Journal of Methods and Measurement in the Social Sciences,
Vol.5, No.2, 76-106, 2014

No matter Where You Go, There You Are: The Genetic Foundations of Temporal Stability

Aurelio José Figueredo **Tomás Cabeza de Baca**

Candace Jasmine Black
University of Arizona

We present empirical tests of the stability of individual differences over the lifespan using a novel methodological technique to combine behavior-genetic data from twin dyads with longitudinal measures of life history-related traits (including health and personality) from non-twin samples. Using data from The Midlife in the United States (MIDUS) Longitudinal Survey, we constructed a series of "hybrid" models that permitted the estimation of both temporal stability parameters and behavior-genetic variance components to determine the contributions of genetic and environmental influences on individual differences. Our results indicate that changes in a higher-order factor of life history strategy (Super-K, composed of the K-Factor, Covitality, and Personality) over the study period were very small in magnitude and that this temporal stability is under a considerable degree of shared genetic influence and a substantial degree of non-shared environmental influence, but a statistically non-significant degree of shared environmental influence. Implications and future directions are discussed.

Keywords: life history theory, evolutionary psychology, temporal stability, MIDUS, behavioral genetics, personality, individual differences

The stability of individual difference traits (such as personality) has generated fascination and perplexed academics and laypersons, with people collectively asking, "Do people change over time?" or "Do people stay the same over time?" When addressing the topic of temporal stability, Psychologist William James declared that "[i]t is well for the world that in most of us, by the age of thirty, the character has set like plaster, and will never soften again" (1918, p. 121), positing that one's personality and behavioral dispositions canalize into stable, predictable traits. Recording artists Depeche Mode have similarly expressed ideas parallel to James in the song entitled "See You" (Gore, 1982):

> Well, I know five years is a long time
> And that times change
> But I think that you will find
> People are basically the same

The present study seeks to answer whether and to what extent a higher-order *life history strategy* factor (denoted the K-Factor), which encompasses individual differences in the various dimensions of personality, in mental and physical well-being, and in differential

allocations of psychological and bioenergetic resources into various different domains of social relationship (Ellis, Figueredo, Brumbach, & Schlomer, 2009; Figueredo et al., 2006), is a temporally stable trait in adults. If so, the present study also seeks to answer the question of which specific causal factors maintain that temporal stability. We use The Midlife in the United States (MIDUS) Longitudinal survey to produce a unique "hybrid" model by importing previously-estimated biometric heritability parameters ("stout numbers") from the MIDUS twin data into an otherwise empirically-estimated longitudinal model for the MIDUS singleton (non-twin) data. By doing so, we were able to decompose the empirically-derived temporal stability coefficients into biometrically-estimated behavioral-genetic variance components: shared additive genetic variance, shared non-additive genetic variance, shared environmental variance, and non-shared environmental variance. Thus, we were able to compare the relative magnitudes of the pathways that depended on each of these variance components, and thereby assess their relative contributions to the overall temporal stability of these traits.

The Temporal Stability of Individual Difference Traits

The question of temporal stability of individual difference traits has been addressed across many disciplines in psychology. Within comparative psychology, temperament and traits have been observed and examined across multiple taxa, including domestic dogs (*Canis lupus familiaris*; Fratkin, Sinn, Patall, & Gosling, 2013), Rhesus macaques (*Macaca mulatta;* Stevenson-Hinde & Zunz, 1978; 1980), and Stumptail macaques (*Macaca arctoides*; Figueredo, Cox, & Rhine, 1995). For the dogs, Fratkin and colleagues (2013) performed a meta-analysis on personality stability, and those results revealed a moderate overall trait stability effect size in canines (r = .43). The temporal consistency of those traits was higher for adult dogs (r = .51) than for puppies (r = .31); within individual subjects, the correlations for these traits between the puppies and the same dogs as adults were relatively high (r = .40). Among the rhesus monkeys, trait stability was sampled and assessed three times over a four-year interval (Stevenson-Hinde & Zunz, 1978) by means of three separate exploratory principal component analyses, producing highly convergent results and moderate temporal correlations among the consecutive time points for the traits (*Spearman's* ρ = .69 - .92, depending on the trait factor). For Stumptail macaques, trait stability was sampled and assessed six times over an eight-year interval by means of a single Generalizability Theory (GT) analysis of the traits previously identified for Rhesus macaques by Stevenson-Hinde & Zunz, producing high GT coefficients (E^2_{rel} = .682 - .772, depending on the initial assumptions made in two alternative GT models; Figueredo, Cox, & Rhine, 1995). The

findings broadly reveal that animal temperament and traits remain constant over time (see Gosling, 2001 for a review).

When investigated developmentally among humans, the examination of trait stability becomes more complex. For example, according to Hopwood et al. (2011), personality stability can be conceptualized as either *absolute stability* or *differential stability* (see also, Caspi, Roberts, & Shiner, 2005). When examining traits using an absolute stability approach, the researcher focuses on mean-level differences among measurements of the same individuals taken at different times (i.e., intra-individual variation over time in any given trait) and comparing whether age propels absolute trait levels within individuals to increase or decrease over time. For example, individuals generally become more emotionally stable over time, a phenomenon also known as the "maturity principle" (Caspi et al., 2005). The differential stability approach focuses on the consistency of the individual differences themselves on any given trait across different points in time. In this case, the rank-ordering among individuals on that trait is conserved, although absolute levels might change. This implies that if one individual is more emotionally stable than another individual, and both of them increase in emotional stability as they mature, the individual that was higher at the beginning will remain higher than the other individual at the end of the given time interval. For example, when examining Big Five personality stability in a Scandinavian sample of middle-aged adults (Time 1 measures were taken at 33 years old; Time 2 measures were taken at 42 years old), Rantanen and colleagues (2007) reported both significant differences in the means of the all of the Big Five components across that time interval *and* moderate to high test-retest correlations for the given traits across both time points (correlations for men: r = .64 to .81; correlations for women: r = .55 to .81).

Nevertheless, developmental research has consistently found that rank-ordering of individual differences in the levels of personality traits stabilize over time as individuals progress across different life stages, meaning that the older the participants are, the higher are their temporal stability coefficients on any given trait (e.g., Costa & McCrae, 1997; Hopwood et al., 2011; Roberts et al., 2001). At earlier developmental stages, the serial autocorrelations between measures of any given trait at any two consecutive time points are smaller than they are at later developmental ages, suggesting that older individuals become more behaviorally consistent in later adulthood. For instance, for New Zealand adolescents compared at 18 and at 26, the temporal stability coefficients of traits during these 8 years averaged .55 and ranged from .43 to .67 (Roberts, Caspi, & Moffit, 2001), which is consistent with other samples of adolescents (e.g., Morizot & Le Blanc, 2003, Table 6), but generally lower

in magnitude than those found for older adults (for example the temporal stabilities reported in Rantanen et al., 2007).

The present paper examines both absolute and differential stability over time, examining the absolute and relative levels of life history trait values in a single cohort of individuals over a ten-year interval (for a description of the latent covariance structure of these life history traits, see Figueredo et al., 2006).

Mechanisms of Temporal Stability

Whereas the studies described above focus on *whether* and *to what extent* individual difference traits are stable over time, the separate goal of understanding *why* individual differences such as personality show stability over the lifespan has motivated some researchers to examine the role of genes in producing these patterns of behavior. Much of this work comes from behavioral-genetic studies, the fundamental aims of which are to account for both genetic and environmental factors producing individual differences. This is accomplished using one of two predominant research approaches: (1) adoption studies, wherein genetically related individuals are adopted apart or genetically unrelated individuals are adopted together, and (2) twin studies, wherein same-sex monozygotic twins are compared to same-sex dizygotic twins. In each research design, variance is usually partitioned into genetic variance, shared environmental variance, and non-shared environmental variance; however, a number of studies indicate that the contribution of the non-shared environment consumes the lion's share of environmental variance (Bouchard & McGue, 1990; see also Plomin & Daniels, 2011 for a review).

Identifying the contribution of genes in individual differences is done by estimating heritability coefficients that indicate the proportion of variance in a trait accounted for by genetic variance. Heritability may fall under one of two categorizations: broad-sense and narrow-sense heritability. Broad-sense heritability captures all genetic contributions, including both additive and non-additive variance, where non-additive variance is attributable to dominance or epistatic gene-gene interactions. Narrow-sense heritability, on the other hand, includes only additive variance, or the average effects of individual alleles from each parent. Studies focusing on individual differences have consistently shown moderately large heritability estimates; for example, one study by Jang, Livesley, and Vernon (1996) produced (broad genetic) estimates between 40-60% for each of the five factors in the Big 5 (Openness, Conscientiousness, Extraversion, Agreeableness, and Neuroticism). Other stable traits also appear to have a genetic component, such as altruism, empathy, nurturance, aggressiveness, and assertiveness (Rushton, Fulker, Neale, Nias, & Eysenck, 1986).

Life History Traits

The application of life history theory in examining developmental questions has become a burgeoning area of research among evolutionary developmental psychologists (e.g., Belsky, Schlomer, & Ellis, 2012; Ellis et al., 2012; Simpson et al., 2012). One reason for the interest in applying life history theory to explain development findings is that it constitutes an overarching principle that can be used to organize and make sense out of a broad array of individual difference traits.

Life history theory posits that humans are faced with social and ecological challenges they must overcome to successfully survive and reproduce. The manner in which humans must solve these problems relies on the allocation of material and bioenergetic resources toward different facets of development (e.g., reproductive effort and somatic effort; Ellis, Figueredo, Brumbach, & Schlomer, 2009). Within this framework, individuals who invest more on growth and maintenance of the body, as opposed to reproductive effort, will pursue *slow life history strategies*, which are characterized by mutualistic and prosocial behavioral traits. Conversely, *fast life history strategists* will invest more in reproductive effort and less in somatic effort. This means, behaviorally, fast life history strategists may be oriented toward (or more permissive of) opportunistic or antagonistic social strategies (Belsky, Steinberg, & Draper, 1991; Figueredo & Jacobs, 2010).

