Evidence for Shared Genetic Dominance Between the General Factor of Personality, Mental and Physical Health, and Life History Traits

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We reanalyze previously published data on 309 MZ and 333 DZ twin pairs aged 25 to 74 years from the MIDUS survey, a nationally representative archived sample, to examine how much of the genetic covariance between a general factor of personality (GFP), a lower-order life history factor, and a general physical and mental health factor, is of the nonadditive variety. We found nonadditive genetic effects (D) could not be ruled out as a contributor to the shared variance of these three latent factors to a Super-*K* Life History factor. We suggest these genetic correlations support the view that a slow (*K*-selected) life history strategy, good health, and the GFP coevolved and are mutually coadapted through directional selection.

Keywords: Big One, heritability, nonadditive genetic variance, general factor of personality

A recent observation is that a General Factor of Personality (GFP) occupies the apex of the multi-factorial hierarchy in the same way that *g*, the general factor of mental ability, occupies the apex in the organization of cognitive abilities. Individuals high on the GFP are altruistic, agreeable, relaxed, conscientious, sociable, and intellectually open, with high levels of wellbeing, satisfaction with life, self-esteem, and emotional intelligence. The explanation we favor for the GFP is that, like *g*, it arose through evolutionary selection for adaptive traits that facilitate performance across a wide range of contexts (Rushton et al., 2008). The main alternative interpretation of the GFP (and other higher-order factors above the Big Five) is that they arise from artifacts of evaluative bias and scale construction (Ashton et al., 2009; Bäckstrom et al., 2009; McCrae et al., 2008).

The GFP has been found across diverse samples, procedures, and inventories. The inventories so far include several sets of the Big Five, the California Psychological Inventory, the Comrey Personality Scales, the Dimensional Assessment of Personality Pathology, the EAS Temperament Scales, the Guilford-Zimmerman Temperament Survey, the Hexaco Personality Inventory, the Hogan Personality Inventory, the Jackson Personality Inventory, the Millon Clinical Multiaxial Inventory-III, the Minnesota Multiphasic Personality Inventory-2, the Multidimensional Personality Questionnaire, the Personality Assessment Inventory, the Temperament and Character Inventory, and the Trait Emotional Intelligence Questionnaire (Figueredo et al., 2004, 2007; Musek, 2007; Rushton et al., 2008, 2009; Rushton & Irwing, 2008, 2009a, 2009b, 2009c, 2009d; Veselka et al., 2009a, 2009b).

The main empirical impetus to finding the GFP was the increasing overlap and redundancy found among personality measures, including substantial genetic pleiotropy within and across factors (Ando et al., 2004; Jang et al., 2006; Jang et al., 2002; Weiss et al., 2008). One study found the GFP was independent of method variance using a multitrait-multimethod analysis of self-, teacher-, and parent-ratings of 391 13- to 14-year-olds on the Big Five Questionnaire — Children (Rushton et al., 2009). Several cross-national twin studies have found 50% of the variance on the GFP is attributable to genetic influence and 50% to non-shared environmental influence, including from 322 pairs of twins in the United Kingdom, 575 pairs of 2- to 9-year-old twins in South Korea, 651 pairs of 14- to 30-year-old twins in Japan, and 386 pairs of 18- to 74-year-old twins in Canada and the United States (Figueredo et al., 2004; Rushton et al., 2008, 2009; Veselka et al., 2009a, 2009b). Moreover, the GFP is largely a *genetic* factor, as indicated by the cross-twin cross trait correlations that give rise to it being greater among MZ twin pairs than among DZ twin pairs. The South Korean twin

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data showed the GFP had emerged by 2- to 3-years of age (Rushton et al., 2008).

