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The association between *g* and *K* in a sample of 4246 Swedish twins: A behavior genetic analysis

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ABSTRACT

Whereas the heritability of general intelligence (*g*) is very well documented, there are relatively few reports of the heritability of life history speed (*K*). Moreover, the correlation between *g* and *K* is of great theoretical significance. Here, we examine the heritabilities of *g* and *K* in a sample of 2123 complete Swedish twin pairs, as well as looking for evidence of common genetic variance between the two. We find a significant albeit very small correlation between relatively strong measures of *g* (the Wiener Matrizen Test) and *K* (the Mini-K; $r = .03$, $p < .05$). Controlling for attenuation by reliabilities and imperfect validity using validity generalization increased the correlation to *rho* = .05 ($p < .05$). There was no significant common additivity between *g* and *K*, however path elimination in behavior genetic structural equations modeling suggests that the small common variance is nonetheless likely to stem from shared additive genetic influences rather than from environmental influences. The implications of this are discussed. Our new estimate of the heritability of the life history in the Swedish population is a particularly significant result, as the heritability of life history speed has never before been established in non-US samples.

1. Introduction

Life history describes the pattern of allocations of bioenergetic resources into different domains of fitness in response to the presence or absence of cues signifying environmental stability or instability. These cues can have their effect on life history characteristics either in phylogenetic time (i.e. as a consequence of specific patterns of allocation having been selected across generations of organisms persistently exposed to particular environments), or in ontogenetic time (i.e. as a consequence of strategic trade-offs made in response to certain environments throughout the course of an organisms development; Ellis, Figueredo, Brumbach, & Schlomer, 2009). Life history is strongly unidimensional in human populations, and is measured using the concept of speed, which pertains to the tendency for *faster* life history individuals to be characterized by shorter life expectancies, more rapid maturation, higher mating effort and higher extrinsic mortality, than those exhibiting *slower* life histories, who are characterized by lower mating effort, but commensurately higher levels of parenting effort and somatic effort (i.e. resources allocated towards growth, longevity and general maintenance). This continuum of life history speed extends to encompass a panoply of conative and behavioral traits in human populations that cluster along a dimension termed *Super-K* (Figueredo, Cabeza de Baca, & Woodley, 2013; Figueredo & Rushton, 2009; Figueredo, Vásquez, Brumbach, & Schneider, 2004). These traits include behavioral dispositions towards family, sex and community (termed the *K*-factor), individual personality dimensions, in addition to the global general factor of personality and self-reported physical and mental health (Covitality).

The coherence amongst all of these psychometric dimensions was predicted by J. Philip Rushton in a 1985 paper. Rushton perceived human behavior and personality to be a manifestation of a more basic life history continuum characterized by prosociality, behavioral restraint, monogamous pair bonding and greater physical and mental health at the slower pole, and anti-sociality, impulsiveness and aggressiveness, promiscuousness and poorer health at the faster pole. Importantly, Rushton (1985, 2000, 2004) also argued that general intelligence or *g* should constitute a component of this life history continuum owing to the fact that *g* is related to brain size, and bigger brains are more expensive in terms of somatic effort, hence should have been favored under conditions promoting slower as opposed to faster life histories.

Behavior genetic studies have repeatedly shown high heritabilities and strong genetic correlations between the three major domains of *Super-K*; i.e. the behavioral *K*-factor, the GFP and

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Covitality, thus providing essential support for the core of Rushton's highly parsimonious theory (Figueredo & Rushton, 2009; Figueredo, Vásquez, Brumbach, & Schneider, 2007; Figueredo et al., 2004). The *K*-factor in particular has been shown to be highly heritable ($h^2 \approx .65$; Figueredo & Rushton, 2009; Figueredo et al., 2004, 2007). However, this estimate pertains exclusively to North America, and it is therefore important to replicate it for other populations. For one thing, ethnically and genetically less diverse populations may exhibit quite different heritabilities due to different levels of gene-environment interaction. The Swedish population born more than 30 years ago, before the great influx of immigrants that began in the eighties, is quite ethnically and genetically homogeneous, and will provide a stark contrast to the US samples.

