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Hybrid Behavior-Genetic Models of the Confounding Gene-Environment Correlations in the Development of Life History Strategy: Two Convergent Approaches

Aurelio José Figueredo, PhD Tomás Cabeza de Baca, PhD
University of Arizona

George B. Richardson, PhD
University of Cincinnati

Traditional theories of development and evolutionary developmental psychology propose that early environmental experiences shape an individual's developmental trajectory. According to the Adaptive Calibration Model (ACM), for example, calibration of speed of life history strategy to ecological cues encountered during development contributes to behavior that is conditionally adaptive to the organism's environment. These theories emphasize the role of environmental influences and typically do not use designs that control potential genetic confounds. To address this methodological problem, the current study used a genetically informative design to test whether the phenotypic associations of parental instability and abuse with a slow life history factor were confounded by common genetic factors. We analyzed twin and singleton data from the Midlife in the United States (MIDUS) Survey using two convergent structural equation modeling approaches. Both approaches suggest that, when accounting for shared genetic variance across instability, abuse, and slow life history, some hypothesized environmental pathways between the early environmental measures and slow life history were not required. Once genetic factors were controlled, only parental instability was directly related to slow life history, while other hypothesized environmental pathways were non-significant. This suggests that developmental models that emphasize environmental and contextual pathways should control for possible genetic confounds.

Keywords: Life History Theory, Behavioral Genetics, Early Adversity, Behavioral Development, Parental Instability

Evolutionary Developmental Models

Evolutionary perspectives on early life experiences and ontogeny have provided differing interpretations of the role of early experiences on the plasticity of child development, particularly by focusing on the impact of parental quality and household dynamics. The theory of differential susceptibility to environmental influence (Belsky & Pluess, 2009) proposes that offspring residing within the same household and sharing the same biological parents might nonetheless possess differing degrees of genetic susceptibility to immediate environmental influences. Thus, more "susceptibility genes" increases lability and plasticity in response to the

social and contextual cues conveyed by parents and close others to developing children. Conversely, fewer susceptibility genes will produce less lability and plasticity in development, and produce less perturbation and influence to social cues.

A different subset of evolutionary developmental models propose similar ideas as those published by Belsky and colleagues (2009). Similarly, the biological sensitivity to context theory, also posits that there are systematic individual differences between children in sensitivity to rearing environments (Boyce & Ellis, 2005). While acknowledging the impact of heritable genetic differences upon development and sensitivity to immediate context, allelic variation's influence on the collective calibration of the stress response system (e.g., endophenotypes) effected by rearing conditions is more greatly emphasized (Boyce & Ellis, 2005; Del Giudice, Ellis, & Shirtcliff, 2011; Ellis, Boyce, Belsky, Bakermans-Kranenburg & Van IJzendoorn, 2011). Hyperarousal and vigilance, would thus be considered as conditional adaptations to environmental features contingently shifting the development of physiological (Del Giudice et al., 2011) and cognitive systems (Ellis, Bianchi, Griskevicius, & Frankenhuis,, 2017) to produce advantages in survival, navigation, and reproduction (Ellis, Figueredo, Brumbach, & Schlomer,, 2009; Figueredo et al., 2006), but also generating costs in long-term health and quality of life (Shonkoff, Boyce, & McEwen, 2009). Whereas the original differential susceptibility theory posits a bet-hedging model, the biological sensitivity to context theory adds that individual differences in the degree of sensitivity guides children in the regulation of development. These sensitivities therefore represent conditional adaptations for adapting to their putative environment as cued by conditions of rearing (Ellis et al., 2011; Del Giudice et al., 2011).

The latest evolutionary developmental model proposed is the adaptive calibration model (Cabeza de Baca, Wahl, Barnett, Figueredo, & Ellis,, 2016; Belsky, Steinberg, & Draper, 1991; Del Giudice et al., 2011; Taylor, May, & Seeman, 2011). This model builds upon the reasoning used by prior evolutionary developmental models, arguing that development is conditionally adaptive, but adding the influence of sex and other individual differences on developmental regulation. Additionally, the adaptive calibration model explicates how stress response systems coordinate to create emergent life history strategies that are guided by experience during sensitive developmental stages.

Methodological Limitations of Evolutionary Developmental Models

Much research in pediatrics and development suggests that adverse early experiences profoundly affect different facets of ontogeny (e.g.,

physiological, social/behavioral, and reproduction). Standard interpretations of this literature propose that early life adversity produces psychosocial deficits that impair the "normative" development of individuals. Subsequent conceptualizations instead emphasize contextual factors in influencing child development, proposing a continuum of ontogenetic traits in place of a normative paradigm of development in humans (Roubinov & Boyce, 2017). For example, the adaptive calibration model (described above) posits that different developmental trajectories are adaptive under different environmental circumstances. Although the development of a slower life history strategy is presumably more adaptive under more normative circumstances, the development of a faster life history strategy is presumably more adaptive under harsher or more unpredictable conditions (Ellis et al., 2009).

Although standard views of development now incorporate the effect of context in early childhood experiences, they continue to ascribe child outcomes mostly to environmental inputs. Rowe (1994) provided a thought question for researchers who emphasize the influence of the environment, suggesting that when researchers failing to use models that are both genetically and environmentally controlled (e.g., Ellis, Schlomer, Tilley, & Butler, 2012), ascribing developmental effects exclusively to environmental inputs is equivalent to defending a confounded model. However, the prevailing paradigm within psychology no longer debates nature versus nurture but focuses instead on the causal transactions among them in influencing individual differences; nonetheless, genetic influences upon effects of early life experiences on child development are not widely discussed.

