

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

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## 16 **Abstract**

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

34

## 35 **Keywords**

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

## 38 **Introduction**

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

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

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

63 2001; Réale et al. 2007; Uher 2008; Koski 2011; Carter et al. 2013; Sih et al. 2015; Roche et  
64 al. 2016; Weiss 2017b). As such, we will not rehash this literature. Instead, we will focus on  
65 the development of behavioral variation among individuals. First, we will review the current  
66 knowledge about stability of behavioral differences on a phenotypic level and then proceed to  
67 a more detailed overview of the genetic and environmental contributions to behavioral  
68 stability and change. We hereby will follow the broad conceptual separation common to  
69 research in behavior genetics. Hence by “genetic effects” we refer to behavioral variation due  
70 to differences in the sequence of the DNA of individuals and by “environmental effects” we  
71 refer to all other influences affecting behavioral variation that are not caused by variation in  
72 the individuals’ DNA. Towards the end of our review we will also look at the interplay  
73 between genetic and environmental effects. The review will focus on findings from NHPs but  
74 will be complemented by findings from the human literature where appropriate, that is, if it  
75 provides additional insight.

## 76 **Phenotypic stability over the lifetime**

77 Do aggressive children grow up to be aggressive adults? To answer this and similar questions,  
78 we must distinguish between two types of behavioral stability or change. The first is an age-  
79 related metric called mean-level change, which refers to differences in the mean expression of  
80 a behavioral phenotype at different points in development. Mean-level change can be  
81 quantified with regression analysis where age (or different developmental stages, e.g., being  
82 an infant, juvenile, adult, etc.) is included as predictor of behavioral variation. Ideally, mean-  
83 level change is studied in a longitudinal design, with repeated measurements taken from the  
84 same individuals over time. The second is rank-order change, which is quantified by the  
85 magnitude of relative changes in behavior that occur among individuals within a population.  
86 It is independent of mean-level changes in absolute behavior. An example of a situation where  
87 there is little to no rank-order change would be if children who are highly aggressive relative

88 to their age peers become adults who are highly aggressive relative to their age peers. Rank-  
89 order stability (or change) of behavior may be quantified by two techniques. The first involves  
90 conducting a simple correlation among behavioral measurements from two time points. The  
91 second involves computing the repeatability coefficient, which is an intraclass correlation that  
92 is based on multiple measures per individual and which describes the proportion of total  
93 behavioral variance due to differences between individuals (Boake 1989; Nakagawa and  
94 Schielzeth 2010). If the variance within individuals (between different measurements) is zero,  
95 then repeatability equals one. If the total behavioral variance is solely due to variation within  
96 individuals, then repeatability equals zero. We illustrated the difference between mean-level  
97 and rank-order stability in Fig. 1.

98

#### 99 Mean-Level Change

100

101 Knowledge of lifetime age effects on mean-level change in NHP personality stems especially  
102 from a study by King and colleagues (2008). They used cross-sectional data from  
103 chimpanzees that were divided into five age groups and found age-related differences in terms  
104 of lower extraversion and openness to experience scores, and higher agreeableness and  
105 conscientiousness scores, in older individuals. These results are corroborated by behavioral  
106 measurements from chimpanzees, where boldness and exploration tendency, which are related  
107 in their content to extraversion and openness, respectively, also appear to decline with age  
108 (Massen et al. 2013). Such a pattern could also be partly replicated in and transferred to  
109 orangutans by Weiss and King (2015), with the exception that in this species agreeableness is  
110 lower in older subjects. In common marmosets, females also tend to become less agreeable  
111 with increasing age, while both males and females become less inquisitive (Koski et al. 2017).  
112 The same pattern applies to older white-faced capuchin monkeys who are less agreeable and  
113 less open to new experiences as well (Manson and Perry 2013). So, although individuals are

114 rather stable in their average behavioral propensities in relation to each other, age-related  
115 mean-level differences of behavior occur at the level of the population. Some age-related  
116 patterns seem to be similar across species (e.g., declines in openness / inquisitiveness /  
117 exploration tendency), while the development of agreeableness (indicating pro-social and  
118 tolerant behavior) differs among them. The reasons for developmental differences among  
119 species need to be clarified by future studies. Possible reasons for inter-species differences are  
120 the differing content and structure of the personality dimensions or varying selection  
121 pressures between species (Weiss and King 2015). In a sample of adult rhesus macaques  
122 (Brent et al. 2013), age was largely unrelated to personality dimensions, indicating that mean-  
123 level changes could be especially evident when changes over the lifetime or during early  
124 development are considered. Concerning the latter, strong changes in age-specific behavior  
125 have been reported that are tied to sex-specific life histories (Kulik et al. 2015a, b; von Borell  
126 et al. 2016).