While measurement of life history traits can vary, a common form of measurement utilized in life history research is the *psychometric approach,* which captures the allocation of material and bioenergetic resources via behavioral traits, health, and personality (Figueredo et al., 2006; Figueredo, Cabeza De Baca, & Woodley, 2013). Using the psychometric approach toward measurement of human life history strategies, these researchers have identified and validated three lower-order life history factors: (1) the *K-Factor*, encompassing cognitive behavioral indicators of slow life history strategy; (2) the *Covitality Factor*, encompassing medical and psychiatric indicators of physical and mental health; and (3) the *General Factor of Personality*, or *GFP*, encompassing the Five-Factor Model of personality as partially convergent indicators. These researchers have also demonstrated the existence of a higher-order factor for human life history, called the *Super-K* Factor, which encompasses these three components as partially convergent lower-order factors. By creating a higher-order common factor such as *Super-K* with multiple lower-order sub-domains, proponents of the psychometric approach (e.g., Figueredo, Cabeza De Baca, & Woodley, 2013) argue that such a measure produces stable individual difference traits that are both domain-general and possess cross-situational stability across many

ecological contexts *and* cross-temporal stability across different time points.

Accordingly, the investigation of temporal stability of life history traits has important theoretical implications. Because life history theorists posit that life history traits emerge in a cohesive, coordinated pattern to overcome contextual challenges, the measurement of life history traits should reflect that hypothesis. Further, the present study tests whether life history characteristics possess cross-temporal stability, an assumption present in the work of life history researchers utilizing a psychometric approach.

Hypotheses Tested

Putting all of this together, it makes logical sense to apply a behavioral-genetic design to decompose the temporal stability coefficients we observe into their various components of genetic and environmental variance. The problem with this idea is that there are very few data that are both longitudinal and genetically informative in their sampling design. Most longitudinal samples do not include individuals of different degrees of genetic relatedness so that they might be systematically compared; most genetically-informative samples are cross-sectional in design and do not include a sufficiently developed longitudinal component.

Data from *The Survey of Midlife Development in the United States* (MIDUS; Brim et al., 2000) used in the present study, are no exception to this principle. The sample of individuals that were included in the MIDUS longitudinal survey was quite large. However, the genetically-informative sample of monozygotic and dizygotic twins that was included in that survey was relatively small in comparison, and not completely adequate for the testing of complex longitudinal hypotheses.

In this paper, we propose, implement, and report the results of a solution to this widespread methodological problem by creating what we call “hybrid” models that apply the cross-sectionally derived behavioral-genetic parameters of the genetically-informative twin samples to path-analytically decompose the temporal stability parameters of the more longitudinally-informative non-twin samples (due to their greater numerosity). These hybrid methods will be used to determine and compare the relative magnitudes of the longitudinal pathways representing the temporal stability of life history traits that are attributable to shared genetic influence, whether additive or non-additive, and those that are attributable to environmental influences, whether shared or non-shared. This procedure will permit us to test our main hypothesis that the temporal stabilities of life history traits (at least) are largely, although only partially, attributable to the continuing influence of the same genes that are carried by each individual throughout its lifespan.

This hypothesis is based on the presumption that although the expression of these genes may be epigenetically modified by evolved environmental triggers, the nucleotide sequence fundamentally defining them and the biochemical structure of their gene products will generally remain constant over developmental time.

Method

Participants

We used published data from the *Survey of Midlife Development in the United States* (MIDUS; Brim et al., 2000), which consisted of a telephone interview and two follow-up mail surveys given to a nationally representative sample, collected in 2 longitudinal data collection "Waves", the first wave over a one year period from 1995-1996 (N=7108), and the second wave over a two year period from 2004-2006 (N=4963). The Wave 1 sample was limited to English speakers in the United States who completed the MIDUS survey between the ages of 25-74 (at Wave 1) and again (at Wave 2) when they were 35-86. The MIDUS sample included data on singletons (non-twins) as well as a genetically informative sample of MZ and DZ twins:

- *Sample 1* was a subsample of 316 dyads of monozygotic twins (MZ) twins and 274 dyads of same-sex dizygotic (DZ) twins (ages 25-74) who completed Wave 1 of the MIDUS survey (1995-1996), and on which previous LH analyses had been performed (Figueredo et al., 2004; 2007).
- *Sample 2* was a subsample of 215 adult dyads (ages 25-74) of monozygotic twins (MZ) twins and 188 adult dyads of same-sex dizygotic (DZ) twins who completed both Wave 1 and sourced from Wave 2 of the MIDUS survey.
- *Sample 3* was a subsample of 2257 singletons (non-twins, who completed both Wave 1 and Wave 2 of the MIDUS survey. The singletons were also between the ages of 25-74 at Wave 1.

Measures

The life history construct was composed of aggregates of items selected from the MIDUS Survey assessing several facets of a life history strategy. Each facet, or factor, was constructed using items from subscales measuring cognitive and behavioral dimensions of life history strategy. The following is a description of the factors and their corresponding subscales with the number of items extracted from each subscale in parentheses. Specific theoretical justification for the construction of each of these lower-order and higher-order common factors using MIDUS data

was published in Figueredo, Vásquez, Brumbach, and Schneider (2004; 2007). The current hierarchical system for data aggregation, according to domain-specific resource allocations, was detailed in Figueredo, Woodley, Brown, and Ross (2013) and had also been applied in previous biometric behavioral-genetic models by Figueredo and Rushton (2009). However, only the following subset of items and scales that were sampled in both waves of data collection were used in the analyses reported in the present paper:

The K-Factor

- The Self Scale was composed of MIDUS Subscales assessing Insight (4), Persistence (5), Positive Reappraisals (4), Self-Directedness (3), Agency (5), and Financial Status (6), which had been previously analyzed as separate scales in Figueredo, Vásquez, Brumbach, and Schneider (2004; 2007) but were subsequently aggregated in Figueredo, Woodley, Brown, and Ross (2013);
- The Romantic Partner Attachment Scale was constructed from the MIDUS Marital Relationship Quality Subscale (31);
- The Family Support Scale was constructed from the MIDUS Family Support Subscale (6);
- The Friends Support Scale was constructed from the MIDUS Friends Support Subscale (6);
- The General Social Altruism Scale was composed of MIDUS Subscales assessing Close Relationships (2), Children Relationship Quality (5), and Communitarian Beliefs (13), which had been previously analyzed as separate scales in Figueredo, Vásquez, Brumbach, and Schneider (2004; 2007) but were subsequently aggregated in Figueredo, Woodley, Brown, and Ross (2013);
- The Religiosity Scale was constructed from the MIDUS Religiosity Subscale (29)

The Covitality Factor

- The MIDUS Negative Affect (6) Scale;
- The MIDUS Positive Affect (6) Scale;
- The MIDUS General Health (5) Scale;
- The MIDUS General Symptoms (29) Scale;
- The MIDUS Subjective Well-Being (3) Scale

The General Personality Factor (GFP)

- The MIDUS “Big Five” Openness (6) Scale;
- The MIDUS “Big Five” Conscientiousness (4) Scale;
- The MIDUS “Big Five” Agreeableness (5) Scale;

- The MIDUS "Big Five" Extraversion (5) Scale;
- The MIDUS "Big Five" Neuroticism (4) Scale

Missing Data Imputation

Missing data is an important research issue to adequately address when utilizing longitudinal data. If large amounts of missing data are present in a sample, parameter estimates and *p*-values could be biased, potentially increasing both Type I or Type II error rates (Schlomer, Bauman, & Card, 2010). For the present analyses, missing data within the twin and singleton datasets for Wave 1 and Wave 2 was handled with *multivariate imputation* (Figueredo, McKnight, McKnight, & Sidani, 2000; Gorsuch, 1983) and *multiple imputation*, using Proc MI in SAS. Thus, there were 6 datasets imputed: (1) monozygotic twins MIDUS Wave 1, (2) dizygotic twins MIDUS Wave 1, (3) monozygotic twins MIDUS Wave 2, (4) dizygotic twins MIDUS Wave 2, (5) singletons MIDUS Wave 1, and (6) singletons MIDUS Wave 2.

Prior to imputing any data for the twins, twin dyads were selected only if data for both twins were present in both waves of the MIDUS data collection. Following the missing data imputation, the data parameters were aggregated and the imputed correlation matrices were then imported into EQS to run three separate standard behavioral genetic structural equation models (ACE, ADE, AE) for both waves of the MIDUS dataset. A schematic path diagram for the ACE model is shown in Figure 2.1, in which the phenotypic variance in each assessment wave is partitioned among three variance components: (A) shared genetic variance; (C) shared environmental variance; and (E) non-shared environmental variance. A schematic path diagram for the ADE model is shown in Figure 2.2, in which the phenotypic variance in each assessment wave is partitioned among three variance components: (A) shared additive genetic variance; (D) shared non-additive genetic variance; and (E) non-shared environmental variance. The AE model is simply the restricted version of either of these two inclusive models, with either the C or D component, respectively, omitted.

The same selection approach was utilized in the singleton (non-twin) data. Individuals were first screened to confirm they were present in waves 1 and 2 of the MIDUS dataset. Following screening, missing data imputation was then performed prior to structural modeling and the imputed correlation matrices were then imported into PROC CALIS for structural analysis.

Multivariate imputation (MVI) involved estimating unit-weighted factor scores for the component scales and the composite lower-order factors using: (1) the means of the standardized scores for all the items that were not missing within each scale and (2) the means obtained from

the standardized scores for all the indicator scales that were not missing within each factor (Figueredo, McKnight, McKnight, & Sidani, 2000). Most of the scale and lower-order factor scores were recovered this way. For those missing data that remained on the scale and factor scores, we used the EM algorithm, as implemented by SAS PROC MI. Each of the 6 datasets had 30 multiple imputations produced by using this procedure.