Returning to the issue of the association between *g* and *K* predicted by Rushton, research on the individual-differences level relationship indicates that they are in fact not substantially correlated. In meta-analysis the two constructs seem to correlate positively, but not significantly (*rho* = .023, *ns*, *N* = 2056; Woodley, 2011). Testing for heterogeneity reveals substantial and significant dispersion amongst studies also, with an effect size range of −.18 (Figueredo, MacDonald, Wenner, & Howrigan, 2007) to .29 (Bogaert & Rushton, 1989). This strongly contradicts the theory that *g* and *K* should substantially correlate at the individual differences level, thus potentially falsifying this aspect of Rushton's (1985) theory.

Rushton primarily supported his hypothesis of a strong *g*/*K* correlation using aggregate level data (i.e. species and human population level indicators of *K* and *g*; Rushton, 2000, 2004, 2010). Consistent with this, amongst the countries of the world (Meisenberg & Woodley, 2013; Templer, 2008; Woodley & Fernandes, 2014) and also at the regional-level within countries (Fernandes & Woodley, 2013; Templer & Rushton, 2011) there do indeed exist strong associations between indicators of *g* and *K*. The presence of this finding coupled with the absence of a significant correlation between the two constructs at the individual differences level has been termed "Rushton's paradox" (Meisenberg & Woodley, 2013). A plausible solution to the paradox is that environments favouring the evolution of high levels of one trait, also tended to favor higher levels of the other, hence the two are extrinsically correlated at the aggregate level due to historical co-selection (Meisenberg & Woodley, 2013).

This may especially have been the case if environmental challenges particular to the domain of *K* (i.e. the need to engage in future oriented behaviors owing to the presence of seasonal fluctuations in resource availability, the need to cope with higher conspecific densities and the need to maintain social networks over large geographic scales) co-occurred in space and time with respect to environmental challenges specific to the domain of *g* (i.e. 'evolutionarily novel problems' requiring abstract reasoning for their solving; Kanazawa, 2004, 2010; Sefcek & Figueredo, 2010; Woodley, 2011). At the individual differences level it has also been proposed that *g* and *K* are associated with largely exclusive sources of genetic variance (Woodley, 2011). The former is likely a 'fitness indicator' or indicator of underlying genetic quality, which has uniformly positive effects on fitness outcomes across environments, and higher levels of the trait are thus typically favored in social and sexual selection. Individual differences in *g* therefore result from mutation–selection balance (Miller, 2000a,b; Penke, Denissen, & Miller, 2007). *K* and components of life history such as personality are likely principally associated with common polymorphisms having variable effects on fitness across environments, such that stabilizing selection will tend to favor diversity with respect to levels of these traits (Figueredo & Gladden, 2007; Penke, Denissen, & Miller, 2007).

The existence of these separate sources of genetic variance explains the absence of a substantial correlation between *g* and *K* at the individual differences level. Simply put, higher *g* is favored across the spectrum of environments associated with variance in *K*, which leads to broad linkage equilibrium between the genes for both traits. Despite this there might be a reason for predicting a small positive correlation between *g* and *K*. Whilst fast and slow life history strategies might be equally favored amongst those with high *g* and commensurately high genetic quality, the presence of higher levels of mutation load amongst those with low *g* may favor faster life histories via the route of condition dependence (Woodley, 2011). Condition dependence would in this case result from the coupling of a poorer quality phenotype with exposure to poorer quality environments generated by others exhibiting poorer quality phenotypes, which might encourage the development of faster life histories at a higher rate (e.g. Nettle, Frankenhuis, & Rickard, 2013). Consistent with this prediction, when validity generalization is performed on the correlation between fluid intelligence and the Mini-K (a 20 item short-form measure of *K*) a small but *statistically significant* positive correlation results (*rho* = .06; Figueredo et al., 2014).