127

## 128 Rank-Order Stability

129

130 In adult NHPs, the rank-order stability of behavioral differences ranges from being moderate  
131 (above  $r=0.3$ ) to high (above  $r=0.5$ ), and is statistically significant (e.g. King et al. 2008;  
132 Weiss et al. 2011; Brent et al. 2013; Weiss 2015). High levels of stability are found most  
133 often in studies that use ratings on questionnaires. Here, estimates of rank-order stability may  
134 be as high or higher than 0.7 (e.g., Stevenson-Hinde and Zunz 1978; King et al. 2008; Weiss  
135 et al. 2011). These estimates reflect the relative stability of *average* behavior of individuals,  
136 that is, the consistency of displaying certain behavioral phenotypes accumulated across  
137 situations (Weiss et al. 2009). If rank-order stability is calculated as repeatability of  
138 behavioral measurements, the resulting repeatability coefficient is typically lower than in  
139 studies using questionnaire ratings (e.g. Brent et al. 2013; Neumann et al. 2013; von Borell et

140 al. 2016), aligning closer to the meta-analytical mean repeatability of 0.37 measured across  
141 species (Bell et al. 2009). It must be noted though that differences in repeatability among non-  
142 aggregated behavioral measurements and aggregated questionnaire ratings could occur  
143 because averaging single ratings into broader dimensions, that is, into personality “factors”,  
144 “domains”, “dimensions”, or “components”, contributes to the stability of these measures  
145 (Rushton et al. 1983). During early ontogeny, the stability of individual differences is  
146 typically lower than in adults (von Borell et al. 2016) and may show substantial variation  
147 from year to year, which may in turn differ across personality domains (Stevenson-Hinde et  
148 al. 1980).

149

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

151

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

162

163 One possible means by which the influence of the environment on behavior could be tested is  
164 by continuously sampling behavioral observations in free ranging animals (von Borell et al.  
165 2016). Yet, the fallacy of behavioral sampling is that observations, for example single

166 incidents of displaying aggressive behavior, are typically also aggregated over time to form a  
167 reliable estimate of individual propensities. Otherwise, rare coincidences, like a generally  
168 unaggressive individual showing a sign of aggression, could lead to unwarranted conclusions  
169 about a general behavioral tendency. Because naturally occurring observations of certain  
170 behaviors may be scarce, aggregation operates usually on relatively large time scales (e.g.,  
171 year-wise or season-wise). Such aggregation limits the possibility of analyzing behavioral  
172 plasticity in response to the environment to long-term fluctuations, stable population  
173 differences, or permanent changes within populations (such environmental effects will be  
174 discussed in the following section). Whether there are developmental influences on short-term  
175 plasticity (i.e., reaction norms; Dingemanse et al. 2010) is thus often not assessed. This is  
176 despite the fact that it might be hypothesized that NHPs become, for example, less flexible in  
177 their behavior with increasing age. Examples from other species show that individuals may  
178 vary in their seasonal plasticity, that they are repeatable in such plasticity (i.e., temporally  
179 consistent in their rank-order of shown plasticity) and that the mean plasticity across  
180 individuals may decrease with age (e.g., in great tits; Araya-Ajoy and Dingemanse 2017).  
181 These findings of differences in plasticity are likely due to frequency-dependent costs or  
182 benefits leading to individually different behavioral strategies. Furthermore, such costs or  
183 benefits are likely to change with experience, leading to mean level changes in plasticity  
184 during ontogenic development (Wolf et al. 2008). The question of age-related variability in  
185 behavioral plasticity appears to be somewhat of a blind spot in the study of NHP behavioral  
186 development. To address this question requires studies that obtain repeated measurements of  
187 behavior-situation interactions within and across time intervals or that can calculate the effect  
188 of age on behavioral reaction norms in cross-sectional data. One way to gather these kinds of  
189 data is by means of behavioral tests that involve simulating situations that an animal may  
190 encounter in the wild (e.g., encountering a novel environment or object, confrontation with  
191 the vocalization of a predator). For NHPs in captivity behavioral tests have been developed to



192 assess behavioral variation among individuals (e.g., Uher et al. 2013; Staes et al. 2016). If  
193 such behavioral tests are conducted with environmental variation or transferred to the natural  
194 habitats of NHPs, this approach allows for a controlled collection of data that may be linked  
195 to short-term environmental fluctuations. For example, tests of social facilitation that compare  
196 behavioral responses to novelty when individuals are alone to when they are in a social  
197 context show short-term environmental effects on behavior (reviewed in Forss et al. 2017). In  
198 common marmosets, the latency to eat novel food is reduced in a social context, but only in  
199 juveniles, suggesting that individual age affects the strength of social facilitation (Yamamoto  
200 and Lopes 2004). Following these results, behavioral reaction norms of neophobia or  
201 exploration tendency with varying social contexts could be further tested in a longitudinal  
202 setting to assess the degree to which individual differences in reaction norms are stable  
203 throughout development, i.e., their rank-order stability. There are also examples of behavioral  
204 tests conducted with NHPs in the wild (e.g., playback experiments in Neumann et al. 2013;  
205 novel-object and novel-food tests in Arnaud et al. 2017). These could be paired with  
206 environmental information (e.g., current group composition, time elapsed since among-group  
207 conflict, etc.) to form behavioral reaction norms and tested for hypothesized age effects,  
208 preferably in a longitudinal design. Other possibilities would be to use data from continuous  
209 observations in a non-aggregated way or aggregating observations according to relatively  
210 short-term environmental fluctuations and analyze them via linear mixed effects models that  
211 can account for zero-inflated observations in the case of rarely observed behaviors (Zuur et al.  
212 2009; Dingemanse and Dochtermann 2013; Brooks et al. 2017). Such an approach would be  
213 informative about relationships between behaviors, between individuals, (correlated) changes  
214 in behavior within individuals, and whether the interaction among behavior and  
215 environmental factors (plasticity) changes with age (Dingemanse and Dochtermann 2013).  
216 For a “how-to” example of using the full potential of linear mixed models when analyzing  
217 behavioral observations of NHPs see Martin and Suarez (2017).