Traditional developmental and evolutionary developmental researchers have largely focused on the impact of early adversity and parental quality on a range of child outcomes. Both sets of literature have generally found that adverse and poor-quality environments are associated with poorer child outcomes. Evolutionary developmental models assert that poor or deleterious end-points are actually products of a coordinated life history strategy designed to assist the child's navigation and manipulation of the environment. This coordinated life history strategy thus works across varying levels of the child's development, including physiology, behavior, and cognition. Although evolutionary developmental models provide important theoretical and empirical contributions, one limitation found in the research is the genetic confounds apparent in the purported impact of early environmental contexts. It is possible that research tools designed to measure the quality of early rearing environments, such as parental abuse, support, marital stability, reflect environmental factors that may also be unintentionally influenced by a parent's genetics (e.g., Plomin & Bergeman, 1991). Because a child's environment is largely constructed by the parents and the research

conducted on these questions is often genetically uninformed, the degree of genetic relatedness between the child and its parents may confound associations between parental behaviors during childhood and the subsequent offspring behaviors during adulthood. Thus, the offspring may come to resemble the parents behaviorally in adulthood because of the influence of the parentally-constructed childhood environment; alternatively, the offspring may come to resemble the parents behaviorally in adulthood because of the common influence of the genes that are typically shared by the parent and offspring. Either way, the degree of the environmental influence may be impacted via shared heritable mechanisms (Plomin & Bergeman, 1991; Plomin, Reiss, Hetherington, & Howe , 1994). This is because the vast majority of social parents are also genetic parents. Although this circumstance is not universally the case, as in adoptive homes (which constitute 2-4% of all families in the USA; Adoption Statistics, 2012), it is nevertheless the case that for a representative population sample, the overall genetic correlation between social parents and offspring, aggregated across all homes, will be significant and positive. We are not saying that this relation applies to all individual cases, but that the correlations reflected in the sample statistics need to be corrected for the effects of these population parameters

We argue that this heritable influence should work above and beyond the gene-environment correlations that have been proposed in developmental research, such as active, passive, and evocative gene-environment correlations. Certain theorists (e.g., Kong, Thorleifsson, Frigge, Vilhjalmsson, Young, Thorgeirsson, et al., 2018) have proposed that we distinguish between direct effects of genes and those that are mediated through the environment, which may be characterized as indirect. The concept of a "gene swarm" surrounding the developing organism (Hertler, Figueredo, Peñaherrera Aguirre, Fernandes, & Woodley of Menie, 2018) refers to the indirect effects transmitted through modifications to the developmental environment that are produced by genes shared by both the developing offspring and its parents, as well as by any related siblings and alloparents residing in the immediate vicinity.

Although we stress that it is unethical to place the blame on a child for the conditions of their early environment, we argue that a child's shared genetics from closely related conspecifics, such as parents, siblings, and genetically-related alloparents may nevertheless be a major unintentional influence on the conditions of rearing and household environments (Hertler et al., 2018). Thus, we argue that the empirical investigation of early adverse environments and child outcomes must be fitted with genetically-informed models, such as those used in behavioral genetics. Biometric analyses that are performed in behavior genetics can parse out the influence of both genetic and environmental variation and estimate coefficients of the impact of both on outcomes.

Furthermore, we believe that it is insufficient to merely consider the traditional univariate heritabilities of the individual traits in addressing this methodological problem, as has been done by some critics of the developmental literature (e.g., Rowe, 1994). We must also address the bivariate heritabilities or the genetic correlations among these traits to determine whether the associations that have previously been attributed to environmental effects are indeed causal pathways and not spurious correlations generated by common genetic influences.

The Present Study

We use two convergent methods of estimation (Falconer and DeFries-Fulker) in the context of factor analytic structural equation model to explore these behavioral genetic and developmental relations. The data used do not contain an adequate sample of behavior from the parents and their offspring to represent the concept of the gene swarm as a whole, but we hope that this will be minimally sufficient as a proof of concept to demonstrate the novel quantitative methods we developed to address the theoretical dilemmas posed by our broader conceptualizations. The goal of both approaches was to estimate the genetic contributions of the influence of certain parental patterns of behavior on early developmental environments on the adult life history outcomes of the offspring, then determine what environmentally-mediated effects remained statistically significant between the same variables after the prior behavior-genetic influences had been statistically controlled.

For both convergent approaches, we constructed hybrid structural models (see Figueredo, Cabeza de Baca, & Black, 2014) by specifying fixed parameters with values obtained from the genetically-informative twin sample within a structural equations model in which all other parameters were estimated from the non-genetically-informative singleton sample. The use of this procedure is supported by the fact that our independent twin and singleton (non-twin) samples were drawn from the same general population, and should therefore reflect the same underlying population parameters, such as heritability coefficients, according to statistical sampling theory.

General Method

Overview

Sample

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 by random digit dialing (RDD), 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. Wave 1 was collected over a one-year period from 1995-1996 (*N*= 3487), and Wave 2 was collected over a two year period from 2004-2006 (*N*= 2257). The MIDUS sample included data from a genetically-informative random digit dialing sub-sample of MZ and DZ twins, as well as on a non-genetically-informative sample of singletons (non-twins). For the present study, only same-sex DZ twin pairs were used for analysis, to avoid confounding individual differences between twins with the effects of twin sex.

Measures

Parental Instability. Parental instability was conceptualized based on Ellis and colleague's (2009) definition of environmental unpredictability, which they defined at the population level as variable and stochastic levels of environmental harshness (e.g., illness, disability, and death that is agnostic to adaptation). At the individual level, environmental unpredictability – in this case parental instability – has been conceptualized as events or experiences that increase the upheavals with the household during early childhood. For our Parental Instability Scale, we used a unit-weighted factor scale (Gorsuch, 1983) composed of the following items from the MIDUS Survey: (1) whether one or more parents drank often that it caused problems (yes/no); (2) whether one or more parents drug use often caused problems (yes/no); (3) the number of moves to new neighborhoods or towns during childhood; and (4) whether parents were divorced (yes/no). The standardized scores of these items were averaged together to yield a composite parental instability score, in which higher scores denoted more parental instability. The part-whole correlations displayed on Table 1 revealed a satisfactory degree of convergent validity between these items.

