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Developing individual differences in primate behavior: The role of genes, environment and their interplay

Christoph J. von Borell [a, b *], Alexander Weiss [c,d], Lars Penke [a,b]

[a] Institute of Psychology, University of Goettingen, 37073 Göttingen, Germany

[b] Leibniz ScienceCampus Primate Cognition, 37077 Göttingen, Germany

[c] Department of Psychology, School of Philosophy, Psychology and Language Sciences, The University of Edinburgh, Edinburgh EH8 9JZ, United Kingdom

[d] Scottish Primate Research Group

*Corresponding author:
Christoph J. von Borell, Department of Biological Personality Psychology,
Georg Elias Müller Institute of Psychology, University of Goettingen,
37073 Göttingen, Germany
Phone: +49-(0)551-3920704
E-Mail: christoph.borell@psych.uni-goettingen.de

Abstract

As is the case for humans, it has long been thought that nonhuman primates can be described in terms of their personality. Scientific observations that support this view include the presence of individual differences in social behavior and that they are relatively stable throughout life. Consequently, individuals are constrained in their behavioral flexibility when dealing with various environmental challenges. Still, the variation among individuals during development suggests that the environment influences how primates behave. Research in fields including psychology, behavior genetics, and behavioral ecology have tried to identify the mechanisms responsible for this interplay of behavioral stability and change. In this review we integrate theories and findings from research on humans and nonhuman primates that highlight how and to what extent genetic and environmental contributions shape the development of social behavior. To do so we first provide an overview and define what is meant by mean level and rank-order change of behavior. We then review explanations of behavioral stability and change, focusing on the role of genetic effects, how environmental circumstances influence behavioral variation throughout development, and how genetic and environmental influences may interact to produce this variation. Finally, we point to future research directions that could help us to further understand the development of social behavior in primates from within a behavior genetics framework.

Keywords

Primate, Personality Development, Behavioral Plasticity, Behavior Genetics, Behavioral Development

Introduction

Teasing, helping, playing, working, learning – within our circle of acquaintances, for many social behaviors, we can think of individuals that fall somewhere between one or the other extreme of variation in any given behavior. Apparently, social behavior and social relationships among humans are influenced by individual characteristics. Research from the last four decades has shown that this applies equally to our closest relatives, the nonhuman primates (henceforth “NHPs”). But how flexible are these individual characteristics? Where do they come from? And can they be changed? In this review we elaborate on the development of individual differences in behavior by comparing findings on humans and NHPs with a focus on the genetic and environmental forces that influence development.

In NHP personality research, the data underlying the quantification of individual differences typically stems either from questionnaires, completed by people with good knowledge of the individual animals, counted behavioral observations, or individuals’ reactions to behavioral tests, where subjects encounter, for example, a setup containing novel objects or food items. Usually a variety of different behaviors are assessed, the correlations among behaviors are calculated and behaviors are grouped into summarizing dimensions using statistical techniques as factor analysis or principal component analysis. In humans, the investigation of such dimensions led to the formulation of the Five-Factor Model of human personality (Digman 1990), where differences among people can be summarized along the dimensions extraversion, agreeableness, conscientiousness, openness to experience, and neuroticism. The Five-Factor Model often serves as a reference point in NHP studies (see e.g. King and Figueredo 1997; Weiss et al. 2015) and analogues or variations of these factors have been found to a varying extent in different NHP species (Weiss 2017a).

The history of animal personality research and the different approaches used, whether by behavioral ecologists or comparative psychologists, have been reviewed elsewhere (Gosling

2001; Réale et al. 2007; Uher 2008; Koski 2011; Carter et al. 2013; Sih et al. 2015; Roche et al. 2016; Weiss 2017b). As such, we will not rehash this literature. Instead, we will focus on the development of behavioral variation among individuals. First, we will review the current knowledge about stability of behavioral differences on a phenotypic level and then proceed to a more detailed overview of the genetic and environmental contributions to behavioral stability and change. We hereby will follow the broad conceptual separation common to research in behavior genetics. Hence by "genetic effects" we refer to behavioral variation due to differences in the sequence of the DNA of individuals and by "environmental effects" we refer to all other influences affecting behavioral variation that are not caused by variation in the individuals' DNA. Towards the end of our review we will also look at the interplay between genetic and environmental effects. The review will focus on findings from NHPs but will be complemented by findings from the human literature where appropriate, that is, if it provides additional insight.

Phenotypic stability over the lifetime

Do aggressive children grow up to be aggressive adults? To answer this and similar questions, we must distinguish between two types of behavioral stability or change. The first is an age-related metric called mean-level change, which refers to differences in the mean expression of a behavioral phenotype at different points in development. Mean-level change can be quantified with regression analysis where age (or different developmental stages, e.g., being an infant, juvenile, adult, etc.) is included as predictor of behavioral variation. Ideally, mean-level change is studied in a longitudinal design, with repeated measurements taken from the same individuals over time. The second is rank-order change, which is quantified by the magnitude of relative changes in behavior that occur among individuals within a population. It is independent of mean-level changes in absolute behavior. An example of a situation where there is little to no rank-order change would be if children who are highly aggressive relative to their age peers become adults who are highly aggressive relative to their age peers. Rank-order stability (or change) of behavior may be quantified by two techniques. The first involves conducting a simple correlation among behavioral measurements from two time points. The second involves computing the repeatability coefficient, which is an intraclass correlation that is based on multiple measures per individual and which describes the proportion of total behavioral variance due to differences between individuals (Boake 1989; Nakagawa and Schielzeth 2010). If the variance within individuals (between different measurements) is zero, then repeatability equals one. If the total behavioral variance is solely due to variation within individuals, then repeatability equals zero. We illustrated the difference between mean-level and rank-order stability in Fig. 1.

Mean-Level Change

Knowledge of lifetime age effects on mean-level change in NHP personality stems especially from a study by King and colleagues (2008). They used cross-sectional data from chimpanzees that were divided into five age groups and found age-related differences in terms of lower extraversion and openness to experience scores, and higher agreeableness and conscientiousness scores, in older individuals. These results are corroborated by behavioral measurements from chimpanzees, where boldness and exploration tendency, which are related in their content to extraversion and openness, respectively, also appear to decline with age (Massen et al. 2013). Such a pattern could also be partly replicated in and transferred to orangutans by Weiss and King (2015), with the exception that in this species agreeableness is lower in older subjects. In common marmosets, females also tend to become less agreeable with increasing age, while both males and females become less inquisitive (Koski et al. 2017). The same pattern applies to older white-faced capuchin monkeys who are less agreeable and less open to new experiences as well (Manson and Perry 2013). So, although individuals are rather stable in their average behavioral propensities in relation to each other, age-related mean-level differences of behavior occur at the level of the population. Some age-related patterns seem to be similar across species (e.g., declines in openness / inquisitiveness / exploration tendency), while the development of agreeableness (indicating pro-social and tolerant behavior) differs among them. The reasons for developmental differences among species need to be clarified by future studies. Possible reasons for inter-species differences are the differing content and structure of the personality dimensions or varying selection pressures between species (Weiss and King 2015). In a sample of adult rhesus macaques (Brent et al. 2013), age was largely unrelated to personality dimensions, indicating that mean-level changes could be especially evident when changes over the lifetime or during early development are considered. Concerning the latter, strong changes in age-specific behavior have been reported that are tied to sex-specific life histories (Kulik et al. 2015a, b; von Borell et al. 2016).

Rank-Order Stability

In adult NHPs, the rank-order stability of behavioral differences ranges from being moderate (above r=0.3) to high (above r=0.5), and is statistically significant (e.g. King et al. 2008; Weiss et al. 2011; Brent et al. 2013; Weiss 2015). High levels of stability are found most often in studies that use ratings on questionnaires. Here, estimates of rank-order stability may be as high or higher than 0.7 (e.g., Stevenson-Hinde and Zunz 1978; King et al. 2008; Weiss et al. 2011). These estimates reflect the relative stability of *average* behavior of individuals, that is, the consistency of displaying certain behavioral phenotypes accumulated across situations (Weiss et al. 2009). If rank-order stability is calculated as repeatability of behavioral measurements, the resulting repeatability coefficient is typically lower than in studies using questionnaire ratings (e.g. Brent et al. 2013; Neumann et al. 2013; von Borell et al. 2016), aligning closer to the meta-analytical mean repeatability of 0.37 measured across species (Bell et al. 2009). It must be noted though that differences in repeatability among non-aggregated behavioral measurements and aggregated questionnaire ratings could occur because averaging single ratings into broader dimensions, that is, into personality “factors”, “domains”, “dimensions”, or “components”, contributes to the stability of these measures (Rushton et al. 1983). During early ontogeny, the stability of individual differences is typically lower than in adults (von Borell et al. 2016) and may show substantial variation from year to year, which may in turn differ across personality domains (Stevenson-Hinde et al. 1980).

What do our measurements tell us about stability? And what do they not tell us?

The studies presented so far used questionnaire ratings or counted behavioral observations to assess the personalities of the individuals under study. They showed patterns of mean-level change in behavior and rank-order stability of individual differences in behavior that predominantly reflect variation on a year-wise or season-wise timescale. However, these approaches may not be sensitive to short-termed effects of the environment. As indicated above, questionnaire ratings accumulate impressions of an animal’s behavior across situations and therefore do not capture short-term interactions of behavior with environmental fluctuations. Some of the studies also rely on animals kept in captivity (e.g., living in zoos, as in King et al. 2008), which may limit the naturally occurring environmental variation for some species.

