neurogenesis in the dentate gyrus of juvenile rhesus monkeys. *Biological Psychiatry*, *54*, 1025–1034.

Coe, C. L., Lubach, G. R., Crispen, H. R., Shirtcliff, E. A., & Schneider, M. L. (2010). Challenges to maternal wellbeing during pregnancy impact temperament, attention, and neuromotor responses in the infant rhesus monkey. *Developmental Psychobiology*, *52*(7), 625–637. https://doi.org/10.1002/dev.20489

Costa, P. T., Jr., Terracciano, A., & McCrae, R. R. (2001). Gender differences in personality traits across cultures: Robust and surprising findings. *Journal of Personality and Social Psychology*, *81*, 322–331. https://doi.org/10.1037//0022-3514.81.2.322

Doey, L., Coplan, R. J., & Kingsbury, M. (2014). Bashful boys and coy girls: A review of gender differences in childhood shyness. *Sex Roles*, *70*, 255–266. https://doi.org/10.1007/s11199-013-0317-9

Dunn, E. C., Richmond, T. K., Milliren, C. E., & Subramanian, S. V. (2015). Using cross-classified multilevel models to disentangle school and neighborhood effects: An example focusing on smoking behaviors among adolescents in the United States. *Health & Place*, *31*, 224–232.

Elfenbein, H. A., Del Rosso, L., McCowan, B., & Capitanio, J. P. (2016). Effect of indoor compared with outdoor location during gestation on the incidence of diarrhea in indoor-reared rhesus macaques (*Macaca mulatta*). *Journal of AALAS*, *55*, 277–290.

Ellis, B. J., Boyce, W. T., Belsky, J., Bakermans-Kranenburg, M. J., & Van Ijzendoorn, M. H. (2011). Differential susceptibility to the environment: An evolutionary–neurodevelopmental theory. *Development and Psychopathology*, *23*, 7–28.

Fan, M., Liu, B., Jiang, T., Jiang, X., Zhao, H., & Zhang, J. (2010). Meta-analysis of the association between the monoamine oxidase-A gene and mood disorders. *Psychiatric Genetics*, *20*(1), 1–7.

Fox, A. S., Harris, R. A., Del Rosso, L., Raveendran, M., Kamboj, S., Kinnally, E. L., Capitanio, J. P., & Rogers, J. (2021). Infant inhibited temperament in primates predicts adult behavior, is heritable, and is associated with anxiety-relevant genetic variation. *Molecular Psychiatry*, *26*, 6609–6618. https://doi.org/10.1038/s41380-021-01156-4

Garrett, H. E. (1946). A developmental theory of intelligence. *American Psychologist*, *1*(9), 372–378. https://doi.org/10.1037/h0056380

Golub, M. S., Hogrefe, C. E., Widaman, K. F., & Capitanio, J. P. (2009). Iron deficiency anemia and affective response in rhesus monkey infants. *Developmental Psychobiology*, *51*, 47–59.

Gosling, S. D. (2001). From mice to men: What can we learn about personality from animal research? *Psychological Bulletin*, *127*, 45–86. https://doi.org/10.1037//0033-2909.127.1.45

Gottlieb, D. H., Capitanio, J. P., & McCowan, B. (2013). Risk factors for stereotypic behavior and self-biting in rhesus macaques (*Macaca mulatta*): Animal's history, current environment, and personality. *American Journal of Primatology*, *75*, 995–1008.

Gottlieb, D. H., Del Rosso, L., Sheikhi, F., Gottlieb, A., McCowan, B., & Capitanio, J. P. (2018). Personality, environmental stressors, and diarrhea in rhesus macaques: An interactionist perspective. *American Journal of Primatology*, *80*(12), e22908. https://doi.org/10.1002/ajp.22908

Haberstick, B. C., Lessem, J. M., Hopfer, C. J., Smolen, A., Ehringer, M. A., Timberlake, D., & Hewitt, J. K. (2005). Monoamine oxidase A (MAOA) and antisocial behaviors in the presence of childhood and adolescent maltreatment. *American Journal of Medical Genetics B: Neuropsychiatric Genetics*, *5*, 59–64. https://doi.org/10.1002/ajmg.b.30176

Heck, R. H., Thomas, S. L., & Tabata, L. N. (2014). *Multilevel and longitudinal modeling with IBM SPSS*. Routledge.

Herrington, J. A., Del Rosso, L., & Capitanio, J. P. (2016). Biobehavioral consequences of prenatal exposure to a matrilineal overthrow and relocation in captive infant rhesus (*Macaca mulatta*) monkeys. *American Journal of Primatology*, *78*, 895–903.

Hox, J. J., Moerbeek, M., & Van de Schoot, R. (2017). *Multilevel analysis: Techniques and applications*. Routledge.