Parental Abuse. The Parental Abuse Scale included unit-weighted factor scales for the following three sets of parental aggression measures, averaged between those of the mothers and fathers: (1) Emotional Abuse; (2) Physical Abuse; and (3) Severe Abuse. The items were measured on a four-point scale (1= *often*; 4= *never*) and reverse-scored prior to aggregation. The part-whole correlations displayed on Table 1 revealed a satisfactory degree of convergent validity between the measures.

Slow Life History. The slow life history (K-Factor) construct was composed of aggregates of items selected from the MIDUS survey assessing several facets of a life history strategy. Each scale was constructed using items from subscales measuring various cognitive and

behavioral dimensions of life history strategy. The theoretical justifications for the construction of each of these scales using MIDUS data were 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 behavior-genetic models by Figueredo and Rushton (2009).

To avoid the confounding of parental relationship quality content with that of the parental behavior predictors used in the present models, the Parental Investment Scale and the Family Support Scale were omitted from the current life history measurement models. This methodological precaution was taken to avoid inflating these correlations by circumventing the so-called jingle-jangle fallacy (see Pedhazur & Pedhazur-Schmelkin, 1991). In addition, to avoid deleting the records of romantically uncommitted individuals due to substantively inapplicable and therefore missing data, the Partner Attachment Scale was also omitted from the current life history measurement models. Thus, only the following subset of items and scales were used in the analyses reported in the present paper to estimate the slow life history factor (K):

1. The Self Scale was composed of MIDUS subscales assessing Insight, Persistence, Positive Reappraisals, Self-Directedness, Agency, and Financial Status;
2. The Friends Support Scale was constructed from the MIDUS Friends Support Subscale;
3. The General Social Altruism Scale was composed of MIDUS Subscales assessing Close Relationships, Children Relationship Quality, and Communitarian Beliefs;
4. The Religiosity Scale was constructed from the MIDUS Religiosity Subscale.

To show comparability in measuring the latent construct of interest, we correlated this restricted life history factor to the inclusive life history factor previously published, including the three presently omitted scales, as published in Figueredo, et al. (2013). The correlation was high and statistically significant (r= .901, p < .001), indicating a high degree of convergence between the restricted and the inclusive life history factor. There is the possibility that neither this restricted slow LH (K) factor nor the original inclusive one, as estimated from the *MIDUS* data, constitutes a comprehensive assessment of life history strategy, but constitutes an aggregate of some but not all of the relevant facets (Richardson, Sanning, Lai, Copping, Hardesty, & Kruger, 2017). Nevertheless, the restricted life history factor we constructed by omitting certain subscales was virtually equivalent to the inclusive one previously published.

The part-whole correlations displayed on Table 1 revealed a satisfactory degree of convergent validity between the measures.

Table 1
Unit-Weighted Factor Loadings (*Part-Whole Correlations) of the Parental Instability, Parental Abuse, and Slow Life History Scales*

Parental Instability	
Parents' Drinking Caused Problems	.730*
Parent's Drug Use Caused Problems	.701*
Number of Moves to New Neighborhood	.466*
Parents Separated or Divorced	.595*
Parental Abuse	
Parental Emotional Abuse	.874*
Parental Physical Abuse	.893*
Parental Severe Abuse	.839*
Slow Life History (K)	
Self	.680*
Friend Support	.505*
General Social Altruism	.651*
Religiosity	.488*

*$p < .05$

Statistical Analyses

All univariate and multivariate analyses were performed using SAS 9.4 and MPLUS 8. Using SAS PROC STANDARD and DATA, unit-weighted common factor scales (Gorsuch, 1983) were estimated as the means of the standardized scores for all non-missing subscales on each factor (Figueredo, McKnight, McKnight, & Sidani, 2000). Using SAS PROC CORR, we also computed the part-whole correlations of the subscales with the unit-weighted factor scales. All the unit-weighted factor scales estimated were entered as manifest variables for causal analysis. Structural modeling was done using SAS PROC CALIS for Study 1 and MPLUS for Study 2. Prior to analyses, missing data were imputed using PROC MI (imputations= 25). This was done separately for the data of each twin, to avoid inflating cross-twin correlations by imputing data on one twin from data on another. Subsequently, the data were merged into a single data file for the remaining analyses using PROC MEANS.

All structural equation models were evaluated using the following fit indices: χ^2 (chi-square); CFI (the Bentler-Bonnett Comparative Fit Index); and RMSEA (the Root Mean Square Error of Approximation). Chi-square measures the statistical goodness-of-fit of the observed covariance matrix to the expected covariance matrix reproduced by the model. A statistically significant chi-square is therefore grounds for rejection of the model specified, and a nonsignificant chi-square is grounds for its tentative acceptance (but see also Gorsuch & Lehmann, 2017; Meehl, 1978). The CFI and RMSEA are measures of practical goodness-of-fit for large sample

sizes. With such large samples, a small effect will result in a statistically significant lack of fit. However, with such large samples, the CFI values should be greater than .90 to be considered satisfactory levels of practical goodness-of-fit (>.95 for a "close" fit), and the RMSEA values should be lesser than .10 to be considered satisfactory levels of practical goodness-of-fit (<.05 for a close fit), even if statistically significant chi-square values are obtained (Bentler & Bonnett, 1980; Browne & Cudeck, 1993; Hu & Bentler, 1999).

Results

Study 1

Method

In Study 1, we estimated the genetic variance-covariance matrix and then conducted various analyses by means of structural equation modeling.

Results

The first approach consisted of the following steps:

Step 1. We used the MIDUS Twin Sample (N_{mz}= 352, N_{dz}= 327) to first estimate a univariate-bivariate heritability matrix by applying the Falconer (1989) method, representing the genetic variance-covariance matrix among all manifest variables (Table 2) to be used in the models that follow.