One possible means by which the influence of the environment on behavior could be tested is by continuously sampling behavioral observations in free ranging animals (von Borell et al. 2016). Yet, the fallacy of behavioral sampling is that observations, for example single incidents of displaying aggressive behavior, are typically also aggregated over time to form a reliable estimate of individual propensities. Otherwise, rare coincidences, like a generally unaggressive individual showing a sign of aggression, could lead to unwarranted conclusions about a general behavioral tendency. Because naturally occurring observations of certain behaviors may be scarce, aggregation operates usually on relatively large time scales (e.g., year-wise or season-wise). Such aggregation limits the possibility of analyzing behavioral plasticity in response to the environment to long-term fluctuations, stable population differences, or permanent changes within populations (such environmental effects will be discussed in the following section). Whether there are developmental influences on short-term plasticity (i.e., reaction norms; Dingemanse et al. 2010) is thus often not assessed. This is despite the fact that it might be hypothesized that NHPs become, for example, less flexible in their behavior with increasing age. Examples from other species show that individuals may vary in their seasonal plasticity, that they are repeatable in such plasticity (i.e., temporally consistent in their rank-order of shown plasticity) and that the mean plasticity across individuals may decrease with age (e.g., in great tits; Araya-Ajoy and Dingemanse 2017). These findings of differences in plasticity are likely due to frequency-dependent costs or benefits leading to individually different behavioral strategies. Furthermore, such costs or benefits are likely to change with experience, leading to mean level changes in plasticity during ontogenic development (Wolf et al. 2008). The question of age-related variability in behavioral plasticity appears to be somewhat of a blind spot in the study of NHP behavioral development. To address this question requires studies that obtain repeated measurements of behavior-situation interactions within and across time intervals or that can calculate the effect of age on behavioral reaction norms in cross-sectional data. One way to gather these kinds of data is by means of behavioral tests that involve simulating situations that an animal may encounter in the wild (e.g., encountering a novel environment or object, confrontation with the vocalization of a predator). For NHPs in captivity behavioral tests have been developed to assess behavioral variation among individuals (e.g., Uher et al. 2013; Staes et al. 2016). If such behavioral tests are conducted with environmental variation or transferred to the natural habitats of NHPs, this approach allows for a controlled collection of data that may be linked to short-term environmental fluctuations. For example, tests of social facilitation that compare behavioral responses to novelty when individuals are alone to when they are in a social context show short-term environmental effects on behavior (reviewed in Forss et al. 2017). In common marmosets, the latency to eat novel food is reduced in a social context, but only in juveniles, suggesting that individual age affects the strength of social facilitation (Yamamoto and Lopes 2004). Following these results, behavioral reaction norms of neophobia or exploration tendency with varying social contexts could be further tested in a longitudinal setting to assess the degree to which individual differences in reaction norms are stable throughout development, i.e., their rank-order stability. There are also examples of behavioral tests conducted with NHPs in the wild (e.g., playback experiments in Neumann et al. 2013; novel-object and novel-food tests in Arnaud et al. 2017). These could be paired with environmental information (e.g., current group composition, time elapsed since among-group conflict, etc.) to form behavioral reaction norms and tested for hypothesized age effects, preferably in a longitudinal design. Other possibilities would be to use data from continuous observations in a non-aggregated way or aggregating observations according to relatively short-term environmental fluctuations and analyze them via linear mixed effects models that can account for zero-inflated observations in the case of rarely observed behaviors (Zuur et al. 2009; Dingemanse and Dochtermann 2013; Brooks et al. 2017). Such an approach would be informative about relationships between behaviors, between individuals, (correlated) changes in behavior within individuals, and whether the interaction among behavior and environmental factors (plasticity) changes with age (Dingemanse and Dochtermann 2013). For a “how-to” example of using the full potential of linear mixed models when analyzing behavioral observations of NHPs see Martin and Suarez (2017).

What do we know from humans?

Findings from research on human personality development are largely consistent with findings from NHPs. In terms of rank-order stability, humans become more stable throughout their lives, developing from moderate stability (approx. r=0.35) in behavioral differences during childhood to high stability (approx. r=0.70) during late adulthood (Roberts and DelVecchio 2000; Terracciano et al. 2006). Mean-level changes occur primarily during early adulthood, a time often marked by major changes in an individual's environment and increased control over life-history decisions: After a period of decreased psychological "maturity" during early puberty (Denissen et al. 2013), humans typically develop towards a more mature and functional personality in that they become more agreeable, conscientious and show more emotional stability (Roberts et al. 2006; Donnellan et al. 2007). However, they also tend to become less flexible (Roberts et al. 2002).

Determinants of Plasticity and Stability in Behavior

Now that we know that behavioral variation among individuals is not fixed and that rank-order and mean-level changes occur in particular during childhood, adolescence, and young adulthood, the question remains how these changes can be explained. We propose to approach questions about behavioral stability and change using a behavior genetics framework, because it helps us to disentangle whether and how behavioral development is caused by environmental influences, genetic effects, or their interplay.

Genetic Effects on Behavioral Development

The rationale behind genetic effects on behavior is that variation in DNA sequences among individuals will lead to variation in their behavioral propensities. The extent to which genes influence a behavioral phenotype is measured with a population statistic “heritability”. Heritability (or h^2) is the ratio of genetically influenced variance in a trait to the total variance of the trait in a population (Plomin et al. 2012; Johnson 2014). Heritability may also be calculated as the ratio of genetically influenced variance to the repeatable variance (as this “error-free” variance poses an upper limit to the heritability; Adams et al. 2012). A trait’s heritability may reflect additive genetic effects whereby the effects of variants of genes (polymorphisms) independently add up to shape the trait into a specific direction. This is known as narrow-sense heritability. A trait’s heritability may also reflect non-additive genetic effects whereby the interactions among different gene variants affect the expression of the trait. An example of this would be a dominant genetic variant (allele) that suppresses the effect of a recessive genetic variant at the same or different loci. The combined influence of additive and non-additive genetic variance is referred to as broad-sense heritability, which is denoted H^2.

To provide a general impression of how heritable personality traits are in NHPs, we calculated the median and range of published estimates of narrow-sense heritability across NHP species and studies (see Tables S1, S2 in the supplement). For personality factors we calculated a median heritability of $h^2=0.25$ and a range from 0.00 to 0.63 (based on the studies from Weiss et al. 2000; Fairbanks et al. 2004; Adams et al. 2012; Brent et al. 2013; Johnson et al. 2015; Latzman et al. 2015; Staes et al. 2016; Wilson et al. 2017; Inoue-Murayama et al. 2018). The heritability of single behaviors appears to be very similar, with a median $h^2=0.25$ and range of 0.11 to 0.91 (based on studies by Rogers et al. 2008; Fawcett et al. 2014; Hopkins et al. 2014, 2015; Johnson et al. 2015; Watson et al. 2015). Non-additive genetic effects may contribute a significant proportion to genetically influenced variance, leading to higher broad-sense heritability estimates (H^2). Based on a study on orangutans we calculated a median H^2 of 0.69 (Adams et al. 2012). Published estimates of broad-sense heritability are, however, an exception, as this requires extended study designs including twins or a large number of full- and half-siblings (ibid.). Unfortunately for a developmental perspective, we do not know of longitudinal studies that published heritability estimates for a birth cohort across time. Nor do we know of cross-sectional estimates of heritability along different developmental stages. Hence, we cannot say whether the heritability estimates of personality traits, and thus influences relating to environmental factors, increase or decrease throughout development.

In humans the average heritability estimated from meta-analyses is a little higher than in NHPs, accounting for about 40% of variation (Turkheimer et al. 2014; Vukasović and Bratko 2015). Interestingly, estimates coming from family and adoption studies, that include only additive genetic effects, have an average effect size of 0.22 (Vukasović and Bratko 2015), which is close to the median effect size we calculated for narrow-sense heritability in NHPs. This percentage may rise to about 50% when only data from twin studies is considered (van den Berg et al. 2004; Vukasović and Bratko 2015) as these estimates reflect the broad-sense heritability. From a developmental perspective, we know that the heritability of personality tends to decrease with increasing age, dropping from roughly 75% during infancy and early childhood down to the above-mentioned estimate of 40% in later adulthood (Briley and Tucker-Drob 2017). Thus, in the period after birth, individual differences in behavior are largely influenced by genetic effects, with the role of environmental effects increasing with age.

The increasing role of the environment is also reflected in its contribution to the increase in the rank-order stability of personality (from r=0.35 in infancy to about r=0.70 in adults; see above), which can be explained by genetic or environmental influences. Here twin studies find that the genetic contribution remains at a steady 35% during the lifespan, while the environmental contribution increases to account for an additional 35% of rank-order stability during development. This means that the stable proportion of behavioral variation is almost entirely genetically influenced during infancy, but that the post-infancy stability increase is almost entirely influenced by environmental factors (Tucker-Drob and Briley 2019).

Environmental Effects

Given the heritability estimates above, we can expect that environmental effects may contribute to over 50% of behavioral variation in NHPs and about 50% in humans, varying with the age of the individual. An important goal of personality and developmental studies across disciplines has been to identify environmental factors that are capable of altering or shaping behavioral differences among individuals. Here we review two broad categories of well-studied environmental factors that influence developing behavioral differences: stressful life experiences and the influence of maternal care and rearing conditions.

Stressful Life Experiences

Environmental stressors influence behavioral development during prenatal or very early life stages. For example, low food availability is linked to higher prenatal maternal stress in Assamese macaques, which leads to increased growth, but decreased motor skill acquisition and reduced immune function in their offspring (Berghänel et al. 2016). Although this evidence is circumstantial, life-history trade-offs such as these may extend to the development of individual differences in related behavioral traits, for example a trade-off between playing and growth (Berghänel et al. 2015). Fertility is also affected by low-quality early environments with individual differences being linked to drought years in baboons (Lea et al. 2015). Next to the quality of the environment, effects of the dominance hierarchy have been documented as a lasting stressor in NHP development. In chimpanzees, for example, maternal rank during pregnancy is not only related to the stress response of the mother, but also to the stress response of her dependent offspring, and especially males thereof (Murray et al. 2018). A relationship between maternal or individual rank and behavioral differences, and especially those relating to aggressive and fearful/bold behavior, has been shown for NHPs of different ages (e.g., French 1981; Bolig et al. 1992; Brent et al. 2013; von Borell et al. 2016). In an experimental manipulation, Kohn and colleagues (2016) showed that climbing up the dominance hierarchy was causally related to changes in social approachability and boldness. We can thus expect changes in the dominance hierarchy as a possible source of environmentally induced variation in personality development. Related evidence stems from a case of severe and selective tuberculosis infection in wild baboons, where the more aggressive individuals of a troop died at once, because they ate from a neighboring troop’s food resource that was infected. These deaths led to an overall more tolerant social style in the troop. While dominance interactions were concentrated among closely ranked individuals, high-ranking individuals were more tolerant of very low-ranking individuals. The latter finding was related to a disproportionally high number of reversals in the direction of dominance among individuals far apart in rank (Sapolsky and Share 2004). This is in line with the argument that high-ranking individuals can typically afford aggressive or displacing behavior due to agonistic support from other individuals (Silk 2002), which was apparently less the case in the newly stratified troop of baboons after the epidemic infection.