218

219 What do we know from humans?

220

221 Findings from research on human personality development are largely consistent with

222 findings from NHPs. In terms of rank-order stability, humans become more stable throughout

223 their lives, developing from moderate stability (approx.  $r=0.35$ ) in behavioral differences224 during childhood to high stability (approx.  $r=0.70$ ) during late adulthood (Roberts and

225 DelVecchio 2000; Terracciano et al. 2006). Mean-level changes occur primarily during early

226 adulthood, a time often marked by major changes in an individual's environment and

227 increased control over life-history decisions: After a period of decreased psychological

228 "maturity" during early puberty (Denissen et al. 2013), humans typically develop towards a

229 more mature and functional personality in that they become more agreeable, conscientious

230 and show more emotional stability (Roberts et al. 2006; Donnellan et al. 2007). However,

231 they also tend to become less flexible (Roberts et al. 2002).

232

**233 Determinants of Plasticity and Stability in Behavior**

234 Now that we know that behavioral variation among individuals is not fixed and that rank-

235 order and mean-level changes occur in particular during childhood, adolescence, and young

236 adulthood, the question remains how these changes can be explained. We propose to approach

237 questions about behavioral stability and change using a behavior genetics framework, because

238 it helps us to disentangle whether and how behavioral development is caused by

239 environmental influences, genetic effects, or their interplay.

240

241 Genetic Effects on Behavioral Development

242

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

258  
259 To provide a general impression of how heritable personality traits are in NHPs, we calculated  
260 the median and range of published estimates of narrow-sense heritability across NHP species  
261 and studies (see Tables S1, S2 in the supplement). For personality factors we calculated a  
262 median heritability of  $h^2=0.25$  and a range from 0.00 to 0.63 (based on the studies from Weiss  
263 et al. 2000; Fairbanks et al. 2004; Adams et al. 2012; Brent et al. 2013; Johnson et al. 2015;  
264 Latzman et al. 2015; Staes et al. 2016; Wilson et al. 2017; Inoue-Murayama et al. 2018). The  
265 heritability of single behaviors appears to be very similar, with a median  $h^2=0.25$  and range of  
266 0.11 to 0.91 (based on studies by Rogers et al. 2008; Fawcett et al. 2014; Hopkins et al. 2014,  
267 2015; Johnson et al. 2015; Watson et al. 2015). Non-additive genetic effects may contribute a  
268 significant proportion to genetically influenced variance, leading to higher broad-sense

269 heritability estimates ( $H^2$ ). Based on a study on orangutans we calculated a median  $H^2$  of 0.69  
270 (Adams et al. 2012). Published estimates of broad-sense heritability are, however, an  
271 exception, as this requires extended study designs including twins or a large number of full-  
272 and half-siblings (ibid.). Unfortunately for a developmental perspective, we do not know of  
273 longitudinal studies that published heritability estimates for a birth cohort across time. Nor do  
274 we know of cross-sectional estimates of heritability along different developmental stages.  
275 Hence, we cannot say whether the heritability estimates of personality traits, and thus  
276 influences relating to environmental factors, increase or decrease throughout development.

277  
278 In humans the average heritability estimated from meta-analyses is a little higher than in  
279 NHPs, accounting for about 40% of variation (Turkheimer et al. 2014; Vukasović and Bratko  
280 2015). Interestingly, estimates coming from family and adoption studies, that include only  
281 additive genetic effects, have an average effect size of 0.22 (Vukasović and Bratko 2015),  
282 which is close to the median effect size we calculated for narrow-sense heritability in NHPs.  
283 This percentage may rise to about 50% when only data from twin studies is considered (van  
284 den Berg et al. 2004; Vukasović and Bratko 2015) as these estimates reflect the broad-sense  
285 heritability. From a developmental perspective, we know that the heritability of personality  
286 tends to decrease with increasing age, dropping from roughly 75% during infancy and early  
287 childhood down to the above-mentioned estimate of 40% in later adulthood (Briley and  
288 Tucker-Drob 2017). Thus, in the period after birth, individual differences in behavior are  
289 largely influenced by genetic effects, with the role of environmental effects increasing with  
290 age.

291  
292 The increasing role of the environment is also reflected in its contribution to the increase in  
293 the rank-order stability of personality (from  $r=0.35$  in infancy to about  $r=0.70$  in adults; see  
294 above), which can be explained by genetic or environmental influences. Here twin studies

295 find that the genetic contribution remains at a steady 35% during the lifespan, while the  
296 environmental contribution increases to account for an additional 35% of rank-order stability  
297 during development. This means that the stable proportion of behavioral variation is almost  
298 entirely genetically influenced during infancy, but that the post-infancy stability increase is  
299 almost entirely influenced by environmental factors (Tucker-Drob and Briley 2019).