Data Aggregation and Analytic Procedures

Estimation of Heritability Parameters. Heritability coefficients were estimated by Common Pathway Biometric SEMs to estimate the contributions of additive (A) and non-additive genetic (D), and shared (C) and non-shared environment (E) on the higher-order *Super-K* factor (see Figueredo & Rushton, 2009). Twin data from both waves of the MIDUS dataset were used. The biometric pathways were estimated using EQS 6.1 statistical software. Thus, a total of 6 biometric models were estimated and tested, with asterisks indicating conventional levels of statistical significance ($p<.05$):

(1) Wave 1 ACE ($\chi^2_{22} = 23.138^{ns}$; CFI = .999; NFI = .983; RMSEA =.013);
(2) Wave 1 ADE ($\chi^2_{23} = 22.345^{ns}$; CFI = 1.00; NFI = .983; RMSEA =.000);
(3) Wave 1 AE ($\chi^2_{29} = 87.425^{*}$; CFI = .956; NFI = .921; RMSEA =.041);
(4) Wave 2 ACE ($\chi^2_{24} = 37.454^{*}$; CFI = .983; NFI = .955; RMSEA =.053);
(5) Wave 2 ADE ($\chi^2_{22} = 44.244^{*}$; CFI = .972; NFI = .947; RMSEA =.071);
(6) Wave 2 AE ($\chi^2_{29} = 44.248^{*}$; CFI = .981; NFI = .947; RMSEA =.051).

Tables 1.1 to 3.2 display the decompositions of variance for the alternative biometric models. The parameters estimates and factor loadings are presented as geometric means across twins but are annotated as squared parameters as is often the common behavior genetic

Table 1.1
Decomposition of Variance for ACE Model Wave 1

Variable	Super-K^2	A^2	C^2	E^2
K-Factor	.672	.116	.000	.212
Covitality	.491	.135	.059	.314
Personality	.537	.165	.000	.297
Super-K		.594	.000	.406

nomenclature. Tables 1.1 and 1.2 display the ACE parameters from Wave 1 and 2, respectively; Tables 2.1 and 2.2 display the ADE parameters from Wave 1 and 2, respectively; Tables 3.1 and 3.2 display the AE from Wave 1 and 2, respectively.

Table 1.2
Decomposition of Variance for ACE Model Wave 2

Variable	Super-K^2	A^2	C^2	E^2
K-Factor	.593	.168	.000	.237
Covitality	.429	.139	.044	.318
Personality	.575	.126	.013	.285
Super-K		.083	.438	.479

Table 2.1
Decomposition of Variance for ADE Model Wave 1

Variable	Super-K^2	A^2	D^2	E^2
K-Factor	.674	.000	.125	.202
Covitality	.491	.201	.000	.309
Personality	.535	.154	.013	.296
Super-K		.516	.080	.403

Table 2.2
Decomposition of Variance for ADE Model Wave 2

Variable	Super-K^2	A^2	D^2	E^2
K-Factor	.586	.162	.010	.240
Covitality	.428	.191	.000	.376
Personality	.576	.141	.000	.283
Super-K		.560	.000	.441

Table 3.1
Decomposition of Variance for AE Model Wave 1

Variable	Super-K^2	A^2	E^2
K-Factor	.674	.116	.211
Covitality	.691	.201	.309
Personality	.535	.166	.299
Super-K		.594	.406

Table 3.2
Decomposition of Variance for AE Model Wave 2

Variable	Super-K^2	A^2	E^2
K-Factor	0.674	0.116	0.211
Covitality	0.691	0.201	0.309
Personality	0.535	0.166	0.299
Super-K		0.594	0.406

Empirical Hybrid Longitudinal Model of Singletons (Non-Twins)

We developed Hybrid Longitudinal Models of the *MIDUS* singleton (non-twin) data by setting fixed model parameters based on behavioral-genetic (biometric) estimates from *MIDUS* twin data using SAS 9.3 statistical software. This was theoretically justified given that *MIDUS* twin and singleton (non-twin) subsamples are two nationally-representative samples drawn from the *same* USA adult population, meaning that they should reflect the *same* general population parameters (including their biometric heritability coefficients) according to statistical sampling theory. Although it has often been repeated that heritability coefficients may be "sample-specific", what is technically being implied is that they are "population-specific" (given that different samples are often drawn from different populations or subpopulations, such as relative poverty levels, social classes, nationalities, sexes, and birth cohorts; see Branigan, McCallum, & Freese, 2013), as differences between samples drawn representatively from the *same* population reflect *random* errors of sampling and not *systematic* effects.

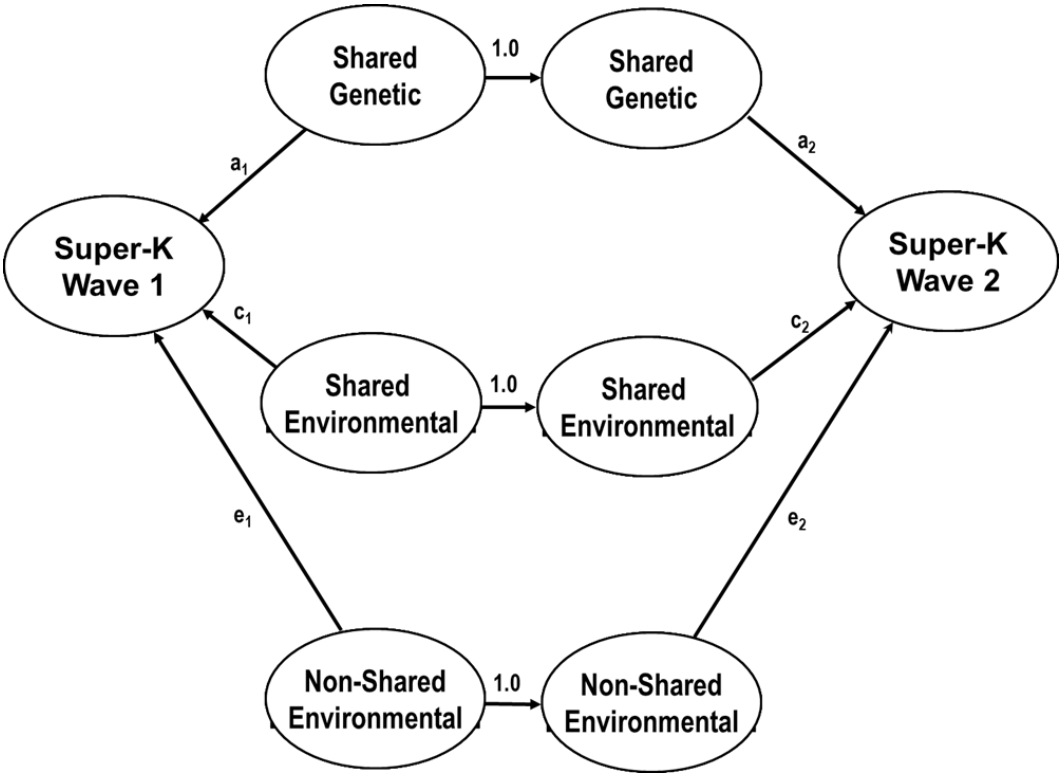
We then freely estimated the remaining longitudinal model parameters based on phenotypic estimates from *MIDUS* singleton (non-twin) data. This "hybrid" procedure was used because the much larger usable sample size (N=2257) of *MIDUS* singleton (non-twin) permit the reliable estimation of longitudinal model parameters not afforded by more limited *MIDUS* twin data (for Wave 1, N_{MZ}= 316 twin dyads and N_{DZ}=274 twin dyads; for Wave 2, N_{MZ}=215 twin dyads and N_{DZ}=188 twin dyads). Longitudinal analyses have been conducted previously using genetically informative data (e.g., Bratko & Butkovic, 2007; Johnson, McGue, & Kreuger, 2005; Kandler, Bleidorn, Riemann, Angleitner, & Spinath, 2012; Kandler, Riemann, & Angleitner, 2013), but never (to our knowledge) using "common pathway" biometric latent factor models containing a hierarchy of nested higher-order and lower-order multivariate constructs, which necessarily increase the need for sufficiently large sample sizes to estimate and test all the requisite model parameters.

Figures 1 and 2.1-2.3 schematically illustrate the fundamental logic used. Figure 1 starts with a single parameter of phenotypic temporal stability of the Super-K Factor (see Figueredo et al., 2004; 2007; Figueredo & Rushton, 2009) estimated from the non-twin longitudinal sample across the 10 years separating the MIDUS Wave 1 (~1995) from the MIDUS Wave 2 (~2005) of longitudinal data collection.

Figure 1.1. Observed Phenotypic Temporal Stability of Super-K Factor.

Figure 2.1 conceptually displays an ACE decomposition of that temporal stability coefficient by using the corresponding behavioral-genetic parameters obtained from the ACE Common Pathway Biometric SEMs that were run separately for the Wave 1 and Wave 2 data.