Although the quality of the natural environment and dominance hierarchies in social groups affect behavioral differences from early life on, new challenges arise around the time of maturation that drive behavioral variation. A prominent example in NHPs is the migration from the natal group to a new group (natal dispersal). Migration is typically accompanied by increases in mortality or injury rates, decreases in access to resources, and social costs, i.e., the loss of social ties or rank (Dittus 1979; Weiß et al. 2016). Following migration, male rhesus macaques show more fearful and less physically aggressive behavior than before (von Borell et al. 2016), which is consistent with findings from captive pigtailed macaques, where individuals that are new to a facility are more cautious (Sussman et al. 2014). Migration may also trigger rank-order changes in behavior, possibly reflecting different reactions or strategies following migration. In the study of von Borell et al. (2016) this was reflected in very low or even negative correlations among fearful behaviors measured in the year before and after migration, despite their overall lifetime repeatability. In female rhesus macaques, the birth of the first infant is a similar developmental milestone and is marked by a decreased frequency of initiating social contacts outside of maternal kin (von Borell et al. 2016).

Maternal Influences and Rearing

Parental care and the quality of mother-offspring interactions are also known to affect the development of individual differences in NHP behavior. Here we highlight some findings in this literature. Interested readers are encouraged to refer to a detailed review of this literature in this topical collection (Maestripieri 2018).

Differences in maternal style are typically described along the two dimensions protectiveness and rejection, but may vary a little between NHP species, that is, maternal behaviors may also load on three different factors in a factor analysis (De Lathouwers and Van Elsacker 2004). Protectiveness and rejection have been linked to individual differences in behavior across various age-stages in NHP development. For example, in an observational study of Japanese macaques, infants of highly protective mothers showed lower levels of exploratory behavior and interacted less with their group members. On the other hand, infants of mothers who rejected them interacted more than average with other group members. These effects diminished, however, over the course of development and were present mostly during early infancy (Bardi and Huffman 2002). A stable effect of maternal style was reported by Bardi and colleagues (2015) who found that juvenile baboons that experienced more stress-related interactions with their mother during early life showed higher locomotor activity and cortisol levels during a stress test than individuals that experienced more affiliative mother-offspring interactions.

Such effects of parental care or mother-offspring interaction were further supported by experimental studies. An effect of maternal protectiveness on offspring caution was shown in vervet monkeys (Fairbanks and McGuire 1993). In this study maternal protectiveness was experimentally increased by introducing new males to some housing groups. Infants and juveniles of mothers from the “protective” condition showed higher latencies to approach a novel object, indicating increased caution. Approach latencies were highly correlated among mothers and infants but not among mothers and juveniles. These results indicate that a mixture of environmental and genetic effects contributed to the development of behavioral differences. Maestripieri and colleagues (2006) could not find an effect of maternal protectiveness on offspring behavior in rhesus macaques, but they did find that higher maternal rejection led to more solitary play in offspring. This effect did not differ between mother-reared and cross-fostered individuals, ruling out the possibility that this observation is simply driven by genetic similarity between mothers and their offspring.

A special case of maternal influence on behavioral differences is maternal deprivation or the disruption of maternal care. Rhesus macaques that spent their first year of life in total isolation showed hardly any positive social responses or activities afterwards and were also consistently fearful. Individuals who spent shorter periods of time in isolation showed a behavioral pattern similar to that of monkeys who spent a year in isolation, followed by highly individualized (adequate and non-adequate) adaptations to social situations, presumably based on inherited individual differences and unique learning experiences (Harlow et al. 1965). Similar differences in the social response to short periods of isolation have been documented in free-ranging rhesus macaque infants (Berman et al. 1994). Here, increased short-term separations of mothers and their infants, which occurred when the mothers resumed mating, led to increased distress in the infants. Like the captive infants, described by Harlow and colleagues (1965), who were isolated for short periods, the free-ranging infants developed differing social responses to and after the separation events. Specifically, some infants reacted with social withdrawal and decrease of social play and others rather increased their social behavior like grooming. Differential responses to maternal separation or maternal style, whether marked by decreased or increased social behavior, have been linked with genetically inherited differences in stress responsivity (Clarke and Boinski 1995; Suomi 2004). Further studies of maternal separation in captivity, typically on hand-raised and later on peer-reared individuals, suggest temporally consistent increases in anxious, shy, and impulsive behavior in comparison with their mother-reared counterparts. These behavioral differences may extend to neglectful or abusive maternal behavior, when peer-reared females become mothers themselves (reviewed in Soumi 1997). More recent studies, albeit in a different species, show mixed results: while nursery-reared chimpanzees were reported to be less agreeable and more extraverted than their mother-reared counterparts (Latzman et al. 2015), a similar study of chimpanzees found no such differences between these groups (Martin 2005).

The effects of differential care appear to extend to scenarios were the intensity of human care varies. Young chimpanzees who experienced enhanced responsive care were less distressed and showed less disorganized attachment than chimpanzees who only received a minimal standard of care from human caregivers (van IJzendoorn et al. 2008). In addition to maternal style, maternal separation, and the amount of care, the time infants spend with conspecifics seems to affect personality development. For example, chimpanzees who as infants spent less time with conspecifics were rated as being less extraverted later in life than individuals who spent more time with conspecifics (Freeman et al. 2016).

Issues of causality

From a behavior genetics standpoint, non-experimental studies and non-genetically-informed quasi-experimental studies cannot establish causal relationships between environmental and behavioral variation. Although environmental effects can be separated in a controlled randomized experiment (at the cost of decreased ecological validity), all other behavior-environment correlations are likely influenced by genetic variation. As Johnson (2014) put it:

> "The situation and the individual's environmental history may set the stage and limit the range of choice of action, but the individual's genotype is involved both in the actions taken and the individual's presence in this situation in the first place. We cannot understand development without taking this into consideration."

Among the findings on stressful life events or rearing experience reviewed above, experimentally separated environmental effects rely largely on captive NHPs, while in studies conducted in the wild, environmental and genetic effects can be confounded. There are several mechanisms of such confounding. Prominent examples include gene-environment correlations (rGE) and gene-environment interactions (G x E), both of which will be discussed below. The main message at this point is, that a neglect of genetic information can lead to premature causal interpretations of the role the environment may play in behavioral development (Briley et al. 2018). For example, the association between early adversity and a faster life-history strategy that has been reported in NHPs, has received theoretical and empirical support from the human literature as well, leading, for example, to earlier puberty and marriage (see reviews by Belsky 2012; Del Guidice 2014). However, findings of life-history embedded behavioral differences related to early adversity did not hold up in a study design that included information of genetic relatedness based on pedigrees to control for genetic confounding. Mendle and colleagues (2009) found that the association among father absence and timing of first intercourse in humans was best explained by genetic risk factors that correlate both with father absence and early sexual activity, diminishing the role of the mere *experience* of an absent father. Likewise, decisions involving changes in the social environment, such as NHP dispersal, are known to carry a genetic component (Trefilov et al. 2000; Krawczak et al. 2005) that could also be correlated to behavioral differences. Also, relationships between rank and behavior may partly be affected by feedback processes entailing a genetic component, for example the interplays of aggressive behavior, which has a heritable component, and changes in the dominance hierarchy in male NHPs (Koyama 1970; Bernstein 1976). In humans, some studies on personality development try to test whether environmental effects are causal by including a control group. Examples can be found in studies on personality development during periods of spatial and social transformation in human adolescents or young adults: events like a high-school student exchange (Hutteman et al. 2015), studying abroad as college student (Zimmermann and Neyer 2013), graduation from high school (Bleidorn 2012), or forming a partner relationship (Neyer and Lehnart 2007) mostly trigger a development towards personality maturation compared to the control group, i.e., increases in conscientiousness, agreeableness and self-esteem, and a decrease in neuroticism. Going abroad was also related to increases in openness to new experiences. The inclusion of a control group is certainly an improvement over not including a control group, as it can be the case in related studies of NHP migration in the wild, where it is often difficult to gather a control group with similar characteristics and a similar sample size as the migrating individuals. Yet, in naturally occurring control group designs, such as the above-described human studies, the decision of whether to participate and the behavioral differences among individuals of the control and quasi-experimental groups may be influenced by common genetic effects. Even if both groups have been matched to be similar in their behavioral characteristics prior to the environmental change, this change may only activate or amplify a genetic predisposition of a behavioral tendency, for example, being open to new experience that was already entailed in the decision of participating in this event.