Huang, F. L. (2018). Multilevel modeling myths. *School Psychology Quarterly*, *33*(3), 492–499. https://doi.org/10.1037/spq0000272

Huizink, A. C. (2012). Prenatal factors in temperament: The role of prenatal stress and substance use exposure. In M. Zentner, & R. L. Shiner (Eds.), *Handbook of temperament* (pp. 297–314). Guilford Press.

Jones, D. N., Ruiz, C. A., Raghanti, M. A., Tosi, A. J., Tanaka, H., & Goto, Y. (2020). Monoamine oxidase polymorphisms in rhesus and Japanese macaques (*Macaca mulatta* and *M. fuscata*). *Journal of Chemical Neuroanatomy*, *103*, 101726. https://doi.org/10.1016/j.jchemneu.2019.101726

Kagan, J. (1994). *Galen's prophecy*. Basic Books.

Kalin, N. H., & Shelton, S. E. (1998). Ontogeny and stability of separation and threat-induced defensive behaviors in rhesus monkeys during the first year of life. *American Journal of Primatology*, *44*, 125–135.

Kanthaswamy, S., von Dollen, A., Kurushima, J. D., Alminas, O., Rogers, J., Ferguson, B., Lerche, N. W., Allen, P. C., & Smith, D. G. (2006). Microsatellite markers for standardized genetic management of captive colonies of rhesus macaques (*Macaca mulatta*). *American Journal of Primatology*, *68*(1), 73–95. https://doi.org/10.1002/ajp.20207

Karere, G. M., Kinnally, E. L., Sanchez, J. N., Famula, T. R., Lyons, L. A., & Capitanio, J. P. (2009). What is an "adverse" environment? Interactions of rearing experiences and MAOA genotype in rhesus monkeys. *Biological Psychiatry*, *65*, 770–777.

Karere, G. M., Sullivan, E., Kinnally, E. L., Capitanio, J. P., & Lyons, L. A. (2012). Enhancing genotyping of MAOA and 5-HTT in rhesus macaques (*Macaca mulatta*). *Journal of Medical Primatology*, *41*, 407–411.

Kinnally, E. L., Gonzalez, M. N., & Capitanio, J. P. (2018). Paternal line effects of early experiences persist across three generations in rhesus macaques. *Developmental Psychobiology*, *60*(8), 879–888. https://doi.org/10.1002/dev.21771

Kohn, J. N., Snyder-Mackler, N., Barreiro, L. B., Johnson, Z. P., Tung, J., & Wilson, M. E. (2016). Dominance rank causally affects personality and glucocorticoid regulation in female rhesus macaques. *Psychoneuroendocrinology*, *74*, 179–188.

Koolhaas, J. M., Korte, S. M., De Boer, S. F., Van Der Vegt, B. J., Van Reenen, C. G., Hopster, H., De Jonga, I. C., Ruis, M. A. W., & Blokhuis, H. J. (1999). Coping styles in animals: Current status in behavior and stress-physiology. *Neuroscience and Biobehavioral Reviews*, *23*, 925–935.

LaHuis, D. M., Hartman, M. J., Hakoyama, S., & Clark, P. C. (2014). Explained variance measures for multilevel models. *Organizational Research Methods*, *17*, 433–451.

Lee, P. C. (1984). Early infant development and maternal care in free-ranging vervet monkeys. *Primates*, *25*(1), 36–47.

Lesch, K. P., Meyer, J., Glatz, K., Flugge, G., Hinney, A., Hebebrand, J., Klauck, S. M., Poustka, A., Poustka, F., Bengel, D., Mossner, R., Riederer, P., & Heils, A. (1997). The 5-HT transporter gene-linked polymorphic region (5-HTTLPR) in evolutionary perspective: Alternative biallelic variation in rhesus monkeys. *Journal of Neural Transmission*, *104*, 1259–1266.

Musca, S. C., Kamiejski, R., Nugier, A., Méot, A., Er-Rafiy, A., & Brauer, M. (2011). Data with hierarchical structure: Impact of intraclass correlation and sample size on type-I error. *Frontiers in Psychology*, *2*, 74.

Newman, T. K., Syagailo, Y. V., Barr, C. S., Wendland, J. R., Champoux, M., Graessle, M., Suomi, S. J., Higley, J. D., & Lesch, K. P. (2005). Monoamine oxidase-A gene promoter variation and rearing experience influences aggressive behavior in rhesus monkeys. *Biological Psychiatry*, *57*, 167–172.

Noldus, L. P. J. J. (1991). The observer: A software system for collection and analysis of observational data. *Behavior Research Methods, Instruments, & Computers*, *23*, 415–429.