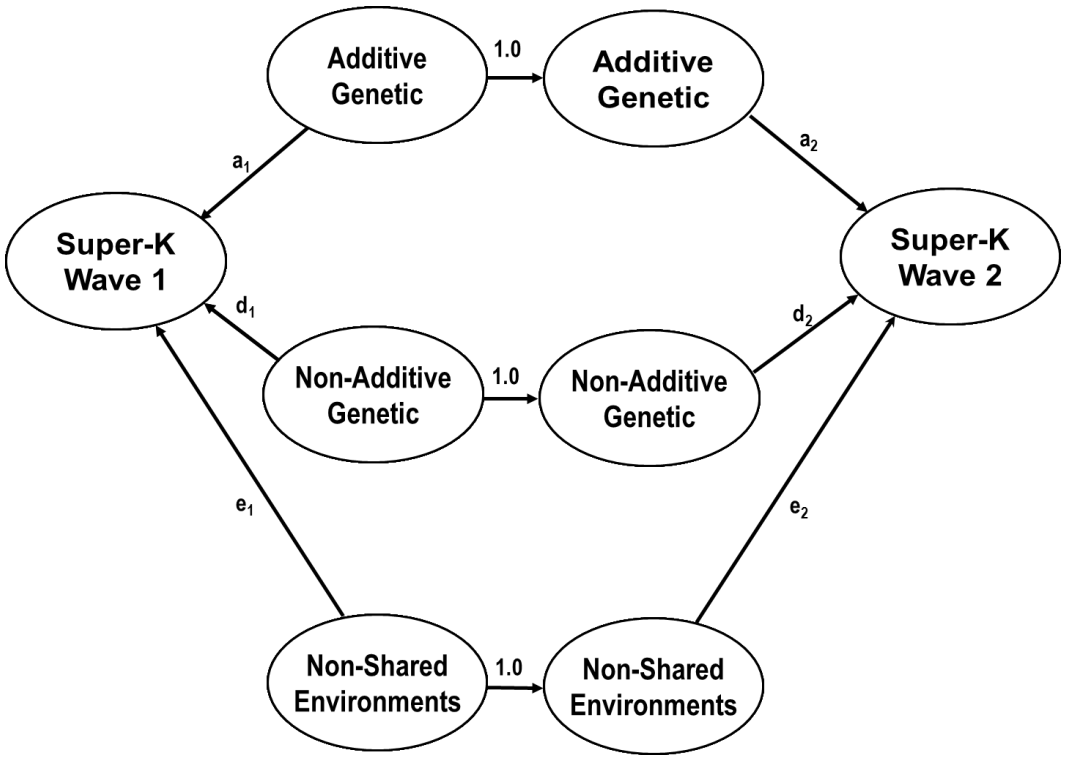
Figure 2.1. ACE Model of Temporal Stability of Super-K Factor.



Similarly, Figure 2.2 conceptually displays an ADE decomposition of that same temporal stability coefficient by using the corresponding behavioral-genetic parameters obtained from the ADE models and Figure 2.3

conceptually displays an AE decomposition of that same temporal stability coefficient by using the corresponding behavioral-genetic parameters obtained from the AE models. In all three alternative longitudinal models, the biometrically-obtained estimates were entered as fixed parameters.

Figure 2.2. ADE Model of Temporal Stability of Super-K Factor.



Figures 3.1-3.37 schematically illustrate that same fundamental logic as extended to the three major lower-order factors underlying the Super-K Factor. These models test the additional hypothesis that the temporal stabilities of these three facets of the Super-K Factor are primarily attributable to the shared behavioral-genetic parameters of the Super-K Factor itself. We tested models of both partial and complete mediation, but only the complete mediation models are shown in these schematics for the sake of simplicity.

Figure 3.1. ACE Model of Temporal Stability of the Higher-Order Super-K Factor as Fully Mediating that of the Lower-Order K-Factor, the Covitality Factor, and the General Factor of Personality.

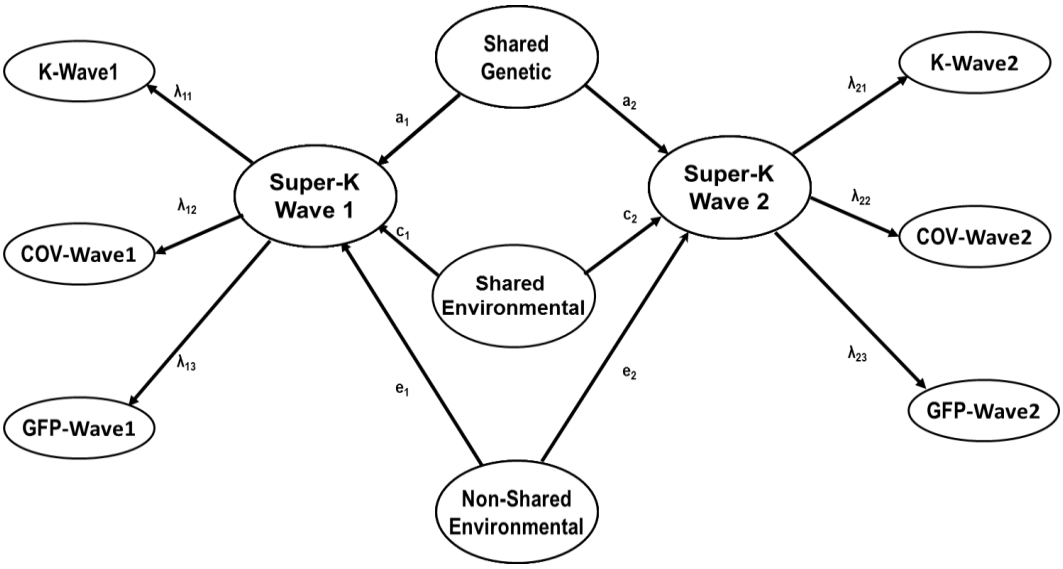


Figure 3.2. ADE Model of Temporal Stability of the Higher-Order Super-K Factor as Fully Mediating that of the Lower-Order K-Factor, the Covitality Factor, and the General Factor of Personality.

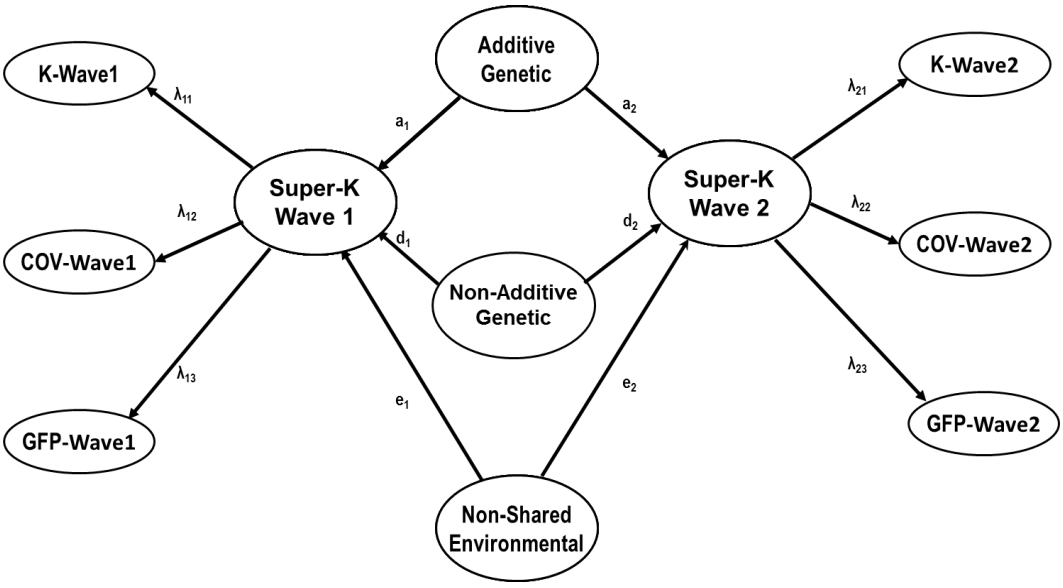
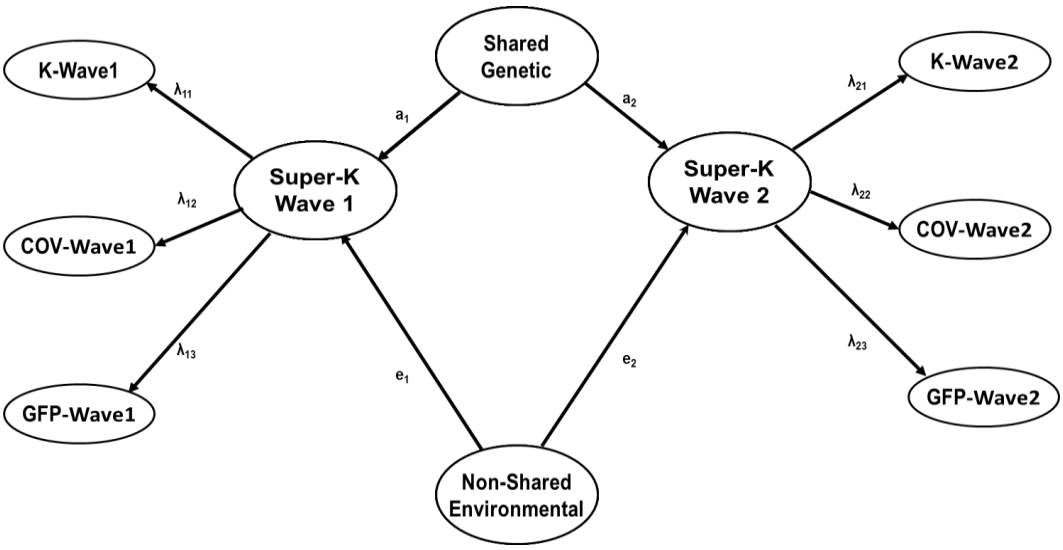


Figure 3.3. AE Model of Temporal Stability of the Higher-Order Super-K Factor as Fully Mediating that of the Lower-Order K-Factor, the Covitality Factor, and the General Factor of Personality.



A systematic quantitative comparison among the relative degrees of fit to the empirical data of these alternative models is thus expected to provide a principled and evidence-based way to decide among these alternative representations of the mechanisms underlying the temporal stability of life history traits.