300

### 301 Environmental Effects

302

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

310

### 311 *Stressful Life Experiences*

312 Environmental stressors influence behavioral development during prenatal or very early life  
313 stages. For example, low food availability is linked to higher prenatal maternal stress in  
314 Assamese macaques, which leads to increased growth, but decreased motor skill acquisition  
315 and reduced immune function in their offspring (Berghänel et al. 2016). Although this  
316 evidence is circumstantial, life-history trade-offs such as these may extend to the development  
317 of individual differences in related behavioral traits, for example a trade-off between playing  
318 and growth (Berghänel et al. 2015). Fertility is also affected by low-quality early  
319 environments with individual differences being linked to drought years in baboons (Lea et al.  
320 2015). Next to the quality of the environment, effects of the dominance hierarchy have been

321 documented as a lasting stressor in NHP development. In chimpanzees, for example, maternal  
322 rank during pregnancy is not only related to the stress response of the mother, but also to the  
323 stress response of her dependent offspring, and especially males thereof (Murray et al. 2018).  
324 A relationship between maternal or individual rank and behavioral differences, and especially  
325 those relating to aggressive and fearful/bold behavior, has been shown for NHPs of different  
326 ages (e.g., French 1981; Bolig et al. 1992; Brent et al. 2013; von Borell et al. 2016). In an  
327 experimental manipulation, Kohn and colleagues (2016) showed that climbing up the  
328 dominance hierarchy was causally related to changes in social approachability and boldness.  
329 We can thus expect changes in the dominance hierarchy as a possible source of  
330 environmentally induced variation in personality development. Related evidence stems from a  
331 case of severe and selective tuberculosis infection in wild baboons, where the more aggressive  
332 individuals of a troop died at once, because they ate from a neighboring troop's food resource  
333 that was infected. These deaths led to an overall more tolerant social style in the troop. While  
334 dominance interactions were concentrated among closely ranked individuals, high-ranking  
335 individuals were more tolerant of very low-ranking individuals. The latter finding was related  
336 to a disproportionately high number of reversals in the direction of dominance among  
337 individuals far apart in rank (Sapolsky and Share 2004). This is in line with the argument that  
338 high-ranking individuals can typically afford aggressive or displacing behavior due to  
339 agonistic support from other individuals (Silk 2002), which was apparently less the case in the  
340 newly stratified troop of baboons after the epidemic infection.

341  
342 Although the quality of the natural environment and dominance hierarchies in social groups  
343 affect behavioral differences from early life on, new challenges arise around the time of  
344 maturation that drive behavioral variation. A prominent example in NHPs is the migration  
345 from the natal group to a new group (natal dispersal). Migration is typically accompanied by  
346 increases in mortality or injury rates, decreases in access to resources, and social costs, i.e.,

347 the loss of social ties or rank (Dittus 1979; Weiß et al. 2016). Following migration, male  
348 rhesus macaques show more fearful and less physically aggressive behavior than before (von  
349 Borell et al. 2016), which is consistent with findings from captive pigtailed macaques, where  
350 individuals that are new to a facility are more cautious (Sussman et al. 2014). Migration may  
351 also trigger rank-order changes in behavior, possibly reflecting different reactions or  
352 strategies following migration. In the study of von Borell et al. (2016) this was reflected in  
353 very low or even negative correlations among fearful behaviors measured in the year before  
354 and after migration, despite their overall lifetime repeatability. In female rhesus macaques, the  
355 birth of the first infant is a similar developmental milestone and is marked by a decreased  
356 frequency of initiating social contacts outside of maternal kin (von Borell et al. 2016).

357

### 358 *Maternal Influences and Rearing*

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

363

364 Differences in maternal style are typically described along the two dimensions protectiveness  
365 and rejection, but may vary a little between NHP species, that is, maternal behaviors may also  
366 load on three different factors in a factor analysis (De Lathouwers and Van Elsacker 2004).

367 Protectiveness and rejection have been linked to individual differences in behavior across  
368 various age-stages in NHP development. For example, in an observational study of Japanese  
369 macaques, infants of highly protective mothers showed lower levels of exploratory behavior  
370 and interacted less with their group members. On the other hand, infants of mothers who  
371 rejected them interacted more than average with other group members. These effects  
372 diminished, however, over the course of development and were present mostly during early

373 infancy (Bardi and Huffman 2002). A stable effect of maternal style was reported by Bardi  
374 and colleagues (2015) who found that juvenile baboons that experienced more stress-related  
375 interactions with their mother during early life showed higher locomotor activity and cortisol  
376 levels during a stress test than individuals that experienced more affiliative mother-offspring  
377 interactions.

378  
379 Such effects of parental care or mother-offspring interaction were further supported by  
380 experimental studies. An effect of maternal protectiveness on offspring caution was shown in  
381 vervet monkeys (Fairbanks and McGuire 1993). In this study maternal protectiveness was  
382 experimentally increased by introducing new males to some housing groups. Infants and  
383 juveniles of mothers from the “protective” condition showed higher latencies to approach a  
384 novel object, indicating increased caution. Approach latencies were highly correlated among  
385 mothers and infants but not among mothers and juveniles. These results indicate that a  
386 mixture of environmental and genetic effects contributed to the development of behavioral  
387 differences. Maestriperi and colleagues (2006) could not find an effect of maternal  
388 protectiveness on offspring behavior in rhesus macaques, but they did find that higher  
389 maternal rejection led to more solitary play in offspring. This effect did not differ between  
390 mother-reared and cross-fostered individuals, ruling out the possibility that this observation is  
391 simply driven by genetic similarity between mothers and their offspring.