Table 2
Bivariate Heritabilities (Genetic Covariances)

	1	2	3	4	5
1.Parental Instability	.394				
2. Parental Emotional Abuse	.156	.136			
3. Parental Physical Abuse	.156	.115	.257		
4.Parental Severe Abuse	.053	.099	.203	.383	
5. Slow Life History Strategy (K)	-.051	-.271	-.209	-.195	.385

*Step 2***.** To create the MIDUS behavior-genetic (BG) factor model, we subjected that matrix to principal components analysis (PCA), retaining the first unrotated principal component based on the Kaiser criterion (eigenvalue > 1.0) representing the set of shared genes influencing all five manifest indicators in common (Table 3). The retention of this single common factor was also strongly supported by the scree plot, and the first principal component accounted for 66% of the genetic covariance among

the five manifest indicators. Although it is unlikely that this common factor explained the separate univariate heritabilities of each of the individual traits, it nonetheless did a very good job of explaining the manifold of genetic correlations among them.

Table 3
Unstandardized Factor Loadings (Factor Structure)

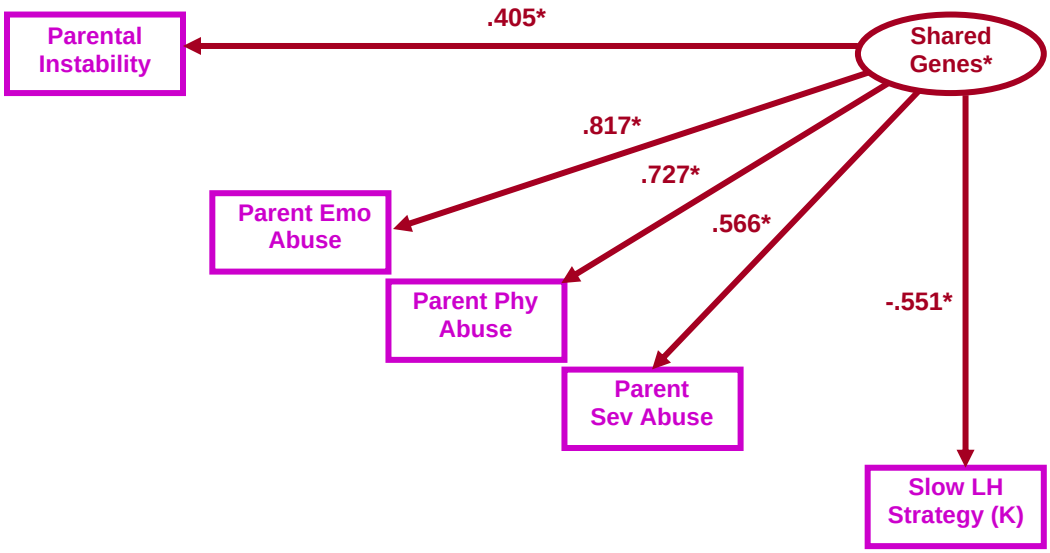
	Shared Genes Common Factor
Parental Instability	.231
Parental Emotional Abuse	.408
Parental Physical Abuse	.408
Parental Severe Abuse	.355
Slow Life History Strategy (K)	-.540

Step 3. We then used the MIDUS singleton sample (*N*= 4243) to impose a confirmatory factor model specified as equivalent to the PCA run on the MIDUS twin sample in Step 2, with model parameters fixed as equal to the unstandardized PCA factor loadings from the unitary genetic common factor. The model was rejectable by all indices assessed: χ^2 (9,4243)= 2399.33, p< .0001, CFI= .565, RMSEA= .250. This indicated that the observed phenotypic covariances could not be accounted for by the influence of shared genes alone.

Step 4. We then used the MIDUS singleton sample to test an alternative pure environmental pathways model for comparing and contrasting to the genetic common factor model constructed in Step 3. This model was rejectable by the strict chi-squared criterion, but acceptable by the practical and parsimonious indices of fit: χ^2 (4,4243)= 42.53, p< .0001, CFI= .993, RMSEA= .048. This indicated that the observed covariances could be accounted for by the influence of environmental pathways alone. Nevertheless, the three main environmental pathways, from parental instability to both parental abuse and offspring slow life history strategy as quite small in magnitude, and the pathway from parental abuse to offspring slow life history strategy is smaller still. Furthermore, the structural model suffers from uncontrolled behavior-genetic confounds.

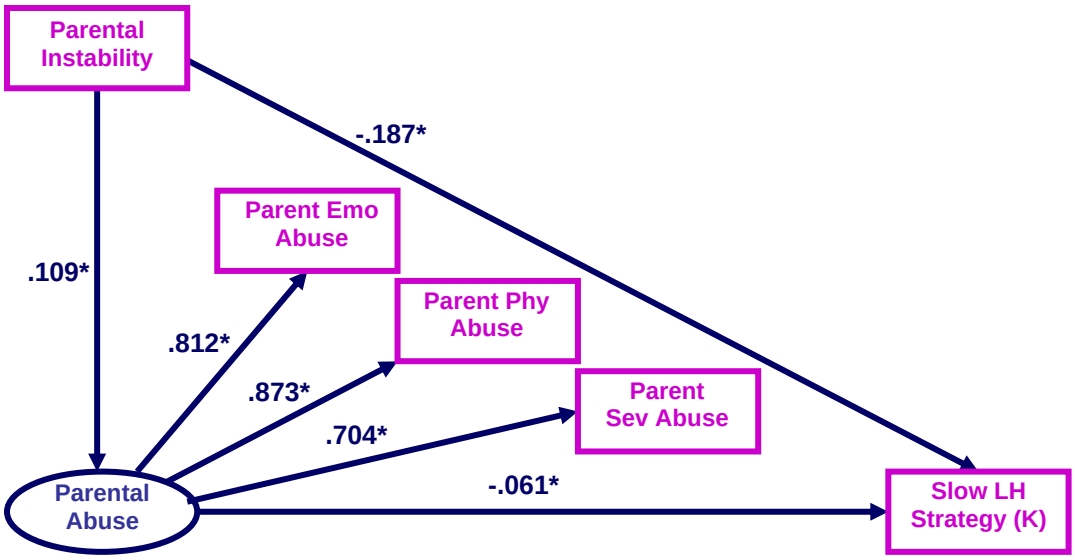
Step 5. We then used the MIDUS singleton sample to specify a more inclusive hybrid structural equations model containing the fixed pathways of the genetic common model and the freely estimated ones of the environmental pathways model. This hybrid model was constructed using the procedures detailed in Figueredo, et al (2014), by specifying fixed parameters with values obtained from the genetically-informative twin sample within a structural equations model with all other parameters

Figure 1. Genetic Common Factor Model with Standardized Parameter Estimates.