In two twin studies, Rushton et al. (2008) found the genetic variance was primarily of the nonadditive variety (dominance and epistasis). For example, 53% of the variance in the GFP extracted from a Prosocial Questionnaire and the EAS temperament scales was nonadditive in a study of 575 pairs of 2- to 9-year-old Korean twins rated by their mothers. Nonadditive genetic variance is important because it indicates a trait has been under recent natural selection, which is expected for a Darwinian fitness character, i.e., one that leads to greater reproductive success (Fisher, 1954; Falconer, 1989). Nonadditive genetic variance has been found for numerous personality scales as well as for cognitive abilities, albeit sometimes only in small quantities (e.g., Bouchard & McGue, 2003; Hur, 2007; Jensen, 1998; Johnson et al., 2008; Lykken et al., 1992). Support for genetic dominance also comes from the preliminary evidence of inbreeding depression on components of the GFP, just as there is for the *g* factor of mental ability (Jensen, 1998). Inbreeding depression occurs on a trait when deleterious recessive alleles combine to lower the scores of offspring relative to parents. Thus, an Italian study found inbred families were lower in extraversion and openness to experience (Camperio Ciani et al., 2007). A Dutch study revealed that the offspring of parents who came from the same region in the Netherlands (and so were more likely to be inbred) scored lower on sensation seeking than those whose parents came from different regions (Rebello & Boomsma, 2007).

The existence of a GFP poses interesting theoretical questions regarding its evolutionary and genetic origins. Rushton et al. (2008) suggested that because individuals at the positive pole of the GFP possessed more cooperative and prosocial personalities, they left more progeny than those at the negative pole, since people prefer as mates, fellow workers, and leaders, those who are agreeable and emotionally stable. This view of social and sexual selection was initially proposed by Darwin (1871), who suggested that directional selection acted to endow contemporary humans with more cooperative and less contentious personalities than their archaic ancestors, or than their nearest living relatives, the chimpanzees.

Rushton et al. (2008) further proposed that the GFP arose from directional selection for a slow life history strategy. Building on MacArthur and Wilson's (1967) analysis of *r-K* reproductive strategies, which explains how animals and plants populate islands, Rushton (1985, 1990) proposed that 'one basic dimension — *K* — underlies much of the field of personality' (1985, p. 445). Rushton postulated that personality traits co-evolved with altruism, intelligence, attachment styles, growth, health, longevity, sexuality, and fecundity to form a cocoordinated suite of traits organized to meet the trials of life — survival, growth, and reproduction. Unlike conventional personality psychology, life history theory predicts hierarchically organized traits, culminating in a single, heritable, super-factor. Traits need to be harmonized, not work independently of each other.

Life History Theory describes the strategic allocation of bioenergetic and material resources between major components of fitness such as survival and reproduction. The fast-slow (*r-K*) continuum represents a covarying range of reproductive behavioral strategies inversely relating life history traits such as fecundity and parenting. Life History Theory predicts that species living in harsh (high-risk of mortality), unpredictable, and uncontrollable environments evolve clusters of 'fast' life history traits, whereas species living in predictable, stable, and relatively safe and controllable environmental conditions evolve clusters of 'slow' life history traits (Ellis et al., 2009). Hence, the fast life history strategist is a short-term planner, taking benefits opportunistically with little regard for long-term consequences. In contrast, the slow life history strategist is a long-term planner, delaying immediate gratification in the service of future eventualities.

These considerations suggest that the slow life history strategy predisposes individuals to favor socially and sexually mutualistic or prosocial over antagonistic or antisocial strategies (Figueredo & Jacobs, 2009). Slow life history strategists are more likely to engage in reciprocally altruistic relationships with both kin and non-kin, as well as with both romantic (not just sexual) partners and their offspring. Slow life history strategists prefer long-term and cooperative social as well as sexual relationships, which are easier and more profitable to maintain in their characteristically more stable, predictable, and controllable environments. We should therefore expect life history evolution to favor the evolution of the GFP, because the conditions favoring slow life history strategy are those favoring the cooperative sociality indicative of the GFP.

Research has confirmed many predictions from life history theory (Bogaert & Rushton, 1989; Figueredo et al., 2004, 2007; Templer, 2008). For example, among university students, Bogaert and Rushton (1989) found correlations between self-reported delinquency, sex guilt, mating effort (e.g., number of sex partners), general intelligence, and an aggregate of items assessing family size, maturational speed, longevity, and altruism. Although the average correlation between single indices of *K*-selection was low, aggregate measures were predictive of a general factor on which single items loaded an average of .31. The results held true when three separate measures of family background were statistically controlled.