Results

Observed Phenotypic Temporal Stability Coefficients

To demonstrate the temporal stability of these life history traits across the 10-year interval between the two successive waves of MIDUS data collection, we first calculated the correlations between subscale scores at Wave 1 with scale scores at Wave 2 for each lower-order factor (K-Factor, Personality, and Covitality) to assess whether participants reports on those subscales were consistent over time. Table 4.1 shows the autocorrelations for the subscales used as measures of the K-Factor, which ranged from .54 (Friend Support) to .83 (Religiosity). For Covitality variables (presented in Table 4.2), autocorrelations ranged from .45 (Subjective Well-Being) to .57 (General Health). Autocorrelations of the Big 5 (Openness, Conscientiousness, Extraversion, Agreeableness, and Neuroticism) on the Personality factor remained high across both time points (Table 4.3), and ranged from .64 (Conscientiousness) to .71 (Extraversion). All subscale

autocorrelations were significant. Thus, participants appeared to report similar characteristics across each time period.

Table 4.1
Wave 1 with Wave 2 K-Factor Scale Stabilities

MIDUS Scale	*r*
Self	.70*
Marital	.67*
Family Support	.57*
Friend Support	.54*
Social	.65*
Religiosity	.83*

* $p < .05$

Table 4.2
Wave 1 with Wave 2 Covitality Scale Stabilities

MIDUS Scale	*r*
Negative Affect	.51*
Positive Affect	.52*
General Health	.57*
General Symptoms	.55*
Subjective Well-Being	.45*

* $p < .05$

Table 4.3
Wave 1 with Wave 2 Personality Scale Stabilities

MIDUS Scale	*r*
Openness	.70*
Conscientiousness	.64*
Extraversion	.71*
Agreeableness	.67*
Neuroticism	.66*

* $p < .05$

We then examined the stability of the latent factors across Wave 1 and Wave 2. Results are shown in Table 4.4. Autocorrelations for the K-Factor, Covitality, and Personality remained high (and significant), ranging from .60 (Covitality) to .74 (K-Factor). Super-K, the higher-order factor representing shared variance among these lower-order factors, was also consistent over Wave 1 and Wave 2, with a significant autocorrelation of .72.

Table 4.4
Wave 1 with Wave 2 Super-K Lower-Order and Higher-Order Factor Stabilities

MIDUS Scale	*r*
K-Factor	.74*
Covitality	.60*
Personality	.70*
Super-K	.72*

* $p < .05$

To further examine how the characteristics measured by the subscales changed over time, we calculated the mean difference in scores across the two time points for each individual subscale as well as the corresponding factor. Table 5.1 shows the results of this analysis for the K-Factor. The mean differences for each subscale range from -0.06 (Self) to 0.08 (Family Support). Although these differences are of a small magnitude, we found

Table 5.1
Wave 1 – Wave 2 Scale/Factor Mean Differences: K-Factor

	Mean-Difference	*t*	*p*
Self	-.06	-8.13*	.0001
Marital	.07	7.29*	.0001
Family Support	.08	5.53*	.0001
Friend Support	-.02	-1.34	.1816
Social	.02	1.98	.0477
Religiosity	-.03	-3.57*	.0004
K-Factor	.01	1.53	.1253

* $p < .05$

Table 5.2
Wave 1 – Wave 2 Scale/Factor Mean Differences: Covitality

	Mean-Difference	*t*	*p*
Negative Affect	-.02	-1.26	.2081
Positive Affect	.03	1.86	.0629
General Symptoms	-.00	-1.50	.6167
General Health	-.01	-.38	.7074
Subjective Well-Being	.03	1.56	.1193
Covitality	.02	1.60	.1091

* $p < .05$

that several were statistically significant. Scores on the Self and Religiosity subscales decreased from Wave 1 to Wave 2 (Self: t = -8.13, p = .0001; Religiosity: (t = -3.57, p = .0004). Scores on the Marital, Family Support, and Social subscales increased over this same time period (Marital: t =

7.29, p = .0001; Family Support: t = 5.53, p = .0001; Social: t = 1.98, p = .0477). However, when aggregated into the K-Factor, the mean difference between Wave 1 and Wave 2 was 0.01 (not significant).

Table 5.3
Wave 1 – Wave 2 Scale/Factor Mean Differences: Personality

	Mean-Difference	*t*	*p*
Openness	-.14	-13.88*	.0001
Conscientiousness	.02	1.71	.0874
Extraversion	-.12	-1.78*	.0001
Agreeableness	-.05	-4.39*	.0001
Neuroticism	-.17	-13.38*	.0001
Personality	-.03	-3.57*	.0004

* $p < .05$

Table 5.4
Wave 1 – Wave 2 Factor Mean Differences: Super-K

	Mean-Difference	*t*	*p*
K Factor	.01	1.53	.1253
Covitality	.02	1.60	.1091
Personality	-.03	-3.57*	.0004
Super-K	-.00	-.11	.9136

* $p < .05$

Table 5.2 shows the Covitality subscale and factor mean differences between Wave 1 and Wave 2. The mean differences ranged from -0.02 (Negative Affect) to 0.03 (Positive Affect, Subjective Well-Being), and none were significant at either the subscale level or the factor level.

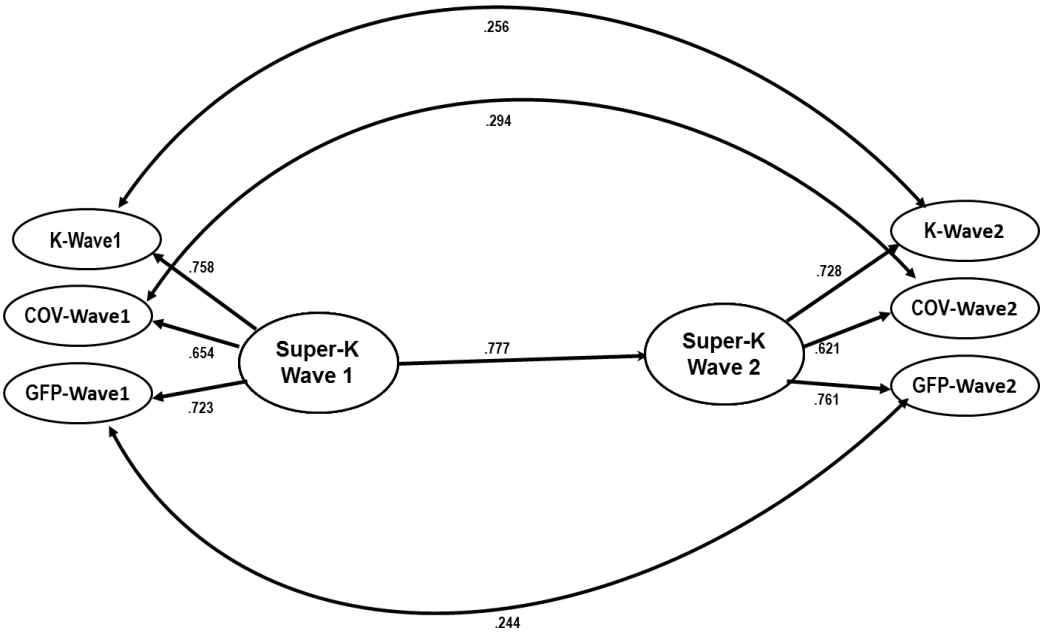
Table 5.3 shows the mean differences for the Personality subscales and factor, which ranged from -0.17 (Neuroticism) to 0.02 (Conscientiousness). We found that Openness, Extraversion, Agreeableness, and Neuroticism decreased from Wave 1 to Wave 2 (Openness: t = -13.88, p = .0001; Extraversion: t = -10.78, p = .0001; Agreeableness: t = -4.39, p = .0001; Neuroticism: t = -13.38, p = .0001). The composite Personality factor also showed a significant decrease over time (t = -3.57, p = .0004).

In Table 5.4, we reproduced the factor mean differences found in Tables 5.1-5.3 and also include our results for the higher-order factor, Super-K. The mean difference is -0.00 and is not significant (p = .9136).

"Hybrid" Longitudinal Structural Equation Models Synthesized from Twin and Non-Twin Data

We start the construction of these hybrid models by first decomposing the temporal stabilities of the three lower-order factors that serve as convergent indicators of the Super-K Factor (the K-Factor, the Covitality Factor, and the General Factor of Personality) into common factor and specific factor pathways with respect to Super-K. This was done using data exclusively from the MIDUS non-twin sample, for which more reliable longitudinal parameters could be estimated, and is displayed graphically in Figure 4.

Figure 4. Common Factor and Specific Factor Pathways for the Temporal Stabilities of the Three Convergent Indicators of the Super-K Factor.