392  
393 A special case of maternal influence on behavioral differences is maternal deprivation or the  
394 disruption of maternal care. Rhesus macaques that spent their first year of life in total  
395 isolation showed hardly any positive social responses or activities afterwards and were also  
396 consistently fearful. Individuals who spent shorter periods of time in isolation showed a  
397 behavioral pattern similar to that of monkeys who spent a year in isolation, followed by  
398 highly individualized (adequate and non-adequate) adaptations to social situations,



399 presumably based on inherited individual differences and unique learning experiences  
400 (Harlow et al. 1965). Similar differences in the social response to short periods of isolation  
401 have been documented in free-ranging rhesus macaque infants (Berman et al. 1994). Here,  
402 increased short-term separations of mothers and their infants, which occurred when the  
403 mothers resumed mating, led to increased distress in the infants. Like the captive infants,  
404 described by Harlow and colleagues (1965), who were isolated for short periods, the free-  
405 ranging infants developed differing social responses to and after the separation events.  
406 Specifically, some infants reacted with social withdrawal and decrease of social play and  
407 others rather increased their social behavior like grooming. Differential responses to maternal  
408 separation or maternal style, whether marked by decreased or increased social behavior, have  
409 been linked with genetically inherited differences in stress responsivity (Clarke and Boinski  
410 1995; Suomi 2004). Further studies of maternal separation in captivity, typically on hand-  
411 raised and later on peer-reared individuals, suggest temporally consistent increases in anxious,  
412 shy, and impulsive behavior in comparison with their mother-reared counterparts. These  
413 behavioral differences may extend to neglectful or abusive maternal behavior, when peer-  
414 reared females become mothers themselves (reviewed in Soumi 1997). More recent studies,  
415 albeit in a different species, show mixed results: while nursery-reared chimpanzees were  
416 reported to be less agreeable and more extraverted than their mother-reared counterparts  
417 (Latzman et al. 2015), a similar study of chimpanzees found no such differences between  
418 these groups (Martin 2005).

419

420 The effects of differential care appear to extend to scenarios where the intensity of human care  
421 varies. Young chimpanzees who experienced enhanced responsive care were less distressed  
422 and showed less disorganized attachment than chimpanzees who only received a minimal  
423 standard of care from human caregivers (van IJzendoorn et al. 2008). In addition to maternal  
424 style, maternal separation, and the amount of care, the time infants spend with conspecifics

425 seems to affect personality development. For example, chimpanzees who as infants spent less  
426 time with conspecifics were rated as being less extraverted later in life than individuals who  
427 spent more time with conspecifics (Freeman et al. 2016).

428

#### 429 *Issues of causality*

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

435        “The situation and the individual’s environmental history may set the stage and limit  
436        the range of choice of action, but the individual’s genotype is involved both in the  
437        actions taken and the individual’s presence in this situation in the first place. We  
438        cannot understand development without taking this into consideration.”

439 Among the findings on stressful life events or rearing experience reviewed above,  
440 experimentally separated environmental effects rely largely on captive NHPs, while in studies  
441 conducted in the wild, environmental and genetic effects can be confounded. There are  
442 several mechanisms of such confounding. Prominent examples include gene-environment  
443 correlations (rGE) and gene-environment interactions (G x E), both of which will be  
444 discussed below. The main message at this point is, that a neglect of genetic information can  
445 lead to premature causal interpretations of the role the environment may play in behavioral  
446 development (Briley et al. 2018). For example, the association between early adversity and a  
447 faster life-history strategy that has been reported in NHPs, has received theoretical and  
448 empirical support from the human literature as well, leading, for example, to earlier puberty  
449 and marriage (see reviews by Belsky 2012; Del Giudice 2014). However, findings of life-  
450 history embedded behavioral differences related to early adversity did not hold up in a study

451 design that included information of genetic relatedness based on pedigrees to control for  
452 genetic confounding. Mendle and colleagues (2009) found that the association among father  
453 absence and timing of first intercourse in humans was best explained by genetic risk factors  
454 that correlate both with father absence and early sexual activity, diminishing the role of the  
455 mere *experience* of an absent father. Likewise, decisions involving changes in the social  
456 environment, such as NHP dispersal, are known to carry a genetic component (Trefilov et al.  
457 2000; Krawczak et al. 2005) that could also be correlated to behavioral differences. Also,  
458 relationships between rank and behavior may partly be affected by feedback processes  
459 entailing a genetic component, for example the interplays of aggressive behavior, which has a  
460 heritable component, and changes in the dominance hierarchy in male NHPs (Koyama 1970;  
461 Bernstein 1976). In humans, some studies on personality development try to test whether  
462 environmental effects are causal by including a control group. Examples can be found in  
463 studies on personality development during periods of spatial and social transformation in  
464 human adolescents or young adults: events like a high-school student exchange (Hutteman et  
465 al. 2015), studying abroad as college student (Zimmermann and Neyer 2013), graduation from  
466 high school (Bleidorn 2012), or forming a partner relationship (Neyer and Lehnart 2007)  
467 mostly trigger a development towards personality maturation compared to the control group,  
468 i.e., increases in conscientiousness, agreeableness and self-esteem, and a decrease in  
469 neuroticism. Going abroad was also related to increases in openness to new experiences. The  
470 inclusion of a control group is certainly an improvement over not including a control group,  
471 as it can be the case in related studies of NHP migration in the wild, where it is often difficult  
472 to gather a control group with similar characteristics and a similar sample size as the  
473 migrating individuals. Yet, in naturally occurring control group designs, such as the above-  
474 described human studies, the decision of whether to participate and the behavioral differences  
475 among individuals of the control and quasi-experimental groups may be influenced by  
476 common genetic effects. Even if both groups have been matched to be similar in their

477 behavioral characteristics prior to the environmental change, this change may only activate or  
478 amplify a genetic predisposition of a behavioral tendency, for example, being open to new  
479 experience that was already entailed in the decision of participating in this event.