In a twin study, Figueredo et al. (2004) reported substantial phenotypic and genetic correlations of the GFP with a latent *K*-Factor, measuring life history strategy, as well as with Covitality, another latent factor representing a composite of mental and physical

health. These three lower-order factors formed a substantially heritable 'Super-*K*' dimension. Figueredo et al.'s (2004) study was based on the National Survey of Midlife Development in the US (MIDUS), a representative sample of 50,000 households that included 309 MZ and 333 DZ twin pairs aged 25 to 74 years. Figueredo et al. (2007) replicated these results using a sub-sample of 2,095 non-twin parents who by middle-age had chosen their life niches to marry (or not), to bear and raise offspring (or not), and to create social networks. Both studies controlled for 'social privilege' by regressing out level of education, race, and family income and found it accounted for less than 10% of the variance and did not change the pattern of factor loadings.

In the present study, we reanalyze Figueredo et al.'s (2004) published twin data to examine whether dominance covariance contributes to the substantially heritable Super-*K* Factor, thereby suggesting a common evolutionary history of recent directional selection. The phenotypic and genetic correlations found between the *K*-Factor and the Covitality Factor resulted from a direct test of life history theory, in which decreased morbidity and mortality were predicted to be features of a slow life history (e.g., Ellis et al., 2009). The phenotypic and genetic correlations of the GFP with both the *K*-Factor and Covitality Factors were hypothesized to represent outward behavioral displays of this increased phenotypic and genetic quality (e.g., Weiss et al., 2002).

Method

The Data for Reanalysis

Figueredo et al. (2004) previously analyzed archival data from the National Survey of Midlife Development in the United States (MIDUS; Brim et al., 2000), a nationally representative sample of 50,000 households that included 309 MZ and 333 DZ twin pairs aged 25 to 74 years. They grouped 253 of 2,000 questions into 30 life history scales to construct three lower-order common factors (a lower-order *K*-factor, a Covitality Factor, and a GFP), which showed bivariate phenotypic correlations of .54, .69, and .43, respectively. A substantially heritable 'Super-*K*' factor loaded on the three factors with standardized regression coefficients of .82, .60, and .75, respectively. The twin correlations for *K*, Covitality, GFP, and Super-*K* were: MZ (.60, .52, .48, and .56, respectively) and DZ (27, .26, .18, and .22, respectively), yielding Falconer (1989) heritabilities of .65, .52, .60, and .68, respectively. The tendency for the DZ twin correlations to be less than half those of the MZ twins implied the presence of nonadditive genetic variance.

New Analysis

We used common pathway models (e.g., Johnson & Vernon, 2005, pp. 150–151) to examine how much of the shared phenotypic covariance among the *K*-factor, the Covitality Factor, and the GFP might be attributable to additive genetic covariance, nonadditive genetic covariance, shared environmental covariance, and nonshared environmental covariance. To this end, we constructed and compared ACE, ADE, and AE Common Pathway Models using the EQS 6.1 software for structural equations modeling.

Results

Alternative Common Pathway Models: ACE, ADE, and AE

We first estimated to what degree the phenotypic correlations among these variables can be attributed to the common genetic (A), shared environmental (C), and nonshared environmental (E) factors that impact on all three of them through a higher-order latent variable (Super-*K*). To the extent that the variables correlated less than perfectly, this was attributed to specific components of genetic, shared environmental, and nonshared environmental factors (A, C, and E) that underlie each of them. Table 1 displays a breakdown of these components of variance.