* $p<.05$

Figure 2. Environmental Pathways Model.



* $p<.05$

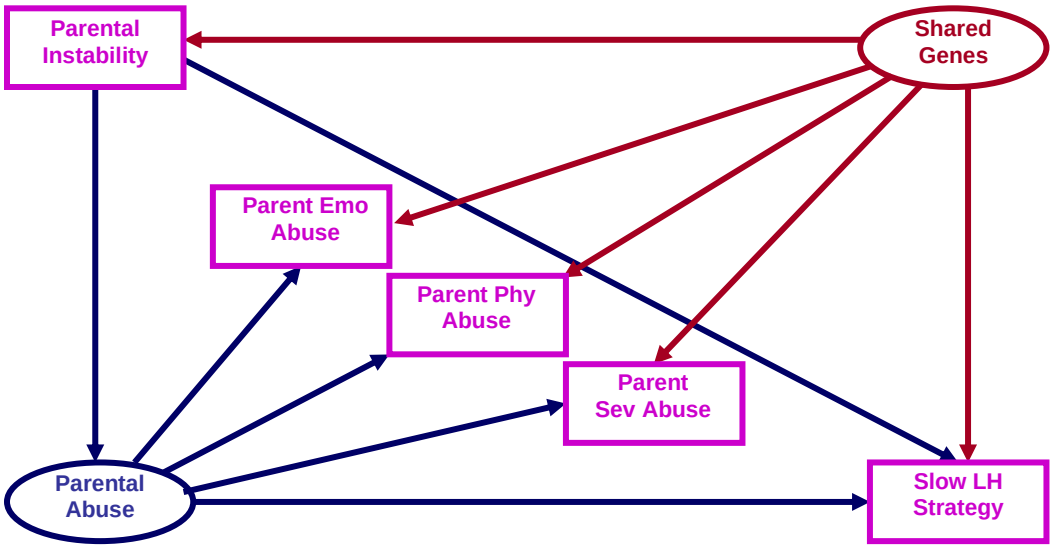
estimated from the non-genetically-informative singleton sample. Thus, we developed hybrid models of the MIDUS singleton (non-twin) data by

setting fixed model parameters based on behavior-genetic estimates from MIDUS twin data.

This procedure was theoretically justified given that MIDUS twin and singleton (non-twin) data are from two national random digit dialing samples drawn from the same USA adult population, meaning that they should reflect the same general population parameters (including their 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. Although some researchers have suggested that perhaps twins might not be representative of singletons, Barnes and Boutwell (2013) found little evidence of differences between twins and singletons. Schwabe, Janss, & Van Den Berg (2017) conducted a comparison of twins and a sample of the entire Dutch population (n = 893,127) and found that twin-based estimates were not an artifact of self-selection or due to differences between twins and singletons. Moreover, as a part of a separate investigation into early rearing influences on alcohol use disorder, the third author conducted an analysis comparing the MIDUS twins and singletons in terms of demographics (e.g., socioeconomic status), early environment (e.g., routines, adverse experiences), and alcohol use in adulthood. Out of the more than 30 tests (α = .05), only three were statistically significant (see Appendix A, Table A1). Furthermore, in these three cases the differences between the groups were trivial in magnitude. The MIDUS twins therefore appear to be generally representative of the MIDUS singletons.

Step 6. The inclusive hybrid gene-environment (G-E) developmental structural equations model created in Step 5 was acceptable in terms of the practical and parsimonious indices of fit, although it was still rejectable by the stricter chi-squared criterion: χ^2 (3,4243)= 25.99, p <.0001, CFI= .996, RMSEA= .043. Nevertheless, some of the environmental pathways in this hybrid model turned out to be statistically nonsignificant. We therefore specified and estimated a restricted hybrid gene-environment (G-E) developmental structural equations model that eliminated (fixed at zero) some of the causal pathways while retaining all of the fixed pathways of the genetic common factor model. This restricted hybrid gene-environment (G-E) developmental structural equations model was also rejectable by the strict chi-squared criterion, but acceptable the practical and parsimonious indices of fit: χ^2 (5,4243)= 41.38, p <.0001, CFI= .993, RMSEA= .041.

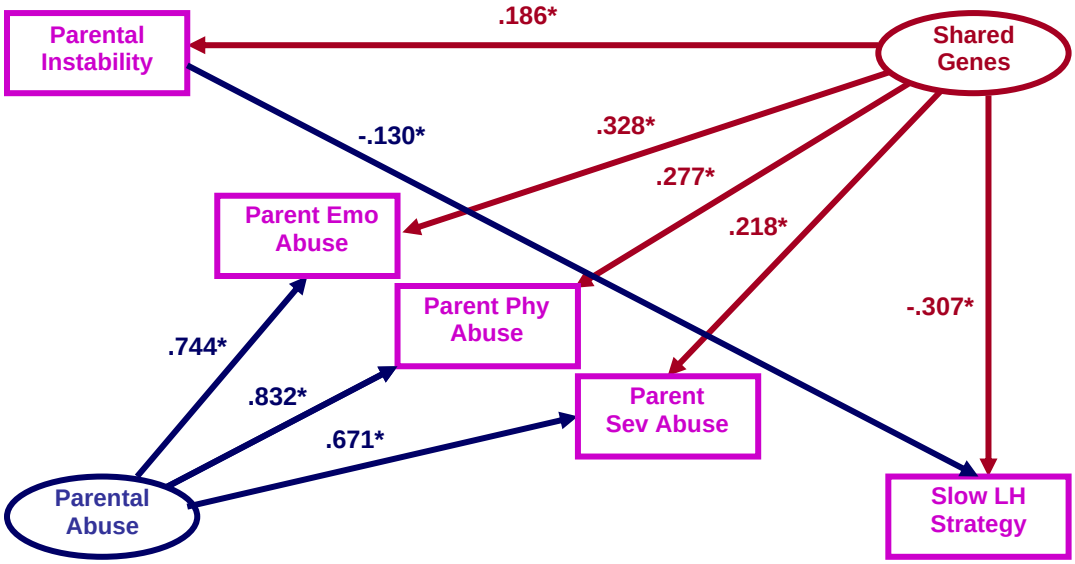
Figure 3. Inclusive Hybrid G-E Developmental Structural Equations Model (Schematic).