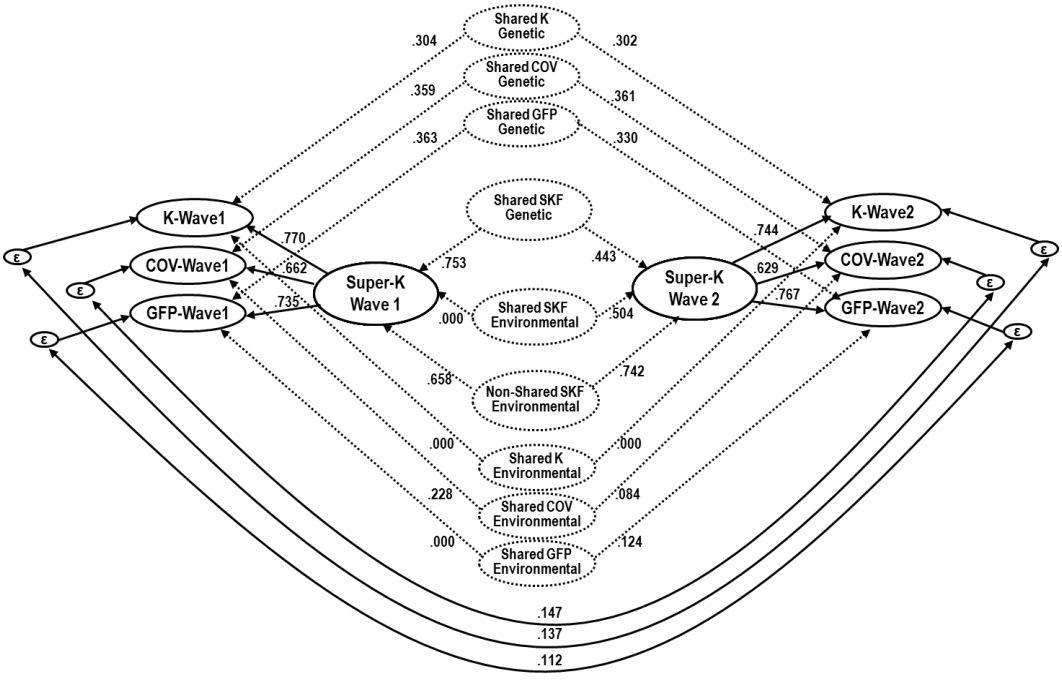
This longitudinal model had an excellent empirical fit to the data by three major practical and parsimonious indices of model goodness-of-fit (χ^2_5 = 27.518*; CFI =.997; NFI = .996; RMSEA =.045). A comparison of the magnitudes of the stability pathways reveals that the common factor temporal stability pathways each accounted for approximately 20% of the stable trait variances and that the specific factor temporal stability pathways each accounted only for less that 10% of the stable trait variances. For example, the expected temporally stable proportion of variance of the lower-order K-Factor would break down path-analytically as (.758*.777*.728) ^{2}= (.429) ^{2}= .184 (or about 18%) for the common factor

pathway but only (.256) ^{2}= .066 (or about 7%) for the specific factor pathway.

For the "hybrid" models, we entered the behavioral-genetic parameters obtained from the biometric models as fixed parameters in place of the empirical stability coefficient for the Super-K Factor. Recall that the validity of imposing of these behavioral-genetic parameters as fixed path coefficients in the longitudinal analysis of the non-twin sample was based on the fact that the *MIDUS* twins and non-twin samples are two nationally-representative samples drawn from the *same* USA adult population, which implies that they should reflect the *same* underlying population parameters, and that the larger usable sample size (*N*=2257) of the *MIDUS* non-twin data permit a degree of reliability for estimation of the longitudinal model parameters not afforded by more limited *MIDUS* twin sample.

The "hybrid" ACE longitudinal model is described graphically in Figure 5.1., with the fixed parameters taken from the biometric estimates shown with dashed lines and the model parameters that were freely-estimated from the phenotypic temporal stabilities of the non-twin sample shown in solid lines.

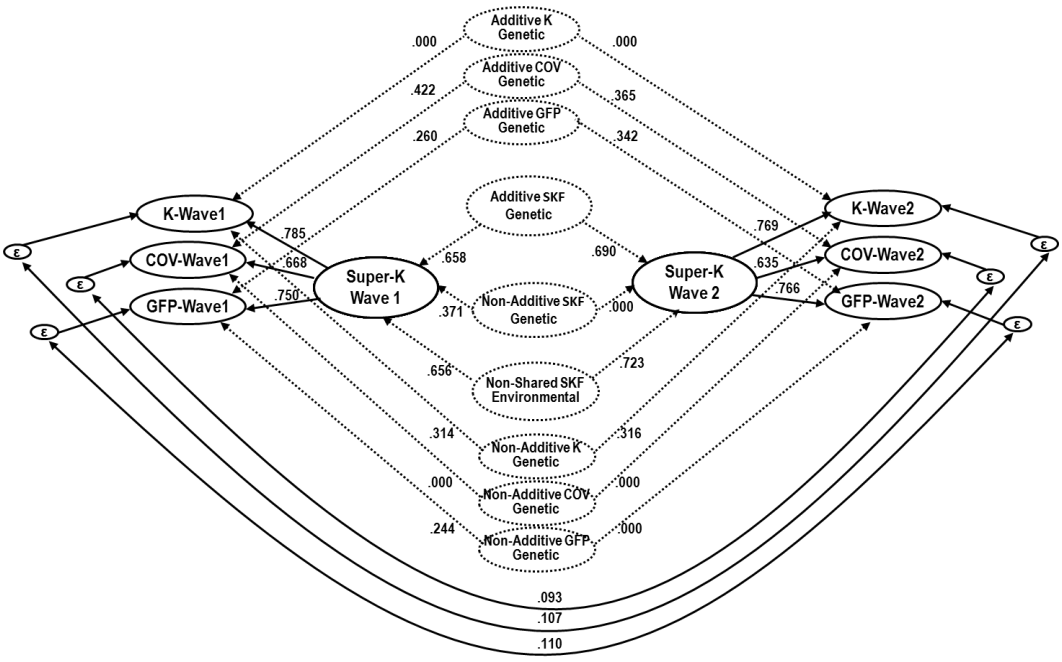
Figure 5.1. "Hybrid" ACE Model for the Common Factor and Specific Factor Pathways for the Temporal Stabilities of the Three Convergent Indicators of the Super-K Factor.



This "hybrid" ACE longitudinal model had an excellent empirical fit to the data by three major practical and parsimonious indices of model goodness-of-fit (χ^2_5 = 41.472*, CFI =.994; NFI = .994; RMSEA =.051).

The "hybrid" ADE longitudinal model is described graphically in Figure 5.2.; again, the fixed parameters taken from the biometric estimates are shown with dashed lines and the model parameters that were freely-estimated from the phenotypic temporal stabilities of the non-twin sample are shown with solid lines.

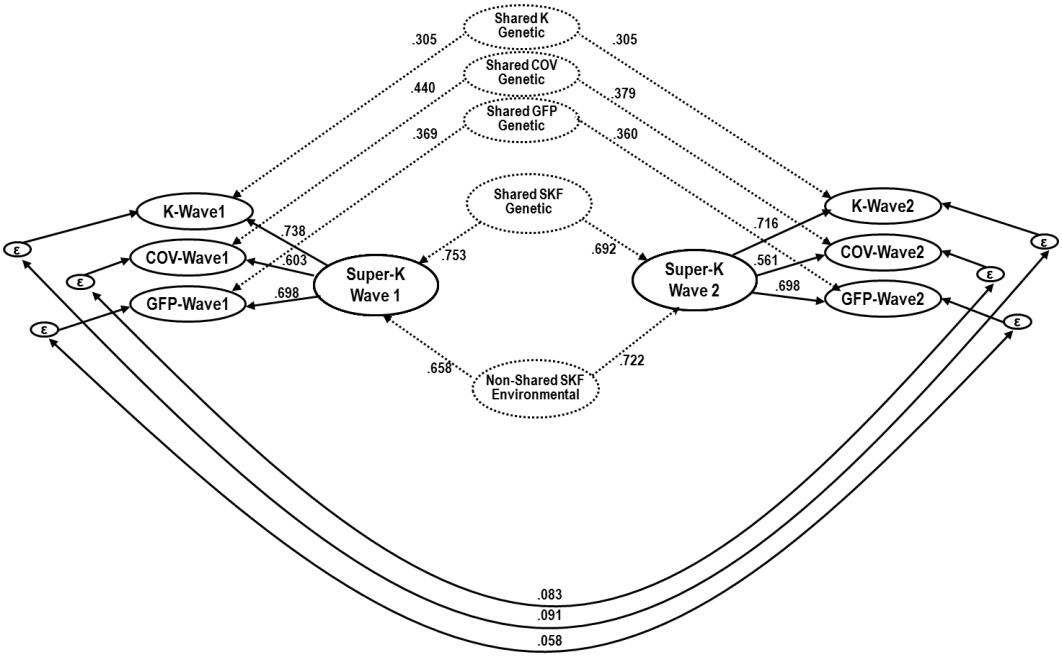
Figure 5.2. "Hybrid" ADE Model for the Common Factor and Specific Factor Pathways for the Temporal Stabilities of the Three Convergent Indicators of the Super-K Factor.



This "hybrid" ADE longitudinal model had a substantially worse empirical fit to the data, as compared with the ACE model, by both the statistical chi-squared test and by the same three practical and parsimonious indices of model goodness-of-fit (χ^2_6 = 289.291*, CFI =.955; NFI = .955; RMSEA =.145).

The "hybrid" AE longitudinal model is described graphically in Figure 5.3.; again, the fixed parameters taken from the biometric estimates are shown with dashed lines and the model parameters that were freely-estimated from the phenotypic temporal stabilities of the non-twin sample are shown with solid lines.

Figure 5.3. "Hybrid" AE Model for the Common Factor and Specific Factor Pathways for the Temporal Stabilities of the Three Convergent Indicators of the Super-K Factor.



This "hybrid" AE longitudinal model had an even worse empirical fit to the data than the ADE model, as compared with the ACE model, by both the statistical chi-squared test and by the same three practical and parsimonious indices of model goodness-of-fit (χ^2_6 = 675.874*, CFI =.895; NFI = .894; RMSEA =.223).

The optimal "hybrid" longitudinal model tested thus appears to be the ACE model, and this is the one that we will focus on in the following discussion.

Discussion

The empirical results we have presented would suggest that a *substantial* portion of the cross-temporal stability of LH traits, and perhaps of individual-difference traits in general, is attributable to *shared genetic influences* that are *invariant* over time. These influences appear to be substantially larger in magnitude at *higher* levels of data aggregation, presumably reflecting higher cross-situational, and thus cross-temporal, stabilities. However, there are also statistically significant shared genetic influences that are non-trivial in magnitude to be found at lower levels of data aggregation, presumably reflecting somewhat *lower* cross-situational, and thus cross-temporal, stabilities.