480

481 In the human literature, the impact of individuals' genetic background on behavior or (life-  
482 history) decisions (e.g., student exchange, marriage, etc.) led to the "first law of behavior  
483 genetics" that all traits are heritable (Turkheimer 2000). It follows that behavior-environment  
484 correlations cannot be interpreted as *prima facie* evidence of a *causal* environmental influence  
485 without considering that such associations are probably genetically mediated (Johnson et al.  
486 2011; Johnson and Penke 2014; Turkheimer et al. 2014). Accordingly, calls for genetically  
487 informed designs in the study of behavior-environment associations have been pointed out in  
488 primatology (e.g. Adams 2014; Brent and Melin 2014) and psychology (Turkheimer and  
489 Harden 2014), that could control for a genetic basis of differences in the environment that  
490 individuals experience. For example, studies looking at the effects of migration on behavioral  
491 differences among individuals could control for the possibility that both share a common  
492 genetic basis. Briley and colleagues (2018) reviewed techniques that are capable of tackling  
493 questions of causality in longitudinal, and even cross-sectional, genetically informative data  
494 (i.e., data where behavioral outcomes and measurements of the environment are paired with  
495 information about relatedness or molecular genetic similarity among individuals). For  
496 example, in a quantitative genetic design, direction-of-causation modeling (DOC modeling)  
497 can be used to estimate the plausibility of a causal direction among an environmental and a  
498 behavioral measure. This approach involves comparing the proportion of variance attributable  
499 to genetic, shared, and nonshared environmental effects in the possible cause and outcome. If,  
500 for example, differences in maternal style have a large genetic component and causally  
501 explain behavioral differences among children, then a genetic component should be  
502 represented in the children's behavioral differences as well. Comparing the fit of different

503 models with alternative directions of causality can help to assess the likelihood of a  
504 hypothesized cause-outcome-relationship (for details see Briley et al. 2018). In human female  
505 twins, DOC modeling showed that parental behavior was more likely the cause of  
506 psychological distress than psychological distress being the cause of parental behavior (i.e.,  
507 the model specifying a causal relationship from parental behavior to distress had a better fit  
508 than the other way around; Gillespie et al. 2003).

509

### 510 Gene-environment interplay

511

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

526

527 In some species, it is possible to conduct controlled experiments on developmental  
528 psychobiology that allow for a separation of genetic and environmental effects (e.g., by

529 breeding genetically identical individuals in identical conditions; Kain et al. 2012; Bierbach et  
530 al. 2017), but ethical and practical reasons mostly prevent scientists from applying these  
531 methods to humans or NHPs (Turkheimer 2000; but see experimental manipulations of  
532 rearing conditions presented above). Yet, there is no need for primatologists or psychologists  
533 to stop searching for the causes of development. Although we may not be able to causally  
534 reconstruct complex developmental pathways, we can test how genes and the environment  
535 correlate and interact in specific scenarios and how likely they are to shape behavioral  
536 development within the limits of such scenarios.

537

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

554

555 G x E effects on personality development can be detected by quantitative or molecular  
556 genetics methods. Quantitative genetic studies test whether differences in a phenotype  
557 between individuals are associated with information on their genetic relatedness (for example  
558 based on known pedigrees), while molecular genetic studies try to associate differences in a  
559 phenotype with a specific pattern of variation in DNA sequence among individuals. In  
560 behavioral genetic research, the latter's emphasis is on trying to find associations between  
561 genetic variants at specific genetic loci and behavioral traits (candidate gene association  
562 study) or trying to associate a large number of variants that are spread across the genome with  
563 a behavioral trait (genome-wide association study, GWAS). In a quantitative genetics  
564 framework, Lutzman and colleagues (2015) have shown that heritability estimates of  
565 personality dimensions vary among mother- and nursery-reared chimpanzees. Specifically,  
566 they found lower heritability estimates in nursery-reared individuals indicating that their  
567 atypical environmental circumstances at an early age led to a higher proportion of  
568 environmentally influenced behavioral variation among their traits. Results from humans also  
569 support interaction effects of rearing quality and genes. For example, Krueger and colleagues  
570 (2008) showed that the genetic influence on adolescent personality varied with the levels of  
571 regard they received from their parents. In particular, low levels of regard were associated  
572 with an increased environmental contribution to phenotypic variance. On a molecular level,  
573 many NHP studies have examined the interplay of environmental variation and candidate  
574 genes in their contribution to behavioral differences. These studies analyzed for example  
575 polymorphisms in genes such as 5-HTTLPR (Barr et al. 2004; Madrid et al. 2018), MAOA  
576 (Newman et al. 2005), and COMT (Gutleb et al. 2017), which often, but not exclusively, were  
577 reported to interact with differences in rearing condition (for a review see Rogers 2018).  
578

579 In the molecular genetics area, studies of NHPs and humans used to be closely linked and  
580 shared a desire to identify the genetic underpinnings of behavioral or pathological variation by