* $p<.05$

To discriminate between the inclusive and restricted hybrid models, we therefore conducted a nested model comparison and the differences in the main fit indices were as follows: $\Delta\chi^2$ (2,4243)= 15.39, p = .0005, ΔCFI= -.003, ΔRMSEA= -.002. This means that although the chi-squared difference was statistically significant, due to the huge sample size (N= 4243), the differences in the practical and parsimonious indices of fit were negligible in magnitude. We therefore tentatively accepted restricted hybrid model based on the principle of parsimony. These results are shown in Figure 4.

Figure 4. Restricted Hybrid G-E Developmental Structural Equations Model.



* p<.05

Discussion of Results for Study 1

The genetic common factor model with fixed parameters, imported from MIDUS BG factor model based on the MIDUS Twin Sample data, was found rejectable by all pertinent statistical and practical criteria: (1) the observed covariances among variables was not adequately explained by the single BG factor alone; and (2) the unitary BG factor nevertheless accounted for statistically significant component of variance in the MIDUS singleton data.

Both the environmental pathways model and the hybrid G-E developmental SEM show excellent practical indices of fit: (1) despite statistically significant χ^2 values due to huge sample size (N= 4243); and (2) despite the imposition of multiple fixed model parameters imported from MIDUS BG factor model. Furthermore, the restricted hybrid G-E developmental SEM fits nearly as well as environmental pathways model and was more parsimonious by one degree of freedom.

Two of the environmental pathways model structural parameters leading to the slow LH factor were no longer required by the hybrid model. These two structural relations were instead modeled as spurious due to the common causal influence of shared genes factor. In total, two out of three environmental pathways in the structural model could therefore be eliminated without appreciably compromising model fit.

The three environmental factor loadings of Par Abuse factor remain, as the observed covariances among indicators of Par Abuse were not completely explained by the fixed factor loadings of the shared genes common factor. Thus, the behavior-genetic critiques (e.g., Rowe, 1994) of genetically-uninformed research in developmental psychology appear to be valid, in that at least some developmental pathways are entirely explainable by shared genetic influence. Furthermore, these include some pathways commonly identified in psychosocial research on life history development.

Study 2

Method

In Study 2, we attempted to reproduce the results of Study 1 using a DeFries-Fulker (DF) model-fitting approach. The DF model (DeFries & Fulker, 1985; 1988) was originally developed as part of a regression-based approach for data from selected samples (e.g., probands selected on an outcome such as low reading performance). The model was later extended to other contexts and Rodgers and McGue (1994) showed that it yields unbiased estimates of additive genetic (the A component in classical twin models) and shared environmental (the C component in classical twin models) effects in unselected samples. Researchers in behavioral genetics continue to use the DF model (e.g., Christopher et al., 2016) and recent studies have also applied it fields such as criminal justice (Barnes & and Boutwell, 2013; Nedelec & Beaver, 2014; Teneyck & Barnes, 2015).

The DF model is typically given as:

$$Y_1 = b_0 + b_1 Y_2 + b_2 R + b_3 (Y_2 * R) + e \quad (1)$$

where Y_1 is the target twin's score on an outcome of interest, Y_2 is the co-twin's score on the outcome, and R is the coefficient of genetic relatedness among the twins (MZ twins= 1.00 and DZ twins= .50). b_2 provides an initial test for genetic influence in the context of selected samples[1]. In unselected samples, b_2 is often statistically non-significant and not interpreted because there is no expectation of average target twin differences on the outcome as a function of genetic relatedness (Smith & Hatemi, 2013). In both selected and unselected samples, b_1 and b_3 provide unbiased estimates of shared environmental and addictive genetic influence, respectively (i.e., A and C; Rodgers & McGue, 1994).

[1]In studies of selected samples, all target twins are selected on the basis of their deviant scores on the outcome relative to the population mean and therefore all have deviant scores. The current study examines an unselected sample—no twins were selected on the basis of deviant scores.