The temporal stability results of the present study are consistent with past research investigating trait consistency among human (e.g., Roberts & Del Vecchio, 2000) and non-human animals (e.g., Gosling, 2001). Among humans, individual difference traits, depending on the design of the study and the selection of traits examined, ranged from medium to large in magnitude (e.g., r = .46 to .69, Bratko & Butkovic, 2007; r = .32 to .56, Ganiban, Saudino, Ulbricht, Neiderhiser, & Reiss, 2008; r = .70 to .86, Johnson, McGue, & Krueger, 2005).

Further, when we examined the *mechanisms* of temporal stability, the results of the present study are consistent with behavioral genetic research investigating the stability of individual differences. Broadly, past research utilizing behavioral genetic biometric models revealed that the temporal stability of individual difference traits such as briskness, perseveration, extraversion, neuroticism, can typically be explained predominantly by a combination of shared genetic and non-shared environmental influences (Bratko & Butkovic, 2007; Johnson, McGue, & Kreuger, 2005; Kandler, Bleidorn, Riemann, Angleitner, & Spinath, 2012; Kandler, Riemann, & Angleitner, 2013).

Future Research Directions

The implications of this work are both substantial and far-reaching. The parametric results of these models indicate that there is a substantial genetic component underlying the temporal stability of individual differences (See Rowe, 1994, for a discussion on the genetic effects on individual difference traits). Still, almost a quarter of the temporally stable variance — $(.658*.742)^2 = (.488)^2 = .238$ or about 24% — in the Super-K Factor over this time interval is attributable to non-shared environmental factors. Unlike when using measured variable models of heritability, it would be inaccurate to characterize any portion of this variance as simply random "error" variance. Although, statistically speaking, this residual is made up of remaining variance after accounting for genetic and shared environmental variance, it is the *residual common factor variance*, or "disturbance" in classical psychometric terms, which constitutes a portion of the true score variance that is merely not explained by the predictors modeled and does not (at least in principle) include measurement error.

Moreover, although random idiosyncratic events (e.g., accidents, illnesses) are sure to contribute to differential outcomes within families, there remain potentially interesting avenues to explore systematic events that produce non-shared environmental effects. Plomin and Daniels (2011) describe several such events, including birth-order and gender differences, differential treatment among siblings and parents, and extrafamilial networks, such as peer groups, teachers, and media. These

types of events constitute "objective" non-shared events, or events that are experienced by only one sibling. Another type of non-shared event has been termed "effective" non-shared events, which are defined by the differential outcomes produced. For instance, even a shared experience such as parental divorce may produce different effects on siblings, and would therefore be absorbed by non-shared environmental variance (Turkheimer & Waldron, 2000).

Thus, non-shared environment offers a potentially rich source of empirical study. Future research in this area should examine these systematic sources contributing to non-shared environmental variance. This may be achieved by implementing a basic research design wherein both shared and non-shared environment events are actually *measured* for pairs of siblings, rather than merely *inferred* and *estimated* as model residuals (see Turkheimer & Waldron, 2000 for a full explanation), whether these represent random measurement *errors*, systematic test-specific *method* effects, or true-score *disturbance* terms.

A relevant limitation of biometric models, such as those applied above, is that the shared genetic and the non-shared environmental variance are modeled as orthogonal (uncorrelated) components. This construction, however, does not completely reflect real human development in that gene-environment *correlations* (which are fundamentally different from gene-environment *interactions*, although these may co-occur) are common. These gene-environment correlations may be generated by a variety of distinct causal mechanisms. Buss (1987) listed the following three common sources of such correlations: (1) *Selection*, in which an individual organism either seeks out or avoids certain environments; (2) *Evocation*, in which the organism elicits predictable reactions from other individuals in its environment, whether intentionally or unintentionally; and (3) *Manipulation*, in which the organism uses specific tactics to alter its environment, presumably modifying it into one to which the individual is genetically better adapted.

Another potentially productive future direction for research derives from the fact that life history theory provides a broad explanation for intra-individual variation in physiological functioning, suggesting that biological operating system constraints are ecologically-contingent responses to the environment. One possible application of the life history approach is in the fields of health psychology and epidemiology, which seek to explain the linkage between socioeconomic status and health (Adler & Newman, 2002). It has been speculated that this correlation might be spurious, which means that a third variable might be the common cause of the observed discrepancies both socioeconomic status and health (Adler, Boyce, Chesney, Cohen, Folkman, Kahn, & Syme, 1994). The evolutionary-developmental approach would suggest that life history strategy represents a plausible candidate for that possibly causal third

variable, in that different modal life history strategies may be contributing to differential degrees of physical deterioration among socioeconomic strata – providing a consilient mechanistic explanation for this established finding (Del Giudice, Ellis, & Shirtcliff, 2011).

The results of this paper, however, take life history theorizing a step further by providing a more nuanced method for explaining elevated levels of physiological deterioration in unstable environments. By biometrically disentangling the shared genetic (a^2) and non-shared environment (e^2) proportions of variance in both the higher-order and lower-order factors, as were modeled in this paper, health researchers could parse and quantify the relative influences of these various explanations. Recent progress has, in fact, been made in estimating the individual-genome-level heritabilities (called *individual transmissibilities*; Woodley, Figueredo, Cabeza De Baca, Fernandes, Madison, Wolf, Black, & Olderbak, under review) for life history traits, making it possible to assess differential heritable levels of both preparedness and plasticity with respect to the ecological stabilities that shape their evolution and development (see also Ellis et al., 2009).

Broader Theoretical Implications

While the content of the biometric model focused solely on the individual difference trait of life history strategy (*K*), the results of this model provide far-reaching implications in other relevant fields of inquiry. The heart of the argument that we have presented in this paper can be summarized as an essentially psychometric argument. Latent common factors representing stable individual difference traits are typically multi-operationalized so as to be *domain-general,* possessing *cross-situational stability* across many specific environmental contexts. This means that only the *common* or domain-general variance will be extracted from any array of domain-specific indicators such as the resource-allocation-based life history traits, *e.g.,* the subscales of the Arizona Life History Battery (ALHB), comprising the *K*-Factor. Domain-general latent common factors should therefore also possess substantial *cross-temporal stability* because what typically changes over time are the *specific environmental contexts* that the individual encounters over the lifespan. This means that such changing environmental contexts cannot logically be held responsible for the cross-temporal stability of traits, and only any *cross-temporal stability* that may exist across *varying* environmental contexts could reasonably be held accountable for the cross-temporal stability of traits (as also demonstrated by Bratko & Butkovic, 2007). Exactly *how much* cross-temporal stability of environmental contexts exists across the human lifespan, and whether this is sufficient to explain the stability of traits, is a matter for future empirical research to estimate (see Figueredo, Woodley, & Jacobs, 2014). Nevertheless, the *single* common causal influence that is

unquestionably persistent over time is the individual's *genome* (meaning *DNA* nucleotide sequence). Although epigenetic modifications of gene-expression can and do occur over the lifespan, these are often triggered by external developmental events and must thus be logically attributed to varying environmental contexts. These additional effects of varying environmental contexts are modeled in the *non-shared environment* portion of the temporal stability, which was also of substantial magnitude in the model presented here.

More broadly speaking, our results might be interpreted to suggest (albeit very indirectly) that the subjective persistence of the "Self" might therefore be largely attributable to and defined by each individual's unique *genome* in a perpetual state of mutual interaction (*causal transaction*) with one's unique and partially self-constructed (meaning *selected*, *evoked*, and *manipulated*) environment. We understand that some might characterize this view as one of genetic determinism, which we explicitly eschew. Our estimated heritability coefficients indicate that the degree of genetic influence actually in evidence would render the word as outrageously inflated the use of the word "determinism" as a realistic description. Nevertheless, humans naturally fear control by outside agencies, even if it is only partial control or "influence", because they suspect that other living things (whether allospecific parasites or conspecific competitors) might use this leverage to manipulate them into functioning contrary to their own evolved self-interest. However, to those who fear the notion that individuals might be "biochemically-controlled" or "biologically determined" by one's own genes, we can simply paraphrase a former King of France (Louis XIV, 1638–1715) with the retort: *"Les gènes, c'est moi!"* We would follow this potential insight with another famous quip from the Eastern sage Confucius (孔夫子 Kǒng Fūzǐ, 551–479 BC), as applied to our highly hereditarian interpretation of the causes underlying the temporal consistency of individual difference traits: "No matter where you go, there you are!

Author Notes: We wish to thank Michael Anthony Woodley of Menie, Heitor Barcellos Ferreira Fernandes, Hobart Harrington Cleveland III, and the University of Arizona's Ethology and Evolutionary Psychology (*EEP*) Laboratory and Evaluation Group for Analysis of Data (*EGAD*) for feedback on this work while still in progress.

References

Adler, N. E., Boyce, T., Chesney, M. A., Cohen, S., Folkman, S., Kahn, R. L., & Syme, S. L. (1994). Socioeconomic status and health: The challenge of the gradient. *American Psychologist, 49,* 15-23.

Adler, N. E., & Newman, K. (2002). Socioeconomic disparities in health: Pathways and policies. *Health Affairs, 21,* 60-76.

Belsky, J., Schlomer, G. L., & Ellis, B. J. (2012). Beyond cumulative risk: Distinguishing harshness and unpredictability as determinants of parenting and early life history strategy. *Developmental Psychology, 48*, 662-673.

Belsky, J., Steinberg, L., & Draper, P. (1991). Childhood experience, interpersonal development, and reproductive strategy: An evolutionary theory of socialization. *Child Development, 62*, 647-670.

Bouchard, T. J. & McGue, M. (1990). Genetic and rearing environmental influences on adult personality: An analysis of adopted twins reared apart. *Journal of Personality, 58*, 263-292.

Branigan, A. R., McCallum, K. J. & Freese, J. (2013). *Variation in the Heritability of Educational Attainment: An International Meta-Analysis*. Working Paper Series. Northwestern University: Institute for Policy Research.