The first column (Super-*K*) of Table 1 shows the loadings of the higher-order common factor on all three trait factors, indicating a high degree of convergence among the three indicators. The remaining columns show the genetic variance (A^2), shared environmental variance (C^2), and non-shared environmental variance (E^2). The first three rows show the specific squared A, C, and E components of the *K*-Factor, the Covitality Factor, and the GFP; the last row shows the common squared A, C, and E components of the common higher-order Super-*K* Factor. It is clear from this first analysis that, as with most other behavioral genetic results of personality, the degree of shared environmental variance is generally negligible. Several of these parameters were constrained at the lower bound (β = 0.000) by the structural modeling software. A small amount of specific shared environmental variance was found for Covitality, but virtually none was found in the higher-order Super- *K* Factor. The broad-sense heritability coefficient (A^2 = .61) of the higher-order factor was comparable to that obtained initially by means of the Falconer method (h^2 = .68), albeit slightly lower. Furthermore, as with the previous findings, these results indicated that most of the genetic variance was shared among the three convergent indicators of the Super-*K* Factor. The fit of the ACE Common Pathway Model was acceptable by all major criteria: $\chi^2(22)$ = 20.859, p = .5295, AIC = –23.141, NFI = 0.988, CFI = 1.000, RMSEA = 0.000, and SRMR = 0.027.

Table 1

Components of Variance for the ACE Model

Variable	Super-K	A^2	C^2	E^2
K-Factor	0.887	0.108	0.000	0.106
Covitality	0.627	0.165	0.083	0.359
Personality	0.793	0.109	0.000	0.261
Super-K		0.610	0.000	0.389

Table 2
Components of Variance for the ADE Model

	Super-*K*	A^2	D^2	E^2
K-Factor	0.888	0.015	0.097	0.099
Covitality	0.626	0.257	0.000	0.350
Personality	0.792	0.089	0.024	0.260
Super-*K*		0.472	0.142	0.386

We then estimated to what degree the phenotypic correlations among these variables can be attributed to the common additive genetic (A), nonadditive genetic (D), and nonshared environmental (E) factors that impact on all three of them through a higher-order latent variable (Super-*K*). As with the ACE model, to the extent that the variables correlate less than perfectly, this is attributed to specific additive genetic, nonadditive genetic, and nonshared environmental factors (A, D, and E) that underlie each of them. Table 2 displays a complete breakdown of these components of variance.

As in Table 1, the first column (Super-*K*) of Table 2 shows the loadings of the higher-order common factor on all three trait factors, indicating a high degree of convergence among the three indicators. The remaining columns show the additive genetic variance (A^2), nonadditive genetic variance (D^2), and nonshared environmental variance (E^2). The first three rows show the specific squared A, D, and E components of the *K*-Factor, the Covitality Factor, and the GFP; the last row shows the common squared A, D, and E components of the common higher-order Super-*K* Factor. Small amounts of specific additive genetic variance were found for all three traits and small amounts of specific nonadditive genetic variance were found for the *K*-Factor and the GFP, whereas virtually no specific nonadditive genetic variance was found for the Covitality Factor. As with the ACE Common Pathway Model, some parameters were constrained at the lower bound ($\beta = 0.000$) by the structural modeling software. However, these results indicated that most of the additive as well as nonadditive genetic variance was shared among the three convergent indicators of the Super-*K* Factor. As expected, however, the narrow-sense heritability coefficient ($A^2 = .47$) of the higher-order factor was substantially lower than the broad-sense heritability coefficient obtained initially by means of the Falconer method ($h^2 = .68$) as well as by the ACE Common Pathway Model ($A^2 = .61$), due to the presence of nonadditive genetic variance ($D^2 = .14$). The fit of the ADE Common Pathway Model was acceptable by all major criteria: $\chi^2(24) = 20.282$, $p = .6806$, AIC = –27.718, NFI = 0.988, CFI = 1.000, RMSEA = 0.000, and SRMR = 0.023.

Finally, we estimated to what degree the phenotypic correlations among these variables can be attributed to the common additive genetic (A) and nonshared environmental (E) factors that impact on all three of them through a higher-order latent variable (Super-*K*), eliminating the nonadditive genetic (D) factor from the model. As with the ADE model, to the extent that the variables correlate less than perfectly, this is attributed to specific additive genetic and nonshared environmental factors (A and E) that underlie each of them. Table 3 displays a complete breakdown of these components of variance.