In the human literature, the impact of individuals' genetic background on behavior or (life-history) decisions (e.g., student exchange, marriage, etc.) led to the "first law of behavior genetics" that all traits are heritable (Turkheimer 2000). It follows that behavior-environment correlations cannot be interpreted as prima facie evidence of a *causal* environmental influence without considering that such associations are probably genetically mediated (Johnson et al. 2011; Johnson and Penke 2014; Turkheimer et al. 2014). Accordingly, calls for genetically informed designs in the study of behavior-environment associations have been pointed out in primatology (e.g. Adams 2014; Brent and Melin 2014) and psychology (Turkheimer and Harden 2014), that could control for a genetic basis of differences in the environment that individuals experience. For example, studies looking at the effects of migration on behavioral differences among individuals could control for the possibility that both share a common genetic basis. Briley and colleagues (2018) reviewed techniques that are capable of tackling questions of causality in longitudinal, and even cross-sectional, genetically informative data (i.e., data where behavioral outcomes and measurements of the environment are paired with information about relatedness or molecular genetic similarity among individuals). For example, in a quantitative genetic design, direction-of-causation modeling (DOC modeling) can be used to estimate the plausibility of a causal direction among an environmental and a behavioral measure. This approach involves comparing the proportion of variance attributable to genetic, shared, and nonshared environmental effects in the possible cause and outcome. If, for example, differences in maternal style have a large genetic component and causally explain behavioral differences among children, then a genetic component should be represented in the children's behavioral differences as well. Comparing the fit of different models with alternative directions of causality can help to assess the likelihood of a hypothesized cause-outcome-relationship (for details see Briley et al. 2018). In human female twins, DOC modeling showed that parental behavior was more likely the cause of psychological distress than psychological distress being the cause of parental behavior (i.e., the model specifying a causal relationship from parental behavior to distress had a better fit than the other way around; Gillespie et al. 2003).

Gene-environment interplay

As pointed out above, in observational studies, whenever a complex interplay among genes and the environment is present during development, separating the environmental and genetic sources of variance can be difficult (but still see Briley et al. 2018). In the case of gene-environment correlations (rGE), individuals evoke, pick, or create environmental experiences based on genetically influenced needs or preferences, or grow up in an environment that is influenced by genes they share with their parents (see, e.g., Scarr and McCartney 1983; Bleidorn et al. 2014; Weiss 2017b). Another possibility is that the impact of environmental experiences differs depending on individuals’ genetic backgrounds (e.g., a genetic risk or vulnerability; Moffitt 2005), which is termed gene-environment interaction (G x E). While heritability estimates tell us that the biological underpinnings of behavior cannot be ignored in developmental studies, they are less useful in helping us to understand the developmental mechanisms or processes behind emerging behavioral differences, as variance is here partitioned into being genetic or environmental, and so does not account for gene-environment interplay (Plomin and Bergeman 1991).

In some species, it is possible to conduct controlled experiments on developmental psychobiology that allow for a separation of genetic and environmental effects (e.g., by breeding genetically identical individuals in identical conditions; Kain et al. 2012; Bierbach et al. 2017), but ethical and practical reasons mostly prevent scientists from applying these methods to humans or NHPs (Turkheimer 2000; but see experimental manipulations of rearing conditions presented above). Yet, there is no need for primatologists or psychologists to stop searching for the causes of development. Although we may not be able to causally reconstruct complex developmental pathways, we can test how genes and the environment correlate and interact in specific scenarios and how likely they are to shape behavioral development within the limits of such scenarios.

An example of NHP rGEs is the above-cited genetic influence on dispersal where genetic variation leads to different ages of migration from the natal group, that is, the encounter of a novel environment (Trefilov et al. 2000). Correlations among genes (or genetically influenced traits) and the environment are often referred to as “niche picking” or “niche specialization” (Johnson et al. 2009; Penke 2010; Stamps and Groothuis 2010; for evolutionary and mathematical formalization, see Montiglio et al. 2013). If we consider a developmental pathway where having more of some trait leads to a higher propensity to seek out a specific environment, which in turn affects the manifestation of that trait, then cross-sectional studies cannot distinguish between such bidirectional influences of genetic background and the environment (Kandler et al. 2012). If not explicitly modeled, the variation due to rGE will be confounded with genetic variance, although an environmental influence is entailed as well (Bleidorn et al. 2014). Genetically informed longitudinal studies, however, make it possible to test instantiations of rGE. In humans, Kandler and colleagues (2012) showed that genetic effects on personality traits, such as neuroticism or agreeableness, can explain variation in the likelihood of experiencing negative life events and that negative life events, in turn, have a (small) effect on personality development.

G x E effects on personality development can be detected by quantitative or molecular genetics methods. Quantitative genetic studies test whether differences in a phenotype between individuals are associated with information on their genetic relatedness (for example based on known pedigrees), while molecular genetic studies try to associate differences in a phenotype with a specific pattern of variation in DNA sequence among individuals. In behavioral genetic research, the latter's emphasis is on trying to find associations between genetic variants at specific genetic loci and behavioral traits (candidate gene association study) or trying to associate a large number of variants that are spread across the genome with a behavioral trait (genome-wide association study, GWAS). In a quantitative genetics framework, Latzman and colleagues (2015) have shown that heritability estimates of personality dimensions vary among mother- and nursery-reared chimpanzees. Specifically, they found lower heritability estimates in nursery-reared individuals indicating that their atypical environmental circumstances at an early age led to a higher proportion of environmentally influenced behavioral variation among their traits. Results from humans also support interaction effects of rearing quality and genes. For example, Krueger and colleagues (2008) showed that the genetic influence on adolescent personality varied with the levels of regard they received from their parents. In particular, low levels of regard were associated with an increased environmental contribution to phenotypic variance. On a molecular level, many NHP studies have examined the interplay of environmental variation and candidate genes in their contribution to behavioral differences. These studies analyzed for example polymorphisms in genes such as 5-HTTLPR (Barr et al. 2004; Madrid et al. 2018), MAOA (Newman et al. 2005), and COMT (Gutleb et al. 2017), which often, but not exclusively, were reported to interact with differences in rearing condition (for a review see Rogers 2018).

In the molecular genetics area, studies of NHPs and humans used to be closely linked and shared a desire to identify the genetic underpinnings of behavioral or pathological variation by testing the effects of candidate genes (see, e.g., Caspi et al. 2002, 2003 on G x E in humans, including MAOA and 5-HTTLPR variation affecting violence and depression, respectively). However, meta-analyses and recent studies in humans that use samples that are several magnitudes larger in size and extensive genome-wide genetic information led to the conclusion that complex behavioral traits are unlikely to be substantially influenced by single genes (Munafò and Flint 2004; Plomin and von Stumm 2018; Sallis et al. 2018). That does not mean that genetic polymorphisms in single genes do not matter, but that their effects are usually too small to be detected with the sample sizes of earlier studies, and this is especially the case when they are modeled in interactions with environmental gradients. Reviews of human candidate gene studies show that many associations cannot be replicated across studies and in meta-analyses, and that the effect sizes of statistically significant associations in earlier studies were often inflated (e.g. Sanchez-Roige et al. 2018). These findings led researchers to conclude that the literature on associations among common variants in candidate genes and behavior, for both main effects and G x E interactions, is awash with false positive results (Sallis et al. 2018). Genome-wide association studies that explore associations of common genetic variants and behavior throughout the whole genome show that a large number of genetic variants (single nucleotide polymorphisms; SNPs) contribute to the heritability of complex traits, however with small effect sizes. Replicated SNPs typically explain less than 0.1% of the phenotypic variance (Munafò et al. 2014; Sallis et al. 2018). While many SNPs reported in candidate gene studies did not replicate in sufficiently powered GWAS (e.g., Chabris et al. 2012), many variants that met genome-wide significance levels that have been identified in GWAS could be replicated in large independent samples (> 100,000 individuals; e.g., Okbay et al. 2016). These variants are spread broadly across the genome, including intragenic regions that do not code for proteins (Boyle et al. 2017; Sanchez-Roige et al. 2018). Additionally, extended study designs show that rare genetic variants that are not tagged in GWAS can contribute to individual variation in complex traits (Hill et al. 2018). While these findings and conclusions stem from human studies, they are likely to apply to NHP studies as well (Munafò et al. 2014). That is not to say that all statistically significant results stemming from NHP candidate-gene or GxE studies are false positives. Some gene-behavior associations have replicated across populations, species, and behavioral measures (reviewed in Weiss 2017a; Rogers 2018). For example, variants in the arginine vasopressin receptor 1A gene (AVPR1A) appear to replicate across different samples of chimpanzees (Anestis et al. 2014; Hopkins et al. 2014; Staes et al. 2015; Wilson et al. 2017), bonobos (Staes et al. 2016) and common marmosets (Inoue-Murayama et al. 2018). However, the combination of small sample sizes and relatively large effects of reported genetic variants is similar to the early wave of human studies in the field of behavior genetics. It is thus probably worth retaining one's skepticism about this literature. Reported effect sizes of replicated genetic variants in NHPs (e.g. given in Staes et al. 2015 and Wilson et al. 2017 for AVPR1A) are several magnitudes larger than most of the extensively studied candidate-gene variants and GWAS results in humans (see Sanchez-Roige et al. 2018 for a review). It is possible that the development and the social influences on behavioral variation among humans are more complex and thus less influenced by single genetic variants. Also, studies on captive NHPs provide a more restricted and controlled environment (e.g., controlled diet, less habitat variation), which might lead to stronger genetic effects. A recent study on the effects of variants in OXTR and AVP receptor genes (AVPR1A, AVPR1B) on behavior in rhesus macaques, however, failed to replicate previous results and showed only very small effects of the 12 SNPs that were examined (Madlon-Kay et al. 2018). Alongside the emerging consistency of findings that single genetic variants have only small effects on complex traits, Madlon-Kay and colleagues (2018) discuss other methodological difficulties, including missing control of genetic relatedness within the population and/or missing adjustment of p-values, that raise doubt about earlier positive results.

A promising avenue for matching smaller sample sizes with genetic information appears to be the use of polygenic scores, where genetic variants accounting for small effects are weighted and summed, creating a score for each subject that is a more powerful estimator of behavioral differences. Given a robust knowledge of genetic variants that contribute to behavioral differences in a species, polygenic scores can help relatively small samples to reach sufficient power to detect molecular genetic effects on behavior and be paired with environmental measures to assess G x E (Plomin and von Stumm 2018). For example, a polygenic score that predicts 10% of the variance in a trait only needs a sample size of 60 individuals to detect its effect with 80% power (ibid.). The problem for NHP studies is that, depending on the species, it might be impossible to gather a sufficiently large initial sample to identify genetic variants that are worth including in a polygenic score in the first place.