Woodley's (2011) condition dependence model is only one of three possible models that could account for a small, but significant *g*/*K* correlation. Miller's, (2000a,b) fitness indicator theory would predict a fully genetically mediated correlation between the two stemming from the pleiotropic effects of mutation load (i.e. mutations that damage one brain system are likely to damage others during development also, generating genetic correlations between those systems; Miller, 2000a,b). Rushton's previously discussed Differential-K theory would also predict genetic correlations between *g* and *K*. This is because Rushton argued that brain size is a manifestation of high-*K*, evidencing higher somatic effort allocation, which in turn drives higher-*g* (Rushton, 2010).

Thus we have three theories about the genetic architecture of the putative small *g*/*K* correlation, which can be split into two camps – those which argue that the shared variance is primarily environmental (i.e. Woodley, 2011) and those which argue that the shared variance is primarily genetic (i.e. Miller, 2000a,b; Rushton, 1985). These can be tested using behavior genetic designs of sufficient power.

1.1. Overview

The purposes of the present study are to estimate the heritability of *K* in a genetically and ethnically homogenous population and test the environmental versus genetic theoretical causal link behind a possible small *g*/*K* association. To this end we use a large sample of twins, for which direct but relatively reliable single indicator measures of *g* and *K* were administered, in order to specifically (1) estimate the heritabilities of *g* and *K*, (2) establish the presence of the true (i.e. error disattenuated) magnitude of a *g*/*K* correlation, and (3) examine the genetic architecture of the *g*/*K* association (once established).

2. Methods

2.1. Measures

The data on *g* and *K* were collected from a large sample of twins from the Swedish Twins Registry (STR) – the STAGE cohort (Lichtenstein et al., 2006) with approximately 32,000 twins born between 1959 and 1985. Zygosity has been determined by questions about intra-pair similarities and was subsequently confirmed in 27% of the twins in the STR using genotyping. For further details on the STAGE cohort and zygosity determination in the STR see Lichtenstein et al. (2002, 2006). The present study received approval from the Regional Ethics Review Board in Stockholm (Dnr 2011/570-31/5).

Data were collected as part of a web-survey, which included a Swedish language fluid-matrix reasoning test used to measure general intelligence called the Wiener Matrizen Test (WMT, Formann & Piswanger, 1979). *K* was evaluated using a web survey implementation and Swedish language translation of the Mini-K, a 20 item short form of the Arizona Life History Battery comprised of the most *K*-loaded questions (Figueredo et al., 2006). The Mini-K exhibits satisfactory reliability (.73) and validity (.91) and has been used in previous studies into the relationship between life history and intelligence (Figueredo et al., 2014; Woodley, Figueredo, Brown & Ross, 2013).

An invitation was sent via surface mail to ~32,000 twins, 11,543 of whom completed at least one instrument in a web questionnaire via the Internet. After excluding incomplete answers to either the WMT or the Mini-K, the final sample consisted of 7868 participants, 3455 males and 4413 females, aged between 27 and 54 years (mean 40.23, SD 7.84). This sample contained 2123 full twin pairs (1005 MZ and 1118 DZ twin pairs), i.e. 4246 individuals, and 3622 single twins without the co-twin participating. The single twin-individuals were retained for analysis because they contribute to the estimation of means, variances, and covariate effects.

2.2. Validity generalization

It is well known that correlations are attenuated by the reliabilities and validities of the indicators comprising them. Hunter and Schmidt (2004) proposed a systematic method for disattenuating correlations on this basis via the use of certain corrections. The four corrections that we make are for (1) the reliability of the WMT, (2) the reliability of the Mini-K, (3) the validity of the WMT, and (4) the validity of the Mini-K.