As in Tables 1 and 2, the first column (Super-*K*) of Table 3 shows the loadings of the higher-order common factor on all three trait factors, indicating a high degree of convergence among the three indicators. The remaining columns show the additive genetic variance (A^2) and nonshared environmental variance (E^2). The first three rows show the specific squared A and E components of the *K*-Factor, the Covitality Factor, and the GFP; the last row shows the common squared A and E components of the common higher-order Super-*K* Factor. Some specific additive genetic variance was found in all three traits. As with the ADE Common Pathway Model, however, these results indicated that most of the additive genetic variance (A^2) was shared among the three convergent indicators of the Super-*K* Factor, yielding the same heritability estimate as the ACE Common Pathway Model ($A^2 = .61$) due to the absence of shared environmental effects in the latter. The fit of the AE Common Pathway Model was acceptable by all major criteria: $\chi^2(29) = 21.985$, $p = .8208$, AIC = –36.015, NFI = 0.987, CFI = 1.000, RMSEA = .000, and SRMR = 0.026.

Summary of Results

Figure 1 shows that the results of fitting the ADE Common Pathway Model to the correlations between the *K*-Factor, the Covitality Factor, and the GFP. The A, D, and E path coefficients are shown in unsquared form. All three alternative models fit just about equally well by all statistical and practical measures of goodness of fit. In terms of degrees of freedom, the most parsimonious model is the AE Model, followed by the ACE Model, followed by the ADE Model. However, the advantage in degrees of freedom of the ACE Model over the ADE Model is purely attributable to more of the ACE Model parameters being constrained at the lower bound ($\beta = 0.000$) by the structural modeling software, due to the virtual absence of shared environmental effects in the *K*-Factor and the GFP. Interestingly enough, the only

Table 3
Components of Variance for the AE Model

	Super-*K*	A^2	E^2
K-Factor	0.888	0.106	0.104
Covitality	0.627	0.257	0.350
Personality	0.792	0.110	0.263
Super-*K*		0.610	0.389

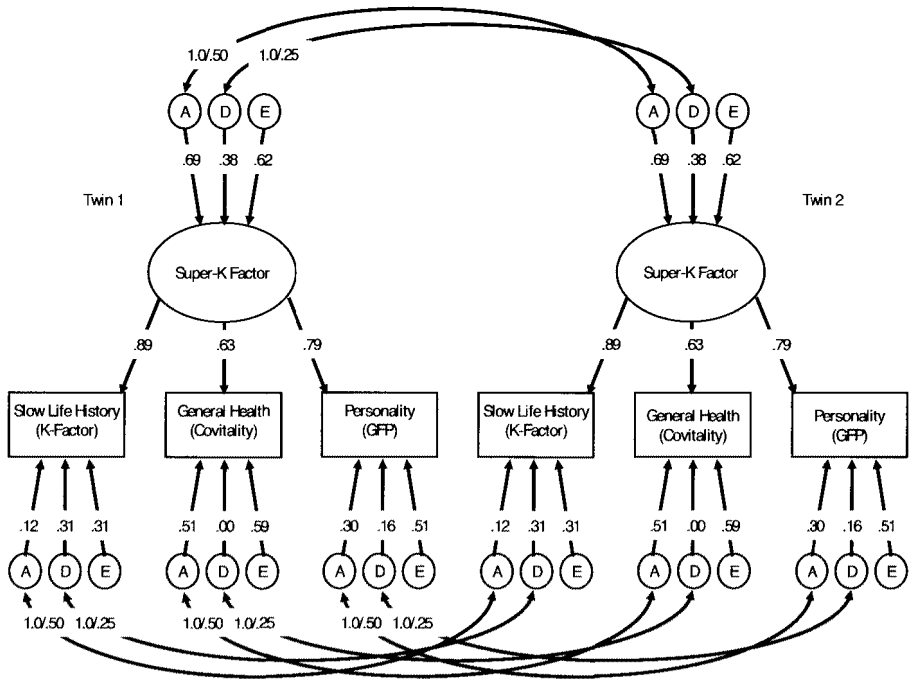

Figure 1

The ADE Common Pathway Model.

trait for which non-zero shared environmental effects were estimated, Covitality, was the one trait for which no specific component of nonadditive genetic variance could be estimated.