In the concluding lines of this section we want to provide a glimpse into the emerging field of epigenetics. Epigenetics refers to processes whereby environmental signals affect genetic variation by mechanisms such as DNA methylation or histone modification. Briefly, these environmentally induced mechanisms can lead to individual differences in gene transcription and expression, which can result in behavioral differences (Kaminsky et al. 2008). In baboons, for example, Runcie and colleagues (2013) found that different aspects of the social environment and social behavior (social connectedness, group size, and maternal dominance rank) interacted with the genotype by means of differences in gene expression along these environmental or behavioral gradients. This suggests that social behaviors, like grooming, are not only influenced by genetic variation, but also influence genetic variation. From an ontogenetic perspective, this means that genes are not destiny for the development of personality, but rather that the environment can alter the genetic tracks individuals are set on. The precise way in which epigenetic mechanisms function in relation to complex traits, as social behavior, is under current investigation (Hu and Barrett 2017). First evidence on the behavioral level indicates, for example, the potential role of epigenetics in the stress response system and associated behavioral differences such as risk-taking or novelty-seeking (Laviola et al. 2003; Kaminsky et al. 2008; Canestrelli et al. 2016). Also epigenetic mechanisms in the domain of memory formation and learning (Duke et al. 2017) may transfer to behavioral differences among individuals. But until we have replicated evidence of epigenetic effects on behavioral traits, a degree of humility about these findings would seem appropriate (see also Cobben and van Oers 2016). In particular, epigenetic explanations centering on specific genes should be interpreted carefully, as associations among single genes and behaviors often do not replicate in studies of humans and NHPs (see above). Given the increasing general understanding of genome-wide DNA methylation patterns in humans and NHPs (Lea et al. 2016, 2018), the role of epigenetics in personality development could become an interesting area of future research (Trillmich et al. 2018).

Summary and Outlook

We can infer that behavioral differences among individual NHPs develop towards increasing rank-order stability and a pattern indicative of what has been described as a “mature” personality in humans (but see exceptions in Manson and Perry 2013; Weiss and King 2015; Koski et al. 2017). Whereas environmental influences on behavioral variation among individuals act in humans especially around the time of adolescence and young adulthood, behavioral variation in NHPs seems to already be affected early in life. Among these early environmental influences are stress-related variation in the natural environment, parenting style or rearing conditions. Later in life, migration or maternity during young adulthood may also affect personality development. As a complex interplay among genotype and the environment is likely, and the statistical power to detect even two-way interactions is low, current research is still far from disentangling the causal pathways that lead to behavioral differences. We propose that one possible way to peek inside this “black box” is to conduct genetically informed longitudinal studies or to use cross-sectional DOC modeling (Turkheimer and Harden 2014; Briley et al. 2018). That said, studies have to be adequately powered if they wish to use these tools. Since statistical power often turns out to be a problem in NHP studies, one possible direction might be to identify polygenic scores for behavioral differences in relatively large samples of a species, for example in breeding facilities, and then to apply this knowledge to the typically smaller populations in the wild or in other captive settings, such as zoos or sanctuaries. This could enable one to conduct genetically informative studies without the need for pedigree data or could supplement studies with (partly) existing pedigree data. Furthermore, testing evolutionary hypotheses stating under which conditions correlations among behavioral differences will occur and how stable these correlations are under changing environments or selection regimes (see Sih et al. 2004; Dochtermann and Dingemanse 2013) could be a fruitful direction for primate personality research. An example would be to test whether environmental variation affecting food resources favors different behavioral strategies or correlations among behaviors that form behavioral syndromes (Dingemanse et al. 2004). Human studies could also be informed, or inspired by, the increasing knowledge of dominance rank and hierarchy effects on behavioral variation in NHPs.

Compliance with Ethical Standards

Conflict of interest

We do not have any potential conflicts of interest to report.

References

Adams MJ (2014) Feasibility and uncertainty in behavior genetics for the nonhuman primates. Int J Primatol 35:156–168. https://doi.org/10.1007/s10764-013-9722-8

Adams MJ, King JE, Weiss A (2012) The majority of genetic variation in orangutan personality and subjective well-being is nonadditive. Behav Genet 42:675–686. https://doi.org/10.1007/s10519-012-9537-y

Anestis SF, Webster TH, Kamilar JM, Fontenot M B, Watts DP, Bradley BJ (2014) AVPR1A variation in chimpanzees (*Pan troglodytes*): Population differences and association with behavioral style. Int J Primatol 35:305–324. https://doi.org/10.1007/s10764-013-9747-z

Araya-Ajoy YG, Dingemanse NJ (2017) Repeatability, heritability, and age-dependence of seasonal plasticity in aggressiveness in a wild passerine bird. J Anim Ecol 86:227–238. https://doi.org/10.1111/1365-2656.12621

Arnaud CM, Suzumura T, Inoue E, Adams MJ, Weiss A, Inoue-Murayama M (2017) Genes, social transmission, but not maternal effects influence responses of wild Japanese macaques (*Macaca fuscata*) to novel-object and novel-food tests. Primates 58:103–113. https://doi.org/10.1007/s10329-016-0572-9

Bardi M, Bode AE, Ramirez SM, Brent LY (2005) Maternal care and development of stress responses in baboons. Am J Primatol 66:263–278. https://doi.org/10.1002/ajp.20143

Bardi M, Huffman MA (2002) Effects of maternal style on infant behavior in Japanese macaques (*Macaca fuscata*). Dev Psychobiol 41:364–372. https://doi.org/10.1002/dev.10065

Barr CS, Newman TK, Lindell S, Shannon C, Champoux M, Lesch KP, Suomi SJ, Goldman D, Higley JD (2004) Interaction between serotonin transporter gene variation and rearing condition in alcohol preference and consumption in female primates. Arch Gen Psychiat 61:1146-1152. https://doi.org/10.1001/archpsyc.61.11.1146

Bell AM, Hankison SJ, Laskowski KL (2009) The repeatability of behaviour: a meta-analysis. Anim Behav 77: 771–783. https://doi.org/10.1016/j.anbehav.2008.12.022

Belsky J (2012) The development of human reproductive strategies: progress and prospects. Curr Dir Psychol Sci 21:310–316. https://doi.org/10.1177/0963721412453588

Berghänel A, Heistermann M, Schülke O, Ostner J (2016) Prenatal stress effects in a wild, long-lived primate: predictive adaptive responses in an unpredictable environment. Proc R Soc B 283:20161304. https://doi.org/10.1098/rspb.2016.1304

Berghänel A, Schulke O, Ostner J (2015) Locomotor play drives motor skill acquisition at the expense of growth: A life history trade-off. Sci Adv 1:e1500451. https://doi.org/10.1126/sciadv.1500451

Berman CM, Rasmussen KLR, Suomi SJ (1994) Responses of free-ranging rhesus monkeys to a natural form of social separation. I. Parallels with mother-infant separation in Ccaptivity. Child Dev 65:1028–1041. https://doi.org/10.1111/j.1467-8624.1994.tb00800.x

Bernstein IS (1976) Dominance, aggression and reproduction in primate societies. J Theor Biol 60:459–472. https://doi.org/10.1016/0022-5193(76)90072-2

Bierbach D, Laskowski KL, Wolf M (2017) Behavioural individuality in clonal fish arises despite near-identical rearing conditions. Nat Commun 8:15361. https://doi.org/10.1038/ncomms15361

Bleidorn W (2012) Hitting the road to adulthood: short-term personality development during a major life transition. Pers Soc Psychol B 38:1594–1608. https://doi.org/10.1177/0146167212456707

Bleidorn W, Kandler C, Caspi A (2014) The behavioural genetics of personality development in adulthood-classic, contemporary, and future trends. Eur J Personality 28:244–255. https://doi.org/10.1002/per.1957

Boake CRB (1989) Repeatability: Its role in evolutionary studies of mating behavior. Evol Ecol 3:173–182. https://doi.org/10.1007/BF02270919

Bolig R, Price CS, O'Neill PL, Suomi SJ (1992) Subjective assessment of reactivity level and personality traits of rhesus monkeys. Int J Primatol 13:287–306. https://doi.org/10.1007/BF02547817

Boyle EA, Li YI, Pritchard JK (2017) An expanded view of complex traits: From polygenic to omnigenic. Cell 169:1177–1186. https://doi.org/10.1016/j.cell.2017.05.038

Brent LJN, Melin AD (2014). The genetic basis of primate behavior: Genetics and genomics in field-based primatology. Int J Primatol 35:1–10. https://doi.org/10.1007/s10764-013-9732-6

Brent LJN, Semple S, MacLarnon A, Ruiz-Lambides A, Gonzalez-Martinez J, Platt ML (2014) Personality traits in rhesus macaques (*Macaca mulatta*) are heritable but do not predict reproductive output. Int J Primatol 35:188–209. https://doi.org/10.1007/s10764-013-9724-6

Briley DA, Livengood J, Derringer J (2018) Behaviour genetic frameworks of causal reasoning for personality psychology: behaviour genetics and causal reasoning. Eur J Personality 32:202–220. https://doi.org/10.1002/per.2153

Briley DA, Tucker-Drob EM (2017) Comparing the developmental genetics of cognition and personality over the life span: Comparing developmental genetics. J Pers 85:51–64. https://doi.org/10.1111/jopy.12186

Brooks ME, Kristensen K, van Benthem KJ, Magnusson A, Berg CW, Nielsen A, Skaug HJ, Machler M, Bolker BM (2017) Modeling zero-inflated count data with glmmTMB. bioRxiv, https://doi.org/10.1101/132753