Reliability is always controlled by dividing by the square root of the coefficient of reliability. Validity coefficients are usually corrected (via division) after reliability has been corrected (te Nijenhuis & van der Flier, 2013). Data on the reliability of the WMT comes from the manual, which gives the value of .81 estimated on the basis of internal consistency (Formann & Piswanger, 1979). For the Mini-K Figueredo et al. (2014) found a meta-analytic reliability of .73. This suggests a relatively much lower reliability for the *K* indicator. For a fluid-reasoning test such as the WMT, te Nijenhuis and van der Flier (2013) suggest .90 as a conservative validity coefficient. Figueredo et al. (2014) suggest .91 as a meta-analytic estimate of the validity of the Mini-K. Implementing these corrections simply requires that we divide .03 (the raw *g*/*K* correlation) by .90 (square-root of the reliability of *g*), .85 (square-root of the reliability of *K*), .90 (validity of *g*) and .91 (validity of *K*).

2.3. Behavior genetic studies

2.3.1. Twin modeling

The classical twin design utilizes the differences in genetic sharing between monozygotic (identical; MZ) and dizygotic (non-identical; DZ) twins, with the former sharing 100% of their segregating genes while the latter only share 50% on average (like normal siblings), to partition trait variance into that due to additive genes (A), common environment (C) – all influences shared between the twins and making the pair more alike to each other and unique (E) – all influences not shared between the twins and making them more different including measurement error. With the use of structural equation modeling the combination of ACE influences that best explains the population variance in a trait or the covariance between two or more traits can be precisely estimated. Using maximum likelihood (ML) modeling procedures in the flexible matrix algebra program Mx (Neale, Boker, Xie, & Maes, 2006). Parameter estimates for the saturated model can be derived (through optimization) and subsequently specific hypotheses regarding the significance of particular parameters can be tested statistically. This is done by comparing the goodness-of-fit to the observed data (distributed as χ^2) of various models using the minus two times loglikelihood (−2LL) statistic. If the change in χ^2 ($\Delta\chi^2$) is not significant, the more parsimonious model can be regarded the one of choice.

Prior to genetic modeling we tested for age and sex effects on the mean and for normality of the two constructs. A bivariate ACE Cholesky decomposition was fitted to explore genetic and environmental influences on the covariance between the two variables (*g* and *K*). Subsequently different parameters were dropped to determine their significance. Finally, to determine the simplest model explaining the relationship between the two variables, the model was successively reduced starting with dropping the smallest parameter estimates and then comparing the model fit to the previous model.

3. Results

3.1. Controlling for reliability and validity

Controlling for the reliabilities and validities of both *g* and *K* boosted the correlation from .03 to *rho* = .05. Given the very large sample size, this was statistically significant.

3.2. Preliminary results of twins analysis and descriptive statistics

Sex showed a significant effect on the Mini-K while sex as well as age were significant for the WMT. Therefore, both were included as covariates in all subsequent twin-analyses. The Mini-K as well as WMT were approximately normally distributed (skewness fell between 1 and −1 in both cases), with a mean of 12.84 in the case of the WMT and 21.79 in the case of the Mini-K. Standard deviations were 5.22 for the WMT and 13.638 for the Mini-K, with an *N* of 8220. This indicates that our sample has on average a moderately slow LH (where 0 is intermediary on a scale running from −60 at the extreme fast pole to +60 at the extreme slow pole; Figueredo et al., 2006).

3.3. Twin modeling results

Phenotypic correlations and twin correlations for MZ and DZ twins are shown in Table 1. DZ twin correlations were more than half the MZ correlations suggesting an ACE model would fit the data best. Sex-limitation was not indicated as MZ and DZ correlations were similar across sexes.

Genetic modeling results showed heritability estimates of 53% (CI: 40–62%) on WMT and 33% (CI: 21–48%) on the Mini-K. Although common environment did not significantly influence WMT (5%; CI: 0–17), it explained 13% (CI: 1–26%) of the variance in *K*. The significant remainder was due to non-shared environmental influences.