581 testing the effects of candidate genes (see, e.g., Caspi et al. 2002, 2003 on G x E in humans,  
582 including MAOA and 5-HTTLPR variation affecting violence and depression, respectively).  
583 However, meta-analyses and recent studies in humans that use samples that are several  
584 magnitudes larger in size and extensive genome-wide genetic information led to the  
585 conclusion that complex behavioral traits are unlikely to be substantially influenced by single  
586 genes (Munafò and Flint 2004; Plomin and von Stumm 2018; Sallis et al. 2018). That does  
587 not mean that genetic polymorphisms in single genes do not matter, but that their effects are  
588 usually too small to be detected with the sample sizes of earlier studies, and this is especially  
589 the case when they are modeled in interactions with environmental gradients. Reviews of  
590 human candidate gene studies show that many associations cannot be replicated across studies  
591 and in meta-analyses, and that the effect sizes of statistically significant associations in earlier  
592 studies were often inflated (e.g. Sanchez-Roige et al. 2018). These findings led researchers to  
593 conclude that the literature on associations among common variants in candidate genes and  
594 behavior, for both main effects and G x E interactions, is awash with false positive results  
595 (Sallis et al. 2018). Genome-wide association studies that explore associations of common  
596 genetic variants and behavior throughout the whole genome show that a large number of  
597 genetic variants (single nucleotide polymorphisms; SNPs) contribute to the heritability of  
598 complex traits, however with small effect sizes. Replicated SNPs typically explain less than  
599 0.1% of the phenotypic variance (Munafò et al. 2014; Sallis et al. 2018). While many SNPs  
600 reported in candidate gene studies did not replicate in sufficiently powered GWAS (e.g.,  
601 Chabris et al. 2012), many variants that met genome-wide significance levels that have been  
602 identified in GWAS could be replicated in large independent samples (> 100,000 individuals;  
603 e.g., Okbay et al. 2016). These variants are spread broadly across the genome, including  
604 intragenic regions that do not code for proteins (Boyle et al. 2017; Sanchez-Roige et al. 2018).  
605 Additionally, extended study designs show that rare genetic variants that are not tagged in  
606 GWAS can contribute to individual variation in complex traits (Hill et al. 2018). While these



607 findings and conclusions stem from human studies, they are likely to apply to NHP studies as  
608 well (Munafò et al. 2014). That is not to say that all statistically significant results stemming  
609 from NHP candidate-gene or GxE studies are false positives. Some gene-behavior  
610 associations have replicated across populations, species, and behavioral measures (reviewed  
611 in Weiss 2017a; Rogers 2018). For example, variants in the arginine vasopressin receptor 1A  
612 gene (AVPR1A) appear to replicate across different samples of chimpanzees (Anestis et al.  
613 2014; Hopkins et al. 2014; Staes et al. 2015; Wilson et al. 2017), bonobos (Staes et al. 2016)  
614 and common marmosets (Inoue-Murayama et al. 2018). However, the combination of small  
615 sample sizes and relatively large effects of reported genetic variants is similar to the early  
616 wave of human studies in the field of behavior genetics. It is thus probably worth retaining  
617 one's skepticism about this literature. Reported effect sizes of replicated genetic variants in  
618 NHPs (e.g. given in Staes et al. 2015 and Wilson et al. 2017 for AVPR1A) are several  
619 magnitudes larger than most of the extensively studied candidate-gene variants and GWAS  
620 results in humans (see Sanchez-Roige et al. 2018 for a review). It is possible that the  
621 development and the social influences on behavioral variation among humans are more  
622 complex and thus less influenced by single genetic variants. Also, studies on captive NHPs  
623 provide a more restricted and controlled environment (e.g., controlled diet, less habitat  
624 variation), which might lead to stronger genetic effects. A recent study on the effects of  
625 variants in OXTR and AVP receptor genes (AVPR1A, AVPR1B) on behavior in rhesus  
626 macaques, however, failed to replicate previous results and showed only very small effects of  
627 the 12 SNPs that were examined (Madlon-Kay et al. 2018). Alongside the emerging  
628 consistency of findings that single genetic variants have only small effects on complex traits,  
629 Madlon-Kay and colleagues (2018) discuss other methodological difficulties, including  
630 missing control of genetic relatedness within the population and/or missing adjustment of p-  
631 values, that raise doubt about earlier positive results.

632

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

644

645 In the concluding lines of this section we want to provide a glimpse into the emerging field of  
646 epigenetics. Epigenetics refers to processes whereby environmental signals affect genetic  
647 variation by mechanisms such as DNA methylation or histone modification. Briefly, these  
648 environmentally induced mechanisms can lead to individual differences in gene transcription  
649 and expression, which can result in behavioral differences (Kaminsky et al. 2008). In baboons,  
650 for example, Runcie and colleagues (2013) found that different aspects of the social  
651 environment and social behavior (social connectedness, group size, and maternal dominance  
652 rank) interacted with the genotype by means of differences in gene expression along these  
653 environmental or behavioral gradients. This suggests that social behaviors, like grooming, are  
654 not only influenced by genetic variation, but also influence genetic variation. From an  
655 ontogenetic perspective, this means that genes are not destiny for the development of  
656 personality, but rather that the environment can alter the genetic tracks individuals are set on.  
657 The precise way in which epigenetic mechanisms function in relation to complex traits, as social  
658 behavior, is under current investigation (Hu and Barrett 2017). First evidence on the behavioral

659 level indicates, for example, the potential role of epigenetics in the stress response system and  
660 associated behavioral differences such as risk-taking or novelty-seeking (Laviola et al. 2003;  
661 Kaminsky et al. 2008; Canestrelli et al. 2016). Also epigenetic mechanisms in the domain of  
662 memory formation and learning (Duke et al. 2017) may transfer to behavioral differences  
663 among individuals. But until we have replicated evidence of epigenetic effects on behavioral  
664 traits, a degree of humility about these findings would seem appropriate (see also Cobben and  
665 van Oers 2016). In particular, epigenetic explanations centering on specific genes should be  
666 interpreted carefully, as associations among single genes and behaviors often do not replicate  
667 in studies of humans and NHPs (see above). Given the increasing general understanding of  
668 genome-wide DNA methylation patterns in humans and NHPs (Lea et al. 2016, 2018), the role  
669 of epigenetics in personality development could become an interesting area of future research  
670 (Trillmich et al. 2018).