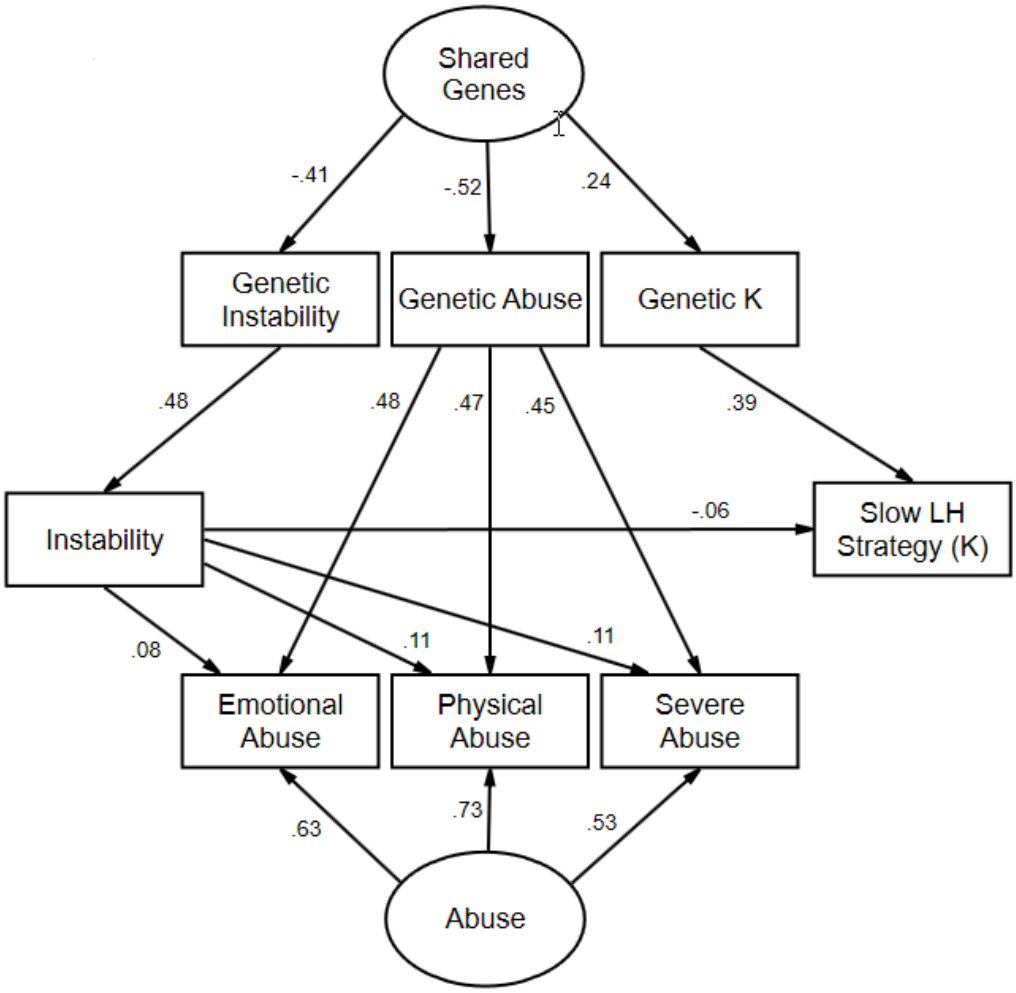
Results

In Study 2, we integrated the DF model into the structural equation modeling (SEM) framework to examine whether the results described in Study 1 could be reproduced using an alternative approach.

Step 1. First, we constructed the same unit-weighted factor scale composites for the three convergent parental abuse indicators, as used in Study 1. We carried out this procedure because we wished to model the three abuse indicators as reflecting the additive genetic variance they shared, rather than specifying a term to capture the genetic variance in each type of abuse.

Step 2. Next, we specified a hybrid DF developmental SEM (see Figure 5) in which K-factor scores, parental instability, and the parental abuse indicators had their own DF equations. As in Study 1, this hybrid model was constructed using the procedures detailed in Figueredo, et al. (2014). This model allowed us to estimate the direct and indirect effects of early environment on slow life history (K) while controlling for potentially confounding genetic factors, as in the previously tested inclusive hybrid gene environment developmental structural equations model (see Figure 3). Because the objective in this study was to control common genetic factors that might confound effects of early environment on K, we omitted Y_2 (i.e., the term capturing C) from each equation and did not distinguish shared and non-shared variance. As mentioned in Step 1, all three parental abuse indicators were regressed on one genetic variable (co-twin component abuse scores * zygosity). We specified a group-specific factor that subsumed the covariance among these indicators not accounted for by genetic factors (i.e., the environmental variance they shared). We specified a common genetic factor that subsumed $Y_2 * R$ in each equation (i.e., the term capturing A). As in Study 1, this factor subsumed common genetic liability to the indicators of the three phenotypes. Finally, the cascade of effects specified in the previously tested environmental pathways model was included. Each type of abuse was regressed on instability, rather than their common factor, so we could examine whether instability had differential effects on the different types of abuse. We tested this model and fit to the data was excellent (χ^2 (10) = 15.52, p= .11; CFI= 1.00; RMSEA= .02).

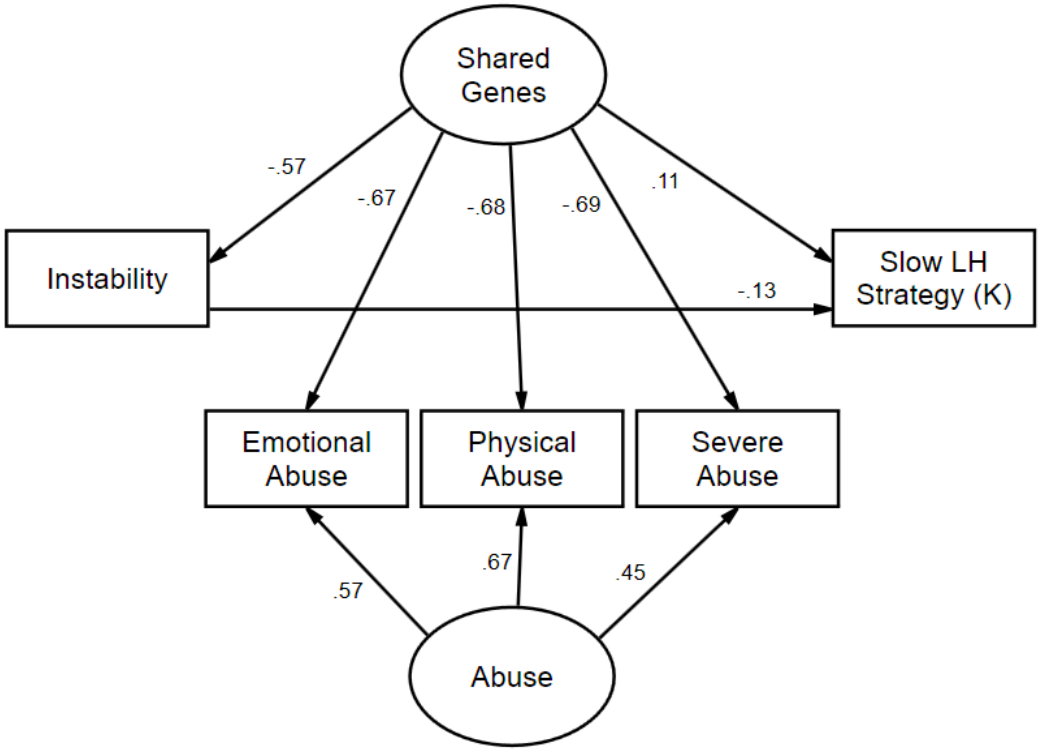
Figure 5. Hybrid DF Developmental SEM — Twins.