Canestrelli D, Bisconti R, Carere C (2016) Bolder takes all? The behavioral dimension of biogeography. Trends Ecol Evol 31:35–43. https://doi.org/10.1016/j.tree.2015.11.004

Carter AJ, Feeney WE, Marshall HH, Cowlishaw G, Heinsohn R (2013) Animal personality: what are behavioural ecologists measuring? Biol Rev 88:465–475. https://doi.org/10.1111/brv.12007

Caspi A, McClay J, Moffitt TE, Mill J, Martin J, Craig IW, Taylor A, Poulton R (2002) Role of genotype in the cycle of violence in maltreated children. Science 297:851–854. https://doi.org/10.1126/science.1072290

Caspi A, Sugden K, Moffitt TE et al (2003) Influence of life stress on depression: moderation by a polymorphism in the 5-HTT gene. Science 301:386–389. https://doi.org/10.1126/science.1083968

Chabris CF, Hebert BM, Benjamin DJ et al (2012) Most reported genetic associations with general intelligence are probably false positives. Psychol Sci 23:1314–1323. https://doi.org/10.1177/0956797611435528

Clarke AS, Boinski S (1995) Temperament in nonhuman primates. Am J Primatol 37:103–125. https://doi.org/10.1002/ajp.1350370205

Cobben MMP, van Oers K (2016) Bolder takes all and the role of epigenetics. A comment on Canestrelli et al. Trends Ecol Evol 31:498–499. https://doi.org/10.1016/j.tree.2016.04.007

De Lathouwers M, Van Elsacker L (2004) Comparing maternal styles in bonobos (*Pan paniscus*) and chimpanzees (*Pan troglodytes*). Am J Primatol 64:411–423. https://doi.org/10.1002/ajp.20089

Del Giudice M (2014) An evolutionary life history framework for psychopathology. Psychol Inq 25:261–300. https://doi.org/10.1080/1047840X.2014.884918

Denissen JJA, van Aken MAG, Penke L, Wood D (2013) Self-regulation underlies temperament and personality: an integrative developmental framework. Child Dev Perspect 7:255–260. https://doi.org/10.1111/cdep.12050

Digman JM (1990) Personality structure: emergence of the five-factor model. Annu Rev Psychol 41:417–440. https://doi.org/10.1146/annurev.ps.41.020190.002221

Dingemanse NJ, Both C, Drent PJ, Tinbergen JM (2004) Fitness consequences of avian personalities in a fluctuating environment. Proc R Soc Lond B 271:847–852. https://doi.org/10.1098/rspb.2004.2680

Dingemanse NJ, Dochtermann NA (2013) Quantifying individual variation in behaviour: mixed-effect modelling approaches. J Anim Ecol 82:39–54. https://doi.org/10.1111/1365-2656.12013

Dingemanse NJ, Kazem AJN, Réale D, Wright J (2010) Behavioural reaction norms: animal personality meets individual plasticity. Trends Ecol Evol 25:81–89. https://doi.org/10.1016/j.tree.2009.07.013

Dittus WPJ (1979) The evolution of behaviors regulating density and age-specific sex ratios in a primate population. Behaviour 69:265–301. https://doi.org/10.1163/156853979X00511

Dochtermann NA, Dingemanse NJ (2013) Behavioral syndromes as evolutionary constraints. Behav Ecol 24:806–811. https://doi.org/10.1093/beheco/art002

Donnellan MB, Conger RD, Burzette RG (2007). Personality development from late adolescence to young adulthood: Differential stability, normative maturity, and evidence for the maturity-stability hypothesis. J Pers 75:237–264. https://doi.org/10.1111/j.1467-6494.2007.00438.x

Duke CG, Kennedy AJ, Gavin CF, Day JJ, Sweatt JD (2017) Experience-dependent epigenomic reorganization in the hippocampus. Learn Memory 24:278–288. https://doi.org/10.1101/lm.045112.117

Fairbanks LA, McGuire MT (1993) Maternal protectiveness and response to the unfamiliar in vervet monkeys. Am J Primatol 30:119–129. https://doi.org/10.1002/ajp.1350300204

Fairbanks LA, Newman TK, Bailey JN, Jorgensen MJ, Breidenthal SE, Ophoff RA, Comuzzie AG, Martin LJ, Rogers J (2004) Genetic contributions to social impulsivity and aggressiveness in vervet monkeys. Biol Psychiat 55:642–647. https://doi.org/10.1016/j.biopsych.2003.12.005

Fawcett GL, Dettmer AM, Kay D, Raveendran M, Higley JD, Ryan ND, Cameron JL, Rogers J (2014) Quantitative genetics of response to novelty and other stimuli by infant rhesus macaques (*Macaca mulatta*) across three behavioral assessments. Int J Primatol 35:325–339. https://doi.org/10.1007/s10764-014-9750-z

Forss SIF, Koski SE, van Schaik CP (2017) Explaining the paradox of neophobic explorers: the social information hypothesis. Int J Primatol 38:799–822. https://doi.org/10.1007/s10764-017-9984-7

Freeman HD, Weiss A, Ross SR (2016) Atypical early histories predict lower extraversion in captive chimpanzees: early histories predict extraversion. Dev Psychobiol 58:519–527. https://doi.org/10.1002/dev.21395

French JA (1981) Individual differences in play in *Macaca fuscata*: The role of maternal status and proximity. Int J Primatol 2:237–246. https://doi.org/10.1007/BF02739332

Gillespie NA, Zhu G, Neale MC, Heath AC, Martin NG (2003) Direction of causation modeling between cross-sectional measures of parenting and psychological distress in female twins. Behav Genet 33:383–396

Gosling SD (2001) From mice to men: what can we learn about personality from animal research? Psychol Bull 127:45–86

Gutleb DR, Roos C, Noll A, Ostner J, Schülke O (2018) COMT Val^{158} Met moderates the link between rank and aggression in a non-human primate. Genes Brain Behav 17:e12443. https://doi.org/10.1111/gbb.12443

Harlow HF, Dodsworth RO, Harlow MK (1965) Total social isolation in monkeys. P Natl Acad Sci USA 54:90–97

Hill WD, Arslan RC, Xia C et al (2018) Genomic analysis of family data reveals additional genetic effects on intelligence and personality. Mol Psychiatr 23:2347-2362. https://doi.org/10.1038/s41380-017-0005-1

Hopkins WD, Keebaugh AC, Reamer LA, Schaeffer J, Schapiro SJ, Young LJ (2015) Genetic influences on receptive joint attention in chimpanzees (*Pan troglodytes*). Sci Rep 4:3774. https://doi.org/10.1038/srep03774

Hopkins WD, Reamer L, Mareno MC, Schapiro SJ (2014) Genetic basis in motor skill and hand preference for tool use in chimpanzees (*Pan troglodytes*). Proc R Soc B 282:20141223. https://doi.org/10.1098/rspb.2014.1223

Hu J, Barrett RDH (2017) Epigenetics in natural animal populations. J Evol Biol 30:1612–1632. https://doi.org/10.1111/jeb.13130

Hutteman R, Nestler S, Wagner J, Egloff B, Back MD (2015) Wherever I may roam: Processes of self-esteem development from adolescence to emerging adulthood in the context of international student exchange. J Pers Soc Psychol 108:767–783. https://doi.org/10.1037/pspp0000015

Inoue-Murayama M, Yokoyama C, Yamanashi Y, Weiss A (2018) Common marmoset (*Callithrix jacchus*) personality, subjective well-being, hair cortisol level and *AVPR1a*, *OPRM1*, and *DAT* genotypes. Sci Rep 8:10255. https://doi.org/10.1038/s41598-018-28112-7

Johnson W (2014) Developing Difference. Palgrave Macmillan, Basingstoke

Johnson W, Deary IJ, Iacono WG (2009) Genetic and environmental transactions underlying educational attainment. Intelligence 37:466–478. https://doi.org/10.1016/j.intell.2009.05.006

Johnson W, Penke L, Spinath FM (2011) Heritability in the era of molecular genetics: Some thoughts for understanding genetic influences on behavioural traits. Eur J Personality 25:254–266. https://doi.org/10.1002/per.836

Johnson Z, Brent L, Alvarenga JC et al (2015) Genetic influences on response to novel objects and dimensions of personality in *Papio* baboons. Behav Genet 45:215–227. https://doi.org/10.1007/s10519-014-9702-6

Kain JS, Stokes C, de Bivort BL (2012) Phototactic personality in fruit flies and its suppression by serotonin and white. P Natl Acad Sci USA 109:19834–19839. https://doi.org/10.1073/pnas.1211988109

Kaminsky Z, Petronis A, Wang S-C, Levine B, Ghaffar O, Floden D, Feinstein A (2008) Epigenetics of personality traits: an illustrative study of identical twins discordant for risk-taking behavior. Twin Res Hum Genet 11:1–11. https://doi.org/10.1375/twin.11.1.1

Kandler C, Bleidorn W, Riemann R, Angleitner A, Spinath FM (2012) Life events as environmental states and genetic traits and the role of personality: A longitudinal twin study. Behav Genet 42:57–72. https://doi.org/10.1007/s10519-011-9491-0

King J E, Figueredo AJ (1997) The five-factor model plus dominance in chimpanzee personality. J Res Pers 31:257–271. https://doi.org/10.1006/jrpe.1997.2179

King JE, Weiss A, Sisco MM (2008) Aping humans: Age and sex effects in chimpanzee (*Pan troglodytes*) and human (*Homo sapiens*) personality. J Comp Psychol 122:418–427. https://doi.org/10.1037/a0013125

Kohn JN, Snyder-Mackler N, Barreiro LB, Johnson ZP, Tung J, Wilson ME (2016) Dominance rank causally affects personality and glucocorticoid regulation in female rhesus macaques. Psychoneuroendocrinol 74:179–188. https://doi.org/10.1016/j.psyneuen.2016.09.005