Bratko, D. & Butkovic, A. (2007). Stability of genetic environmental effects from adolescence to young adulthood: Results of Croatian longitudinal twin study of personality. *Twin Research and Human Genetics, 10*, 151-157.

Brim, O. G., Baltes, P. B., Bumpass, L. L., Cleary, P. D., Featherman, D. L., Hazzard, W. R., Kessler, R. C. M., Lachman, E., Markus, H. R., Marmot, M. G., Rossi, A. S., Ryff, C. D., & Shweder, R. A. (2000). *National survey of midlife development in the United States MIDUS), 1995-1996* [Computer file]. ICPSR version. Ann Arbor, MI: DataStat, Inc./Boston, MA: Harvard Medical School, Dept. of Health Care Policy [producers], 1996. Ann Arbor, MI: Inter-university Consortium for Political and Social Research [distributor], 2000.

Buss, D. M. (1987). Selection, evocation, and manipulation. *Journal of Personality and Social Psychology, 53*, 1214-1221.

Caspi, A., Roberts, B. W., & Shiner, R. L. (2005). Personality development: Stability and change. *Annual Review of Psychology*, *56*, 453–484.

Costa, P. T., Jr., McCrae, R. R. (1997). Longitudinal study of adult personality. In R. Hogan, J. Johnson, & S. Briggs (Eds.), *Handbook of Personality Psychology* (pp. 269–92). San Diego: Academic.

Del Giudice, M., Ellis, B. J., & Shirtcliff, E. A. (2011). The adaptive calibration model of stress responsivity. *Neuroscience and Biohavioral Reviews, 35,* 1562-1592.

Ellis, B. J., Figueredo, A. J., Brumbach, B. H., Schlomer, G. L. (2009) Fundamental dimensions of environmental risk: The impact of harsh versus unpredictable environments on the evolution and development of life-history strategies. *Human Nature, 20*, 204–298.

Ellis, B. J., Del Giudice, M., Dishion, T. J., Figueredo, A. J., Gray, P., Griskevicius, V., . . . & Wilson, D. S. (2012). The evolutionary basis of risky adolescent

behavior: Implications for science, policy, and practice. *Developmental Psychology, 48*, 598–623.

Figueredo, A. J., Cabeza De Baca, T., & Woodley, M. A. (2013). The measurement of human life history strategy. *Personality and Individual Differences, 55*, 251-255.

Figueredo, A. J., Cox, R. L., & Rhine, R. J. (1995). A generalizability analysis of subjective personality assessments in the stumptail macaque and the zebra finch. *Multivariate Behavioral Research, 30,* 167-197.

Figueredo, A. J., & Jacobs, W. J. (2010). Aggression, risk-taking, and alternative life history strategies: The behavioral ecology of social deviance. In M. Frias-Armenta & V. Corral-Verdugo (Eds.), *Biopsychosocial Perspectives on Interpersonal Violence*. Hauppauge, NY: Nova Science Publishers.

Figueredo, A. J., McKnight, P. E., McKnight, K. M., & Sidani, S. (2000). Multivariate modeling of missing data within and across assessment waves. *Addiction, 95*(S3), S361-S380.

Figueredo, A. J., & Rushton, J. P. (2009). Evidence for shared genetic dominance between the general factor of personality, mental and physical health, and life history traits. *Twin Research and Human Genetics, 12*, 555-563.

Figueredo, A. J., Vásquez, G., Brumbach, B. H., & Schneider, S. M. R. (2004). The heritability of life history strategy: The K-factor, covitality, and personality. *Social Biology*, *51*, 121-143.

Figueredo, A. J., Vásquez, G., Brumbach, B. H., & Schneider, S. M. R. (2007). The K-factor, covitality, and personality: A psychometric test of life history theory. *Human Nature*, *18*, 47-73.

Figueredo, A. J., Vásquez, G., Brumbach, B. H., Sefcek, J. A., Kirsner, B. R., & Jacobs, W. J. (2005). The K-Factor: Individual differences in life history strategy. *Personality and Individual Differences, 39,* 1349-1360.

Figueredo, A. J., Vásquez, G., Brumbach, B. H., Schneider, S. M. R., Sefcek, J. A., Tal, I. R., Hill, D., Wenner, C. J., & Jacobs, W. J. (2006). Consilience and life history theory: From genes to brain to reproductive strategy. *Developmental Review, 26*, 243-275.

Figueredo, A. J., Woodley, M. A., Brown, S. D., & Ross, S. D. (2013). Multiple successful tests of the strategic differentiation-integration effort (SD-IE) hypothesis. *Journal of Social, Evolutionary, and Cultural Psychology, 7,* 361-383.

Figueredo, A. J., Woodley, M. A., & Jacobs, W. J. (2014). The Evolutionary Psychology of the General Factor of Personality: A Hierarchical Life History Model. In D.M. Buss, (Ed.), *Handbook of Evolutionary Psychology, 2nd Ed*. Hoboken, NJ: John Wiley & Sons.

Fratkin, J. L., Sinn, D. L., Patall, E. A., & Gosling, S. D. (2013). Personality consistency in dogs: A meta-analysis. *PLoS ONE, 8*(1), e54907. doi:10.1371/journal.pone.0054907.

Ganiban, J. M., Saudino, K. J., Ulbricht, J., Neiderhiser, J. M., & Reiss, D. (2008). Stability and change in temperament across adolescence. *Journal of Personality and Social Psychology, 95*, 222-236.

Gore, M. L. (1982). See you. On *A Broken Frame* [CD]. London: Mute Records.

Gorsuch, R. L. (1983). *Factor Analysis* (2nd ed.). Hillsdale, New Jersey: Lawrence Erlbaum Associates.

Gosling, S. D. (2001). From mice to men: What can we learn about personality from animal research? *Psychological Bulletin, 127,* 45-86.

Hopwood, C. J., Donnellan, M. B., Blonigen, D. M., Krueger, R. F., McGue, M., Iancono, W. G., & Burt, S. A. (2011). Genetic and environmental influences on personality trait stability and growth during the transition to adulthood: A three-wave longitudinal study. *Journal of Personality and Social Psychology, 100,* 545-556.

James, W. (1918). *The principles of psychology*. New York: Henry Holt and Company.

Jang, K. L. & Livesley, W. J. (1996). Heritability of the Big Five personality dimensions and their facets: A twin study. *Journal of Personality, 64*, 577-591.

Johnson, W., McGue, M., & Krueger, R. F. (2005). Personality stability in late adulthood: A behavioral genetic analysis. *Journal of Personality, 73*, 523-551.

Kandler, C., Bleidorn, W., Riemann, R., Angleitner, A., & Spinath, F. M. (2012). Life events as environmental states and genetic traits and the role of personality: A longitudinal twin study. *Behavior Genetics, 42,* 57-72.

Kandler, C., Riemann, R., & Angleitner, A. (2013). Patterns and sources of continuity and change of energetic and temporal aspects of temperament in adulthood: A longitudinal twin study of self- and peer reports. *Developmental Psychology, 49,* 1739-1753.

Morizot, J., & Le Blanc, M. (2003). Continuity and change in personality traits from adolescence to midlife: A 25-year longitudinal study comparing representative an adjudicated men. *Journal of Personality, 71,* 705-756.

Plomin, R. & Daniels, D. (2011). Why are children in the same family so different from one another? *International Journal of Epidemiology, 40,* 563-582.

Ratanen, J., Metasäpelto, R-L., Feldt, T., Pulkkinen, L., & Kokko, K. (2007). Long-term stability in big five traits in adulthood. *Scandinavian Journal of Psychology, 48,* 511-548.

Roberts, B. W., Caspi, A., & Moffit, T. E. (2001). The kids are alright: Growth and stability in personality development from adolescence to adulthood. *Journal of Personality and Social Psychology, 81,* 670-683.

Roberts, B. W. & Del Vecchio, W. F. (2000). The rank order consistency of personality traits from childhood to old age: A quantitative review of longitudinal studies. *Psychological Bulletin, 126*, 3–25.

Rowe, D. C. (1994). *The limits of family influence: Genes, experience, and behavior*. New York: The Guilford Press.

Rushton, J. P., Fulker, D. W., Neale, M. C., Nias, D. K. B., & Eysenck, H. J. (1986). Altruism and aggression: The heritability of individual differences. *The Journal of Personality and Social Psychology, 50*, 1192-1198.

Schlomer, G. L., Bauman, S., & Card, N. A. (2010). Best practices for missing data management in counseling psychology. *Journal of Counseling Psychology, 57*(1), 1-10.

Simpson, J. A., Griskevicius, V., Kuo, S. I-C., Sung, S., & Collins, W. A. (2012). Evolution, stress, and sensitive periods: The influence of unpredictability in early versus late childhood on sex and risky behaviour. *Developmental Psychology, 48,* 674-686.

Stevenson-Hinde, J., Stillwell-Barnes, R., & Zunz, M. (1980). Subjective assessment of rhesus monkeys over four successive years. *Primates, 21,* 66-82.

Stevenson-Hinde, J., & Zunz, M. (1978). Subjective assessment of individual rhesus monkeys. *Primates, 19,* 473-482.

Turkheimer, E. & Waldron, M. (2000). Nonshared environment: A theoretical, methodological, and quantitative review. *Psychological Bulletin, 126*, 78-108.

Woodley, M. A., Figueredo, A. J., Brown, S. D., Ross, K. C. (2013). Four successful tests of the Cognitive Differentiation-Integration Effort hypothesis. *Intelligence, 41*, 832-842.

Woodley, M. A., Figueredo, A. J., Cabeza De Baca, T., Fernandes, H. B. F., Madison, G., Wolf, P. S. A., Black, C. J., & Olderbak, S. (Under Review). The genomic-level heritabilities of preparedness and plasticity in human life history: the strategic differentiation and integration of genetic transmissibilities.