Not surprisingly given the very small but significant positive correlation between the variables, the bivariate Cholesky decomposition showed that there was too little power [?] to partition the covariance into that due to A, C and E – all three cross-paths were nonsignificant (Fig. 1). This was further supported by the Multivariate model fitting results (Table 2, Model 3–5), indicating that the three covariance estimates (A_{cov}, C_{cov}, E_{cov}) could be dropped without significant deterioration of the model fit relative to the full model. Next, successive model reduction (starting with the smallest parameter estimate – E_{cov}) indicated that E_{cov} and subsequently C_{cov} could be removed without significantly worsening the fit (Table 2, Models 5 and 6). A_{cov} could not be removed

Table 1
Phenotypic correlations (top) and twin correlations for each zygosity group (bottom) for WMT and Mini-K corrected for sex and age.

	Phenotypic correlations (95% confidence intervals)	
	WMT	Mini-K
Mini-K	.03 (.01; .05)	–
Zygosity	Twin correlations (95% confidence intervals)	
MZ	.58 (.54; .62)	.46 (.41; .51)
DZ	.32 (.27; .38)	.28 (.24; .36)
MZ female	.58 (.53; .63)	.52 (.46; .57)
MZ male	.59 (.52; .65)	.37 (.27; .46)
DZ female	.35 (.25; .44)	.30 (.19; .40)
DZ male	.38 (.25; .48)	.30 (.15; .43)
DZ opposite-sex	.27 (.18; .35)	.30 (.21; .38)

Note: MZ = Monozygotic; DZ = Dizygotic; WMT = Wiener Matrizen Test.

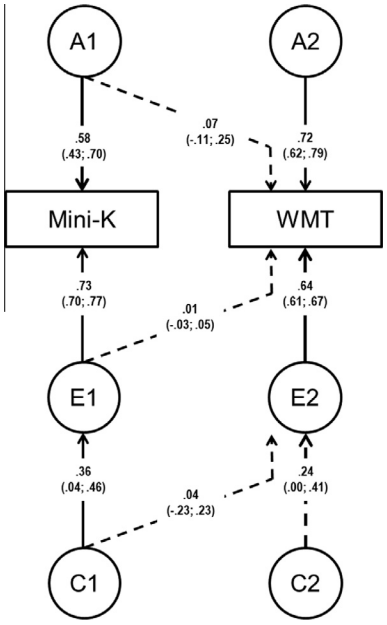

Fig. 1. Full bivariate ACE Cholesky decomposition. *Note*: A = additive genetic; C = common/shared environmental; E = non-shared environmental; WMT = Wiener Matrizen Test.

(Table 2, Model 7) suggesting that what little covariance exists is likely to best be explained by shared additive genetic influences.

4. Discussion

The estimated heritability ranges for the WMT are consistent with heritabilities generated for other similar matrix-based fluid reasoning tasks, such as for the Raven's Progressive Matrices (Jensen, 1998). The heritability of the Mini-K however was lower than predicted based on previous estimates of the heritability of the ALHB (h^2 = .33 vs. ≈.65; Figueredo & Rushton, 2009; Figueredo et al., 2004, 2007). This is possibly a consequence of nomological range restriction amongst the items comprising the Mini-K (i.e. it is not a sufficiently broad measure of the *K*-factor). Nonetheless this finding is the first demonstration of the heritability of life history strategy outside of the US.

The finding that controlling for reliability and validity using validity generalization does not substantially alter the correlation between *g* and *K* is important, as it is consistent with the results of meta-analytic studies in which multiple correlations are used in establishing the population correlation. For example, as has been mentioned, Woodley (2011) found a *rho* of .023 across all individual differences-level *g*/*K* correlational studies either published or presented at conferences up until that point in time. Figueredo et al. (2014) found that with respect to a full psychometric meta-analysis of the Mini-K/Fluid *g* correlation, the largest aggregate effect-size that can be expected is statistically significant at .06 (N = 1111). The current result is consistent with these previous meta-analytic findings (the value fall between the two that have already been established), and reinforces the claim made in Woodley (2011) that *g* and *K* should not correlate substantially.