We conclude that, considering the small sample size available in the MIDUS twin data, the possibility of nonadditive genetic effects (D) cannot be conclusively rejected. Moreover, assortative mating effects have not been taken into account in these models but we know they exist on comparable measures at a statistically significant level and are crossculturally replicable (r = 0.26, p = .0001; Figueredo & Wolf, 2009). As such, they will increase the DZ twin correlations and increase the confidence in our conclusion about not being able to rule out a nonzero nonadditive genetic component. The main point of our analysis, however, is that to the extent that there is nonadditive genetic variance in these three traits, the preponderance of that nonadditive genetic variance is *shared*, presumably indicating a common history of recent directional selection. The fact that this nonadditive component of genetic variance is shared among three traits makes this finding relatively unlikely to be the result of a Type I Error.

Discussion

The present results show that nonadditive (D) models cannot be ruled out to account for the *shared* contribution of three lower-order heritable factors (a lower-order *K*-Factor, a Covitality health factor, and a GFP) to the Super-*K* Life History Factor identified by Figueredo et al. (2004). This suggests these three traits have been under the same recent directional selective pressures. These genetic correlations support the view that slow (*K*-selected) life history strategy, good physical and mental health, and the GFP coevolved and are mutually coadapted. Moreover, it suggests that a slow life history arose from directional selection.

The evolutionary genetics of personality has been re-gaining theoretical momentum in recent years with increasing effort to compare rival hypotheses using quantitative estimates of relevant parameters. For example, Penke et al. (2007) discussed three models of the origins of individual differences — selective neutrality, mutation selection balance, and balancing selection by environmental heterogeneity. The advantage of identifying a GFP at the apex of the hierarchy of personality is that it clarifies the underlying trait structure, integrates lower-level theories, and helps choose between the competing evolutionary theories. According to a life history perspective, traits will be hierarchically organized, culminating in a single, harmonized super-factor (Figueredo et al., 2004, 2007; Rushton, 1985, 1990; Wilson, 1975). Traits need to be coordinated and work together, not act independently or in opposition to each other.

The well-defined positive and negative pole of the GFP (the former being more cooperative and prosocial) suggests how and why unidirectional selection for personality might have occurred from 'primeval man and his ape-like progenitors', as Darwin (1871, p. 159) phrased it. Those at the high end of the GFP —

altruistic, open, conscientious, sociable, agreeable, emotionally stable, and emotionally intelligent — can be expected to enjoy better social relationships and greater reproductive success since people prefer as mates, fellow workers, and leaders those who are agreeable, cooperative and emotionally stable (Figueredo et al., 2006; Miller, 2007).

More recently, Hrdy (2009) reviewed evidence that 'cooperative breeding' helped foster uniquely human traits over the last 2 million years, including extended lifespans, prolonged childhoods, big brains, perspective-taking, language use, cumulative culture, mutual understanding, norm formation, altruistic punishment, and moral judgment. Hrdy argued that it was cooperation among extended kin over child-rearing that produced the human life history (and, by implication, the GFP). She wrote, 'novel [child] rearing conditions among a line of early hominins meant that youngsters grew up depending on a wider range of caretakers than just their mothers, and this dependence produced selection pressures that favored individuals who were better able at decoding the mental states of others, and figuring out who would better help and who would hurt' (p. 66).

Other evolutionary scenarios are possible. Following Fisher (1954), Nesse (2007) has proposed a theory of *runaway social selection* for displays of partner value and altruism in which competition to be selected as a social partner produces runaway selective processes in the same way that competition to be selected as a sexual partner does in runaway sexual selection. Individuals prefer social partners who display valuable resources and bestow them selectively on associates. This produces selective pressure for individuals to accurately identify: (1) socially valued resources, subject to the economic principles of supply and demand; and (2) preferred social partners, based on their resource and altruistic displays. These selective pressures tend to produce genetic correlations between the resource displays and the partner preferences. Thus, these traits will come to be correlated because of runaway social selection for an ideal person. Moreover, people able to cooperate in groups are also more likely to win competitions and wars (Alexander, 1979; Darwin, 1871; Geary, 2005).