671

## 672 **Summary and Outlook**

673 We can infer that behavioral differences among individual NHPs develop towards increasing  
674 rank-order stability and a pattern indicative of what has been described as a “mature”  
675 personality in humans (but see exceptions in Manson and Perry 2013; Weiss and King 2015;  
676 Koski et al. 2017). Whereas environmental influences on behavioral variation among  
677 individuals act in humans especially around the time of adolescence and young adulthood,  
678 behavioral variation in NHPs seems to already be affected early in life. Among these early  
679 environmental influences are stress-related variation in the natural environment, parenting  
680 style or rearing conditions. Later in life, migration or maternity during young adulthood may  
681 also affect personality development. As a complex interplay among genotype and the  
682 environment is likely, and the statistical power to detect even two-way interactions is low,  
683 current research is still far from disentangling the causal pathways that lead to behavioral  
684 differences. We propose that one possible way to peek inside this “black box” is to conduct

685 genetically informed longitudinal studies or to use cross-sectional DOC modeling  
686 (Turkheimer and Harden 2014; Briley et al. 2018). That said, studies have to be adequately  
687 powered if they wish to use these tools. Since statistical power often turns out to be a problem  
688 in NHP studies, one possible direction might be to identify polygenic scores for behavioral  
689 differences in relatively large samples of a species, for example in breeding facilities, and  
690 then to apply this knowledge to the typically smaller populations in the wild or in other  
691 captive settings, such as zoos or sanctuaries. This could enable one to conduct genetically  
692 informative studies without the need for pedigree data or could supplement studies with  
693 (partly) existing pedigree data. Furthermore, testing evolutionary hypotheses stating under  
694 which conditions correlations among behavioral differences will occur and how stable these  
695 correlations are under changing environments or selection regimes (see Sih et al. 2004;  
696 Dochtermann and Dingemanse 2013) could be a fruitful direction for primate personality  
697 research. An example would be to test whether environmental variation affecting food  
698 resources favors different behavioral strategies or correlations among behaviors that form  
699 behavioral syndromes (Dingemanse et al. 2004). Human studies could also be informed, or  
700 inspired by, the increasing knowledge of dominance rank and hierarchy effects on behavioral  
701 variation in NHPs.

702

### 703 **Compliance with Ethical Standards**

#### 704 **Conflict of interest**

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

706

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**1155 Figure Captions**

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**1157 Fig. 1 The difference between mean-level and rank-order stability**

1158 Scenario a): The rank-order of differences in aggressiveness stays stable between all four  
1159 individuals throughout development, while the mean-level aggressiveness in the population  
1160 increases with age.

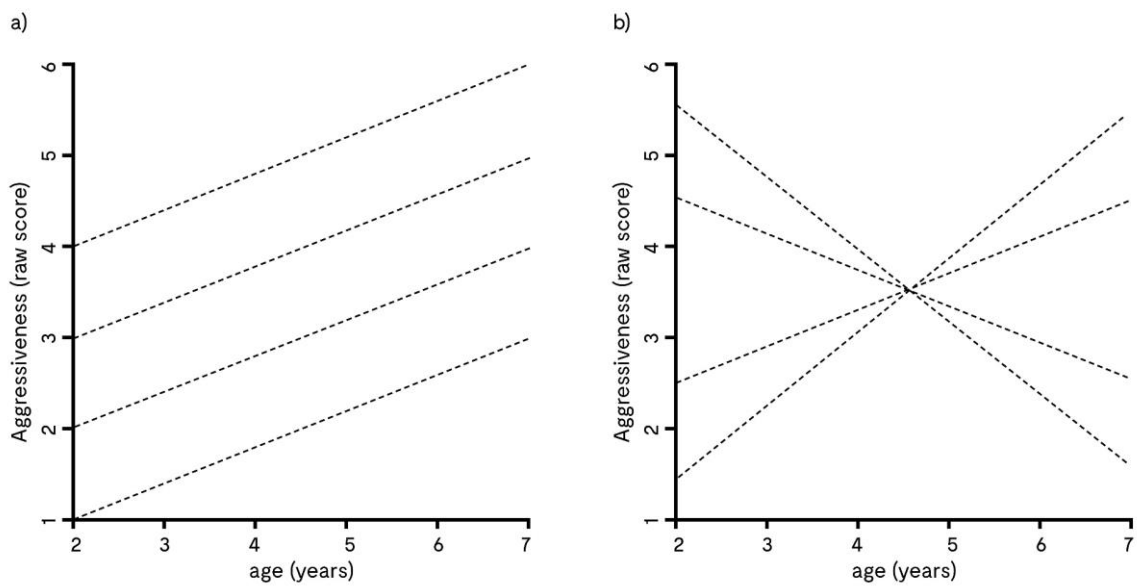
1161 Scenario b): The mean-level aggressiveness in the population stays stable throughout  
1162 development, while the rank-order of aggressiveness changes between the four individuals  
1163 over the years.

1164

1165

1166 **Figure 1**

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