Koski SE (2011) How to measure animal personality and why does it matter? Integrating the psychological and biological approaches to animal personality. In: Inoue-Murayama M, Kawamura S, Weiss A (eds) From Genes to Animal Behavior. Springer Japan, Tokyo, pp 115-136

Koski SE, Buchanan-Smith HM, Ash H, Burkart JM, Bugnyar T, Weiss A (2017) Common marmoset (*Callithrix jacchus*) personality. J Comp Psychol 131:326–336. https://doi.org/10.1037/com0000089

Koyama N (1970) Changes in dominance rank and division of a wild Japanese monkey troop in Arashiyama. Primates 11:335–390. https://doi.org/10.1007/BF01730638

Krawczak M, Trefilov A, Berard J, Bercovitch F, Kessler M, Sauermann U, Croucher P, Nürnberg P, Widdig A, Schmidtke J (2005) Male reproductive timing in rhesus macaques is influenced by the 5HTTLPR promoter polymorphism of the serotonin transporter gene 1. Biol Reprod 72:1109–1113. https://doi.org/10.1095/biolreprod.104.038059

Krueger RF, South S, Johnson W, Iacono W (2008) The heritability of personality is not always 50%: Gene-environment interactions and correlations between personality and parenting. J Pers 76:1485–1522. https://doi.org/10.1111/j.1467-6494.2008.00529.x

Kulik L, Amici F, Langos D, Widdig A (2015a) Sex differences in the development of aggressive behavior in rhesus macaques (*Macaca mulatta*). Int J Primatol 36:764–789. https://doi.org/10.1007/s10764-015-9853-1

Kulik L, Amici F, Langos D, Widdig A (2015b) Sex differences in the development of social relationships in rhesus macaques (*Macaca mulatta*). Int J Primatol 36:353–376. https://doi.org/10.1007/s10764-015-9826-4

Latzman RD, Freeman HD, Schapiro SJ, Hopkins WD (2015) The contribution of genetics and early rearing experiences to hierarchical personality dimensions in chimpanzees (*Pan troglodytes*). J Pers Soc Psychol 109:889–900. https://doi.org/10.1037/pspp0000040

Laviola G, Macrì S, Morley-Fletcher S, Adriani W (2003) Risk-taking behavior in adolescent mice: psychobiological determinants and early epigenetic influence. Neurosci Biobehav R 27:19–31. https://doi.org/10.1016/S0149-7634(03)00006-X

Lea AJ, Altmann J, Alberts SC, Tung J (2015) Developmental constraints in a wild primate. Am Nat 185:809–821. https://doi.org/10.1086/681016

Lea AJ, Altmann J, Alberts SC, Tung J (2016) Resource base influences genome-wide DNA methylation levels in wild baboons (*Papio cynocephalus*). Mol Ecol 25:1681–1696. https://doi.org/10.1111/mec.13436

Lea AJ, Vockley CM, Johnston RA, Del Carpio CA, Barreiro LB, Reddy TE, Tung J (2017) Genome-wide quantification of the effects of DNA methylation on human gene regulation. bioRxiv, https://doi.org/10.1101/146829

Madlon-Kay S, Montague MJ, Brent LJN, Ellis S, Zhong B, Snyder-Mackler N, Horvath JE, Skene JHP, Platt M L (2018) Weak effects of common genetic variation in oxytocin and vasopressin receptor genes on rhesus macaque social behavior. Am J Primatol 80:e22873. https://doi.org/10.1002/ajp.22873

Madrid JE, Mandalaywala TM, Coyne SP, Ahloy-Dallaire J, Garner JP, Barr CS, Maestripieri D, Parker KJ (2018) Adaptive developmental plasticity in rhesus macaques: the serotonin transporter gene interacts with maternal care to affect juvenile social behaviour. Proc R Soc B 285:20180541

Maestripieri D (2018) Maternal influences on primate social development. Behav Ecol Sociobiol 72:130. https://doi.org/10.1007/s00265-018-2547-x

Maestripieri D, McCormack K, Lindell SG, Higley JD, Sanchez MM (2006) Influence of parenting style on the offspring’s behaviour and CSF monoamine metabolite levels in crossfostered and noncrossfostered female rhesus macaques. Behav Brain Res 175:90–95. https://doi.org/10.1016/j.bbr.2006.08.002

Manson JH, Perry S (2013) Personality structure, sex differences, and temporal change and stability in wild white-faced capuchins (*Cebus capucinus*). J Comp Psychol 127:299–311. https://doi.org/10.1037/a0031316

Martin JE (2005) The influence of rearing on personality ratings of captive chimpanzees (*Pan troglodytes*). Appl Anim Behav Sci 90:167–181. https://doi.org/10.1016/j.applanim.2004.08.019

Martin JS, Suarez SA (2017) Personality assessment and model comparison with behavioral data: A statistical framework and empirical demonstration with bonobos (*Pan paniscus*). Am J Primatol 79:e22670. https://doi.org/10.1002/ajp.22670

Massen JJM, Antonides A, Arnold A-M K, Bionda T, Koski SE (2013) A behavioral view on chimpanzee personality: Exploration tendency, persistence, boldness, and tool-orientation measured with group experiments. Am J Primatol 75:947–958. https://doi.org/10.1002/ajp.22159

Mendle J, Harden K P, Turkheimer E, Van Hulle CA, D'Onofrio BM, Brooks-Gun J, Rodgers JL, Emery RE, Lahey BB (2009) Associations between father absence and age of first sexual intercourse. Child Dev 80:1463–1480. https://doi.org/10.1111/j.1467-8624.2009.01345.x

Moffitt TE (2005) The new look of behavioral genetics in developmental psychopathology: Gene-environment interplay in antisocial behaviors. Psychol Bull 131:533–554. https://doi.org/10.1037/0033-2909.131.4.533

Montiglio P-O, Ferrari C, Réale D (2013) Social niche specialization under constraints: personality, social interactions and environmental heterogeneity. Phil Trans R Soc B 368:20120343. https://doi.org/10.1098/rstb.2012.0343

Munafò MR, Flint J (2004) Meta-analysis of genetic association studies. Trends Genet 20:439–444. https://doi.org/10.1016/j.tig.2004.06.014

Munafò MR, Flint J (2011) Dissecting the genetic architecture of human personality. Trends Cogn Sci 15:395-400. https://doi.org/10.1016/j.tics.2011.07.007

Munafò MR, Zammit S, Flint J (2014) Practitioner review: a critical perspective on gene-environment interaction models - what impact should they have on clinical perceptions and practice? J Child Psychol Psychiat 55:1092–1101. https://doi.org/10.1111/jcpp.12261

Murray CM, Stanton MA, Wellens KR, Santymire RM, Heintz MR, Lonsdorf EV (2018) Maternal effects on offspring stress physiology in wild chimpanzees: chimpanzee maternal effects on offspring stress. Am J Primatol 80:e22525. https://doi.org/10.1002/ajp.22525

Nakagawa S, Schielzeth H (2010) Repeatability for Gaussian and non-Gaussian data: a practical guide for biologists. Biol Rev 85:935-956. https://doi.org/10.1111/j.1469-185X.2010.00141.x

Neumann C, Agil M, Widdig A, Engelhardt A (2013) Personality of wild male crested macaques (*Macaca nigra*). PLoS ONE 8:e69383. https://doi.org/10.1371/journal.pone.0069383

Newman TK, Syagailo YV, Barr CS, Wendland JR, Champoux M, Graessle M, Suomi SJ, Higley JD, Lesch K-P (2005) Monoamine oxidase A gene promoter variation and rearing experience influences aggressive behavior in rhesus monkeys. Biol Psychiat 57:167–172. https://doi.org/10.1016/j.biopsych.2004.10.012

Neyer F J, Lehnart J (2007) Relationships matter in personality development: evidence from an 8-year longitudinal study across young adulthood. J Pers 75:535–568. https://doi.org/10.1111/j.1467-6494.2007.00448.x

Okbay A, Beauchamp, JP, Fontana MA et al (2016) Genome-wide association study identifies 74 loci associated with educational attainment. Nature 533:539–542. https://doi.org/10.1038/nature17671

Plomin R, Bergeman CS (1991) The nature of nurture: Genetic influence on “environmental” measures. Behav Brain Sci 14:373–386. https://doi.org/10.1017/S0140525X00070278

Plomin R, DeFries JC, Knopik VS, Neiderhiser JM (2012) Behavioral Genetics, 6th edn. Worth Publishers, New York, NY

Plomin R, von Stumm S (2018) The new genetics of intelligence. Nat Rev Genet 19:148–159. https://doi.org/10.1038/nrg.2017.104

Réale D, Reader SM, Sol D, McDougall PT, Dingemanse NJ (2007) Integrating animal temperament within ecology and evolution. Biol Rev 82:291–318. https://doi.org/10.1111/j.1469-185X.2007.00010.x

Roberts BW, DelVecchio WF (2000) The rank-order consistency of personality traits from childhood to old age: a quantitative review of longitudinal studies. Psych Bull 126:3–25

Roberts BW, Helson R, Klohnen EC (2002) Personality development and growth in women across 30 years: three perspectives. J Pers 70:79–102

Roberts BW, Walton KE, Viechtbauer W (2006) Patterns of mean-level change in personality traits across the life course: A meta-analysis of longitudinal studies. Psych Bull 132:1–25. https://doi.org/10.1037/0033-2909.132.1.1

Roche DG, Careau V, Binning SA (2016) Demystifying animal “personality” (or not): why individual variation matters to experimental biologists. J Exp Biol 219:3832–3843. https://doi.org/10.1242/jeb.146712

Rogers J, Shelton SE, Shelledy W, Garcia R, Kalin NH (2008) Genetic influences on behavioral inhibition and anxiety in juvenile rhesus macaques. Genes Brain Behav 7:463–469. https://doi.org/10.1111/j.1601-183X.2007.00381.x

Runcie DE, Wiedmann RT, Archie EA, Altmann J, Wray GA, Alberts SC, Tung J (2013) Social environment influences the relationship between genotype and gene expression in wild baboons. Phil Trans R Soc B 368:20120345. https://doi.org/10.1098/rstb.2012.0345