This theory might also help inform the debate in other ways. The main alternative to an evolutionary explanation for the GFP is some form of methodological artifact such as social desirability responding (e.g., Bäckstrom et al., 2009; McCrae et al., 2008). However, such criticisms are *psychometrically*, but not *evolutionarily* informed. For example, if one seriously considers Nesse's (2007) theory of runaway social selection, then the generally acknowledged population preference for a personality profile reflecting the pattern of the GFP (Figueredo et al., 2006; Miller, 2007) will inevitably produce selective pressure that creates a GFP even when none previously existed. Moreover, the preference for the trait and the trait itself would become genetically correlated. Therefore, the existence of an evaluative bias does not, by itself, constitute a sufficient theoretical reason to discount the reality of the GFP, but instead provides grounds to propose its existence as a truly substantive phenomenon. In social evolution, an evaluative bias is not a methodological artifact, but a selective pressure (e.g., Fisher, 1954). Individuals with a socially desirable response bias, indicating an evolved preference for socially desirable behaviors in themselves and others, will therefore actually be more likely to carry genes for exhibiting those socially desirable behaviors.

In spite of our present emphasis on the evolutionary significance of shared nonadditive genetic variance for the coevolution of personality with life history strategy, substantial additive genetic effects have been documented by ourselves and others in the GFP and lower-order traits as well as in life history strategies. As Penke et al. (2007) discuss, additive effects are compatible with balancing selection and with mutation-selection balance. If allied to runaway social selection, this too could imply directional selection consistent with a mutation-selection model including both additive and nonadditive effects. Figueredo, Wolf, et al. (in press) have recently outlined the multiple selective pressures that have hypothesized to bear on the evolution of individual behavioral differences. Many evolved adaptations are the product of, not one, but multiple selective forces. In Newtonian Physics, the 'resultant' is the vector sum of two or more vectors representing multiple forces acting simultaneously on a single material particle. The same principles hold in evolutionary biology.

As in contemporary theories of Multilevel Selection (Wilson & Wilson, 2007), multiple selective pressures may act on any given trait simultaneously. Thus, there is seldom a one-to-one correspondence between a specific trait and a specific selective pressure. For example, both intrasexual and intersexual selective pressures clearly influence female mate choice. Females may prefer males with good genetic quality, but may also prefer males that demonstrate intrasexual dominance. Thus, distinguishing the products of intra-sexual competition from intersexual selection, even in female mate choice, is a challenge because both may be responsible for the evolution of a particular trait. Thus Life History Strategy, the GFP, and Personality in general may be the results of both balancing and directional natural selection, as well as some elements of runaway social and sexual selection, giving rise to both additive and nonadditive components of genetic variance.

The confirmation of a GFP may also shed light on the neurobiology of personality and provide help in the search for personality genes. Investigators who view a GFP as occupying the apex of the hierarchy are likely to adopt different research strategies from those who consider the Big Two, the Big Three, or the Big Five as the highest level. Increasingly, a fewer number

of higher-order constructs are being proposed with wider and more pervasive effects such as the importance of effortful control (MacDonald, 2008) and serotonin (Carver et al., 2009).

We therefore conclude that there is evidence for recent directional selection favoring the emergence of the GFP. However, there are obviously other pressures at work to maintain interindividual variability, including various disruptive social selection pressures that have been identified and reviewed elsewhere (Figueredo et al., 2005; Figueredo, Vásquez, & Sefcek, 2009; Figueredo, Gladden, et al., in press; Figueredo, Jacobs, et al., in press; Figueredo, Wolf, et al., in press; Penke et al., 2007). It seems clear that further research is needed on the nature and origins of the GFP and its relation to Life History Theory.

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