Seemingly consistent with predictions in Woodley (2011), there is no significant genetic additivity between *g* and *K* when attempts are made to measure this directly. However there is no substantive environmentality either, contrary to predictions based on the condition-dependence model. Furthermore, reducing the model via the elimination of paths suggests that additivity is in fact the most likely source of the covariance. This is contrary to the predictions of Woodley (2011) and may imply contributions stemming from either mutations or polymorphisms, consistent with either the models of Miller (2000a,b) or Rushton (1985). In either case, the correlation is very small – far smaller than that expected on the basis of Rushton's (1985, 2000) model, which is based on the assumption that *g* is central to life history strategy at both the within and between-population scales. This suggests that the common additivity is more likely to represent the effects of pleiotropic mutations occurring in genes for *g* and *K* that by chance are in physical linkage (i.e. are located close to one another on the same chromosome). The probable rarity of such gene combinations makes them a small mutational target, which is consistent with both the small effect size of the *g*/*K* correlation, even after the association between the constructs is corrected for multiple sources of measurement error, and also the fact that common additivity is likely to be at the root of this shared effect. This finding furthermore intriguingly suggests that the sort of poor environments known to be associated with low-*g* are not causing disproportionate canalization of faster life history speeds, again contrary to Woodley (2011). Perhaps therefore environmental signals associated with poor aggregate-level abstract reasoning capability (such as the presence of low environmental and cultural complexity) do not trigger life history acceleration, which instead may be more dependent upon specific indicators of environmental instability, such as increased extrinsic mortality and morbidity (e.g. Ellis et al., 2009). High-*K* may also be associated with an enhanced

Table 2
Mulitvariate model fitting results for Mini-K and WMT corrected for age and sex with the best-fitting model in bold.

	AIC	−2LL	df	Δ −2LL	Δ −df	*p*-value
1. ACE – Cholesky decomposition	11515.67	43807.67	16146			
2. Drop A_{cov} – compare to Model 1	11514.26	43808.26	16147	.58	1	.45
3. Drop C_{cov} – compare to Model 1	11513.80	43807.80	16147	.13	1	.72
4. Drop E_{cov} – compare to Model 1	11513.95	43807.95	16147	.28	1	.60
5. Drop C_{cov} – compare to Model 4	**11512.30**	**43808.30**	**16148**	**.34**	**1**	**.56**
6. Drop A_{cov}–compare to Model 5	11517.53	43815.53	16149	7.24	1	<.01

Note: A = additive genetic; C = common/shared environmental; Cov = covariance; E = non-shared environmental.

capacity to inceptively niche construct (i.e. counteract) the sorts of environments that are resilient to such signals, and this capacity being independent of the capacity to reason abstractly leads to modest-*g* but high-*K* populations being able to protect themselves from such environments, even when the complexity of such environments is low. This interpretation is also consistent with models of the evolution of general intelligence which posit that the trait is associated with some degree of ecological domain specificity (Kanazawa, 2004, 2010), as it is clear that *g* and *K*, whilst clearly encompassing broad domains, nonetheless encompass evolutionarily distinct domains (Woodley, 2011).

In conclusion, the present findings essentially represent the absolute limit of what can be done with the individual-differences level *g*/*K* correlation. The robustness of the extremely low-magnitude correlation has now been established in two meta-analytic studies – Woodley (2011), Figueredo et al. (2014) in addition to the present one. This should remove any lingering doubt about the lack of affinity that these variables have for one another. What correlation exists is however in the theoretically expected positive direction and is also statistically significant in the current study given the large value of *N*. The implication of our behavior genetic models is that the correlation is likely to be due to additivity, rather than environmentality of one sort or another. This suggests a simple explanation for the correlation (i.e. pleiotropic mutations affecting rare genes for *g* and *K* that have a low probability of being in physical linkage). Perhaps more importantly is the idea that low environmental complexity, which is a product of low *g*, does not entail lower *K* via the condition dependence route, and that *K* might be associated with the capacity for inceptive niche construction, which may buffer low-*g* but high-*K* subpopulations from environmental perturbations of a sort that are likely to trigger life history speed acceleration (e.g. Nettle et al., 2013).

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