Oxford, K. L., Dela Pena-Ponce, M. G. A., Jensen, K., Eberhardt, M. K., Spinner, A., Van Rompay, K. K., Rigdon, J., Mollan, K. R., Krishnan, V. V., Hudgens, M. G., Barry, P. A., & De Paris, K. (2015). The interplay between immune maturation, age, chronic viral infection and environment. *Immunological Ageing*, *12*, 3. https://doi.org/10.1186/s12979-015-0030-3

Paukner, A., Capitanio, J. P., & Blozis, S. A. (2020). A new look at neurobehavioral development in rhesus monkey neonates (*Macaca mulatta*). *American Journal of Primatology*, *82*(5), e23122.

Ray, J., & Hansen, S. (2004). Temperament in the rat: Sex differences and hormonal influences on harm avoicance and novelty seeking. *Behavioral Neuroscience*, *118*, 488–497. https://doi.org/10.1037/0735-7044.118.3.488

Rendina, D. N., Lubach, G. R., & Coe, C. L. (2016). Gestational timing of prenatal disturbance and fetal sex determine the developmental outcomes. *Neonatology*, *109*, 314–320.

Rogers, J., Shelton, S. E., Shelledy, W., Garcia, R., & Kalin, N. H. (2008). Genetic influences on behavioral inhibition and anxiety in juvenile rhesus macaques. *Genes, Brain and Behavior*, *7*(4), 463–469. https://doi.org/10.1111/j.1601-183X.2007.00381.x

Rothbart, M. K., & Derryberry, D. (1981). Development of individual differences in temperament. In M. E. Lamb, & A. L. Brown (Eds.), *Advances in developmental psychology* (Vol. 1, pp. 37–86). Erlbaum.

Sabol, S. Z., Hu, S., & Hamer, D. (1998). A functional polymorphism in the monoamine oxidase-A gene promoter. *Human Genetics*, *103*, 273–279.

Saudino, K. J., & Wang, M. (2012). Quantitative and molecular genetic studies of temperament. In M. Zentner, & R. L. Shiner (Eds.), *Handbook of Temperament* (pp. 315–346). Guilford Press.

Schneider, M. L. (1992). Prenatal stress exposure alters postnatal behavioral expression under conditions of novelty challenge in rhesus monkey infants. *Developmental Psychobiology*, *25*, 529–540.

Shively, C. A. (1998). Social subordination stress, behavior, and central monoaminergic function in female cynomolgus monkeys. *Biological Psychiatry*, *44*(9), 882–891.

Shumay, E., Logan, J., Volkow, N. D., & Fowler, J. S. (2012). Evidence that the methylation state of the monoamine oxidase A (MAOA) gene predicts brain activity of MAO-A enzyme in healthy men. *Epigenetics*, *7*, 1151–1160. https://doi.org/10.4161/epi.21976

Snyder-Mackler, N., Sanz, J., Kohn, J. N., Brinkworth, J. F., Morrow, S., Shaver, A. O., Grenier, J. C., Pique-Regi, R., Johnson, Z. P., Wilson, M. E., Barreiro, L. B., & Tung, J. (2016). Social status alters immune regulation and response to infection in macaques. *Science*, *354*(6315), 1041–1045.

Snyder-Mackler, N., Sanz, J., Kohn, J. N., Voyles, T., Pique-Regi, R., Wilson, M. E., Barreiro, L. B., & Tung, J. (2019). Social status alters chromatin accessibility and the gene regulatory response to glucocorticoid stimulation in rhesus macaques. *Proceedings of the National Academies of Science of the United States of America*, *116*(4), 1219–1228.

Sommet, N., & Morselli, D. (2017). Keep calm and learn multilevel logistic modeling: A simplified three-step procedure using Stata, R, Mplus, and SPSS. *International Review of Social Psychology*, *30*(1), 203–218. https://doi.org/10.5334/irsp.90

Sommet, N., & Morselli, D. (2021). Keep calm and learn multilevel linear modeling: A three-step procedure using SPSS, Stata, R, and Mplus. *International Review of Social Psychology*, *34*(1), 24. https://doi.org/10.5334/irsp.555

Sorenson, A., Sullivan, E. C., Mendoza, S. P., Capitanio, J. P., & Higley, J. D. (2013). Serotonin transporter genotype modulates HPA axis output during stress: Effect of stress, dexamethasone test and ACTH challenge. *Translational Developmental Psychiatry*, *1*, 21130. https://doi.org/10.3402/tdp.v1i0.21130

Suarez-Jimenez, B., Hathaway, A., Waters, C., Vaughan, K., Suomi, S. J., Noble, P. L., Pine, D. S., Fox, N. A., & Nelson, E. E. (2013). Effect of mother's dominance rank on offspring temperament in infant rhesus monkeys (*Macaca mulatta*). *American Journal of Primatology*, *75*, 65–73.