Rushton JP, Brainerd CJ, Pressley M (1983) Behavioral development and construct validity: The principle of aggregation. Psych Bull 94:18–38. https://doi.org/10.1037/0033-2909.94.1.18

Sallis H, Davey Smith G, Munafò MR (2018) Genetics of biologically based psychological differences. Phil Trans R Soc B 373:20170162. https://doi.org/10.1098/rstb.2017.0162

Sanchez-Roige S, Gray JC, MacKillop J, Chen C-H, Palmer AA (2018) The genetics of human personality. Genes Brain Behav 17:12439. https://doi.org/10.1111/gbb.12439

Sapolsky RM, Share LJ (2004) A pacific culture among wild baboons: Its emergence and transmission. PLoS Biol 2:e106. https://doi.org/10.1371/journal.pbio.0020106

Scarr S, McCartney K (1983) How people make their own environments: a theory of genotype greater than environment effects. Child Dev 54:424–435

Sih A, Bell A, Johnson JC (2004) Behavioral syndromes: an ecological and evolutionary overview. Trends Ecol Evol 19:372–378. https://doi.org/10.1016/j.tree.2004.04.009

Sih A, Mathot KJ, Moirón M, Montiglio P-O, Wolf M, Dingemanse NJ (2015) Animal personality and state–behaviour feedbacks: a review and guide for empiricists. Trends Ecol Evol 30:50–60. https://doi.org/10.1016/j.tree.2014.11.004

Silk JB (2002) Kin selection in primate groups. Int J Primatol 23:849-875

Staes N, Koski SE, Helsen P, Fransen E, Eens M, Stevens JMG (2015) Chimpanzee sociability is associated with vasopressin (Avpr1a) but not oxytocin receptor gene (OXTR) variation. Horm Behav 75:84–90. https://doi.org/10.1016/j.yhbeh.2015.08.006

Staes N, Weiss A, Helsen P, Korody M, Eens M, Stevens JMG (2016) Bonobo personality traits are heritable and associated with vasopressin receptor gene 1a variation. Sci Rep 6:38193. https://doi.org/10.1038/srep38193

Stamps J, Groothuis TGG (2010) The development of animal personality: relevance, concepts and perspectives. Biol Rev 85:301–325. https://doi.org/10.1111/j.1469-185X.2009.00103.x

Stevenson-Hinde J, Stillwell-Barnes R, Zunz M (1980) Individual differences in young rhesus monkeys: Consistency and change. Primates 21:498–509. https://doi.org/10.1007/BF02373838

Stevenson-Hinde J, Zunz M (1978) Subjective assessment of individual rhesus monkeys. Primates 19:473–482. https://doi.org/10.1007/BF02373309

Suomi SJ (1997) Early determinants of behaviour: evidence from primate studies. Brit Med Bull 53:170–184

Suomi SJ (2004) How gene-environment interactions shape biobehavioral development: Lessons from studies with rhesus monkeys. Res Hum Dev 1:205–222. https://doi.org/10.1207/s15427617rhd0103_5

Sussman AF, Mates EA, Ha JC, Bentson KL, Crockett CM (2014) Tenure in current captive setting and age predict personality changes in adult pigtailed macaques. Anim Behav 89:23–30. https://doi.org/10.1016/j.anbehav.2013.12.009

Terracciano A, Costa PT, McCrae RR (2006) Personality plasticity after age 30. Pers Soc Psychol Bull 32:999–1009. https://doi.org/10.1177/0146167206288599

Trefilov A, Berard J, Krawczak M, Schmidtke J (2000) Natal dispersal in rhesus macaques is related to serotonin transporter gene promoter variation. Behav Genet 30:295–301

Trillmich F, Müller T, Müller C (2018) Understanding the evolution of personality requires the study of mechanisms behind the development and life history of personality traits. Biol Lett 14:20170740. https://doi.org/10.1098/rsbl.2017.0740

Tucker-Drob EM, Briley DA (2019) Theoretical concepts in the genetics of personality development. In: McAdams DP, Shiner RL, Tackett JL (eds) The handbook of personality development. Guilford, New York, pp 40-58.

Turkheimer E (2000) Three laws of behavior genetics and what they mean. Curr Dir Psychol Sci 9:160–164. https://doi.org/10.1111/1467-8721.00084

Turkheimer E, Harden KP (2013) Behavior genetic research methods. In: Reis HT, Judd CM (eds) Handbook of Research Methods in Social and Personality Psychology, 2nd edn. Cambridge University Press, New York, pp 159–187

Turkheimer E, Pettersson E, Horn EE (2014) A phenotypic null hypothesis for the genetics of personality. Annu Rev Psychol 65:515–540. https://doi.org/10.1146/annurev-psych-113011-143752

Uher J (2008) Comparative personality research: methodological approaches. Eur J Personality 22:427–455. https://doi.org/10.1002/per.680

Uher J (2011) Individual behavioral phenotypes: An integrative meta-theoretical framework. Why “behavioral syndromes” are not analogs of “personality.” Dev Psychobiol 53:521–548. https://doi.org/10.1002/dev.20544

Uher J, Addessi E, Visalberghi E (2013) Contextualised behavioural measurements of personality differences obtained in behavioural tests and social observations in adult capuchin monkeys (*Cebus apella*). J Res Pers 47:427–444. https://doi.org/10.1016/j.jrp.2013.01.013

van den Berg SM, de Moor MHM, McGue M et al (2014) Harmonization of neuroticism and extraversion phenotypes across inventories and cohorts in the genetics of personality consortium: an application of item response theory. Behav Genet 44:295–313. https://doi.org/10.1007/s10519-014-9654-x

van IJzendoorn MH, Bard KA, Bakermans-Kranenburg MJ, Ivan K (2009) Enhancement of attachment and cognitive development of young nursery-reared chimpanzees in responsive versus standard care. Dev Psychobiol 51:173–185. https://doi.org/10.1002/dev.20356

von Borell C, Kulik L, Widdig A (2016) Growing into the self: the development of personality in rhesus macaques. Anim Behav 122:183–195. https://doi.org/10.1016/j.anbehav.2016.10.013

Vukasović T, Bratko D (2015) Heritability of personality: A meta-analysis of behavior genetic studies. Psychol Bull 141:769–785. https://doi.org/10.1037/bul0000017

Watson KK, Li D, Brent LJN, Horvath JE, Gonzalez-Martinez J, Ruíz-Lambides AV, Robinson AG, Skene JH, Platt ML (2015) Genetic influences on social attention in free-ranging rhesus macaques. Anim Behav 103:267–275. https://doi.org/10.1016/j.anbehav.2015.02.012

Weiss A (2017a) A human model for primate personality. Proc R Soc B 284:20171129. https://doi.org/10.1098/rspb.2017.1129

Weiss A (2017b) Exploring factor space (and other adventures) with the hominoid personality questionnaire. In: Vonk J, Weiss A, Kuczaj SA (eds) Personality in Nonhuman Animals. Springer International Publishing, Cham, pp 19–38

Weiss A, Adams MJ, Widdig A, Gerald MS (2011) Rhesus macaques (*Macaca mulatta*) as living fossils of hominoid personality and subjective well-being. J Comp Psychol 125:72–83. https://doi.org/10.1037/a0021187

Weiss A, Inoue-Murayama M, Hong K-W, Inoue E, Udono T, Ochiai T, Matsuzawa T, Hirata S, King JE (2009) Assessing chimpanzee personality and subjective well-being in Japan. Am J Primatol 71:283–292. https://doi.org/10.1002/ajp.20649

Weiss A, King JE (2015) Great ape origins of personality maturation and sex differences: A study of orangutans and chimpanzees. J Pers Soc Psychol 108:648–664. https://doi.org/10.1037/pspp0000022

Weiss A, King JE, Figueredo AJ (2000) The heritability of personality factors in chimpanzees (*Pan troglodytes*). Behav Genet 30:213–221

Weiss A, Staes N, Pereboom JJM, Inoue-Murayama M, Stevens JMG, Eens M (2015) Personality in bonobos. Psychol Sci 26:1430–1439. https://doi.org/10.1177/0956797615589933

Weiß BM, Kulik L, Ruiz-Lambides AV, Widdig A (2016) Individual dispersal decisions affect fitness via maternal rank effects in male rhesus macaques. Sci Rep 6:32212. https://doi.org/10.1038/srep32212

Wilson VAD, Weiss A, Humle T, Morimura N, Udono T, Idani G, Matsuzawa T, Hirata S, Inoue-Murayama M (2017) Chimpanzee personality and the arginine vasopressin receptor 1A genotype. Behav Genet 47:215–226. https://doi.org/10.1007/s10519-016-9822-2

Wolf M, van Doorn GS, Weissing FJ (2008) Evolutionary emergence of responsive and unresponsive personalities. P Natl Acad Sci USA 105:15825–15830. https://doi.org/10.1073/pnas.0805473105

Yamamoto ME, Lopes FA (2004) Effect of removal from the family group on feeding behavior by captive *Callithrix jacchus*. Int J Primatol 25:489–500. https://doi.org/10.1023/B:IJOP.0000019164.98756.9c

Zimmermann J, Neyer FJ (2013) Do we become a different person when hitting the road? Personality development of sojourners. J Pers Soc Psychol 105:515–530. https://doi.org/10.1037/a0033019

Zuur AF, Ieno EN, Walker N, Saveliev AA, Smith GM (2009) Mixed effects models and extensions in ecology with R. Springer, New York, NY

Figure Captions

Fig. 1 The difference between mean-level and rank-order stability

Scenario a): The rank-order of differences in aggressiveness stays stable between all four individuals throughout development, while the mean-level aggressiveness in the population increases with age.

Scenario b): The mean-level aggressiveness in the population stays stable throughout development, while the rank-order of aggressiveness changes between the four individuals over the years.

Figure 1