Tamashiro, K. L., Sakai, R. R., Shively, C. A., Karatsoreos, I. N., & Reagan, L. P. (2011). Chronic stress, metabolism, and metabolic syndrome. *Stress*, *14*(5), 468–474.

Tanaka, I. (1989). Variability in the development of mother-infant relationships among free-ranging Japanese macaques. *Primates*, *30*, 477–491.

Tanaka, T., Lerche, N. W., Farver, T. B., Ardeshir, A., & Kass, P. H. (2013). Specific-pathogen-free status is associated with lower infant mortality rate in rhesus macaque (*Macaca mulatta*) colonies at the California National Primate Research Center. *Journal of Medical Primatology*, *42*(4), 186–191. https://doi.org/10.1111/jmp.12049

Tartabini, A., Genta, M. L., & Bertacchini, P. A. (1980). Mother-infant interaction and rank order in Rhesus monkeys (*Macaca mulatta*). *Journal of Human Evolution*, *9*, 139–146.

Taylor, S. E., Way, B. M., Welch, W. T., Hilmert, C. J., Lehman, B. J., & Eisenberger, N. I. (2006). Early family environment, current adversity, the serotonin transporter promoter polymorphism, and depressive symptomatology. *Biological Psychiatry*, *60*(7), 671–676.

Tees, M. T., Harville, E. W., Xiong, X., Buekens, P., Pridjian, G., & Elkind-Hirsch, K. (2010). Hurricane Katrina-related maternal stress, maternal mental health, and early infant temperament. *Maternal and Child Health Journal*, *14*, 511–518.

Vandeleest, J. J., McCowan, B., & Capitanio, J. P. (2011). Early rearing interacts with temperament and housing to influence the risk for motor stereotypy in rhesus monkeys (*Macaca mulatta*). *Applied Animal Behaviour Science*, *132*, 81–89.

Vaughn, B. E., Bradley, C. F., Joffe, L. S., Seifer, R., & Barglow, P. (1987). Maternal characteristics measured prenatally are predictive of ratings of temperamental "difficultly" on the Carey Infant Temperament Questionnaire. *Developmental Psychology*, *23*, 152–161.

Walker, C. K., VandeVoort, C. A., Li, C. -S., Chaffin, C. L., & Capitanio, J. P. (2018). Adiposity and weight gain during pregnancy associate independently with behavior of infant rhesus monkeys (*Macaca mulatta*). *Developmental Psychobiology*, *60*, 629–638.

Weinstein, T. A. R., Bales, K. L., Maninger, N., Hostetler, C. M., & Capitanio, J. P. (2014). Early involvement in friendships predicts later plasma concentrations of oxytocin and vasopressin in juvenile rhesus macaques (*Macaca mulatta*). *Frontiers in Behavioral Neuroscience*, *8*, 295.

Worlein, J. M., & Sackett, G. P. (1995). Maternal exposure to stress during pregnancy: Its significance for infant behavior in pigtail macaques (*Macaca nemestrina*). In C. R. Pryce, R. D. Martin, & D. Skuse (Eds.), *Motherhood in human and nonhuman primates* (pp. 142–151). Karger.

Yee, J. L., Vanderford, T. H., Didier, E. S., Gray, S., Lewis, A., Roberts, J., Taylor, K., & Bohm, R. P. (2016). Specific pathogen free macaque colonies: A review of principles and recent advances for viral testing and colony management. *Journal of Medical Primatology*, *45*, 55–78.

Young, S. E., Smolen, A., Hewitt, J. K., Haberstick, B. C., Stallings, M. C., Corley, R. P., & Crowley, T. J. (2006). Interaction between MAO-A genotype and maltreatment in the risk for conduct disorder: Failure to confirm in adolescent patients. *American Journal of Psychiatry*, *163*(6), 1019–1025. https://doi.org/10.1176/ajp.2006.163.6.1019

Ziegler, C., Richter, J., Mahr, M., Gajewska, A., Schiele, M. A., Gehrmann, A., Schmidt, B., Lesch, K. P., Lang, T., Helbig-Lang, S., Pauli, P., Kircher, T., Reif, A., Rief, W., Vossbeck-Elsebusch, A. N., Arolt, V., Wittchen, H. U., Hamm, A. O., Deckert, J., & Domschke, K. (2016). MAOA gene hypomethylation in panic disorder-reversibility of an epigenetic risk pattern by psychotherapy. *Translational Psychiatry*, *6*(4), e773. https://doi.org/10.1038/tp.2016.41

SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

How to cite this article: Capitanio, J. P., Sommet, N., & Del Rosso, L. (2022). The relationship of maternal rank, *5-HTTLPR* genotype, and *MAOA-LPR* genotype to temperament in infant rhesus monkeys (*Macaca mulatta*). *American Journal of Primatology*, e23374. https://doi.org/10.1002/ajp.23374