Note: Only statistically significant standardized path coefficients ($p < .05$) effects displayed.

Step 3. Using the model tested in Step 2, we estimated the indirect effects of the common genetic factor on each of the variables in model. Using the MIDUS singleton data, we then tested a model in which these estimates served as common genetic factor loadings. That is, a common genetic factor was specified and its loadings were fixed to the previously estimated indirect effects. Model fit was excellent (χ^2 (2)= 1.49, p= .49; CFI= 1.00; RMSEA= .02).

Figure 6. *Hybrid Developmental SEM — Singletons*



Discussion of Results for Study 2

Using Study 2, we were able to reproduce our initial findings, suggesting they are robust to the analytic approach employed. Once the genetic factors common to parental instability, parental abuse, and K are controlled; effects of parental instability on abuse and of parental abuse on K become statistically non-significant. Only the effect of parental instability on K remained statistically significant. This suggests that the first two effects are spurious while the third is robust to genetic controls. It should be noted, however, that the shared genes factor still exerted an indirect effect on K through parental instability.

Summary and Concluding Discussion

Evolutionary models have provided important theoretical and empirical contributions to our understanding of human development. One methodological limitation in this literature, however, is that most research designs have been non-genetically-informative—they have not accounted

for potential confounding by common genetic influences that might be producing spurious correlations among the presumed developmental causes and effects. In the current study, we used two convergent methods of heritability estimation (Falconer and DeFries-Fulker) in the context of factor analytic structural equation modeling to address this gap in the literature. Both approaches used the MIDUS twin sample to produce estimates of the purely genetic contributions of the influence of certain parental patterns of behavior on early developmental environments on the adult life history outcomes of the offspring. These estimates were then were used to determine what environmentally-mediated effects remained statistically significant in the MIDUS twin and singleton samples once such genetic common factors were controlled.

The two genetically informative approaches yielded nearly identical results, indicating our estimates are robust to the analytic approach employed. In both cases, once the genetic factors common to parental instability, parental abuse, and slow life history (K) are controlled, the effects of parental instability on abuse and of parental abuse on slow life history (K) could be eliminated without appreciable loss of model fit. Only the effect of parental instability on slow life history (K) needed to be retained and remained statistically significant. This suggests that the first two effects are spurious while the third is robust to genetic controls. These findings extend upon earlier studies suggesting that genetic confounding may be an important concern for developmental research (Barbaro, Boutwell, Barnes, & Shackelford, 2017), highlight the importance of genetically informative designs in studies of human life history development, and provide case illustrations of two computationally tractable approaches to addressing potentially confounding genetic factors.

Limitations of the Studies. Several limitations should be kept in mind, however, when interpreting our results. It remains possible that specific risk factors in the non-shared environment, or non-transmitted alleles, could play a key role in explaining the development of life history strategy. We did not examine those factors in the current study, but they could be included in models like ours in future research. For example, the concept of gene swarm (Hertler et al., 2018) has been put forth to explain the construction and maintenance of the child's immediate environment. According to Hertler and colleagues (2018), a gene swarm is generated by the high degree of genetic relatedness among close kin conspecifics that is in constant transaction with the immediate environment, which in turn influences a child's development. Family environments that have a denser gene swarm (i.e., more individuals genetically related to the child) should have more influence on a child's development. For instance, this condition can obtain where family groups are either larger or closer or both, as with slow life history strategists (Figueredo et al., 2006).

In addition, the developmental indicators taken from the MIDUS survey were based entirely on retrospective self-report. Such data may contain biases from either of two sources: the retrospective part and the self-report part. Retrospective data may suffer from simple inaccuracy of recall, especially of early childhood events; they may also suffer from memory reconstruction in which undesirable life outcomes may bias respondents towards negative interpretations of the past. Self-report data may suffer from self-presentation bias towards socially desirable responding.

Nevertheless, there are several substantial strengths of the present study that serve to advance the discourse in the relevant developmental literature, and these include the use of nationally representative samples, genetically informative designs, and the convergence of two modeling approaches.

Author notes: Corresponding Author: Aurelio José Figueredo, PhD, Department of Psychology, 1503 East University Boulevard, School of Mind, Brain, and Behavior, College of Science, University of Arizona, Tucson, AZ USA 85721-0068; Email: ajf@email.arizona.edu

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Appendix A

Table A1
Comparison of Singletons and Twins

Variable	χ^2 or *t*	*p*	SMD	*F*	*p*
# times moved to new neighborhood	1.178[t]	.239		.134	.715
Family on welfare	.074	.786			
Ever homeless	.252	.616			
Parents separated/divorced	.187	.665			
Consistent rules – Mother	.417[t]	.676		.197	.692
Consistent rules – Father	2.972[t]	.003*	-.05	1.776	.183
Emotional abuse – Mother	.830[t]	.407		.399	.528
Emotional abuse – Father	.494[t]	.622		.304	.581
Physical abuse – Mother	-.937[t]	.349		.399	.046*
Physical abuse – Father	-.404[t]	.686		1.753	.185
Severe physical abuse – Mother	1.352[t]	.176		.053	.818
Severe physical abuse – Father	1.266[t]	.206		1.281	.258
Father education	-.145[t]	.885		.298	.585
Mother education	.597[t]	.551		1.002	.317
Perceived financial level growing up	-2.374[t]	.018*	.039	.190	.663
SES Index – Father	.236[t]	.813		.123	.726
SES Index – Mother	-.023[t]	.982		.068	.795
Had at least one drink (past mo.)	.297	.586			
How often at least one drink (past mo.)	3.429	.634			
How many days per month	4.996	.172			
# Drinks on drinking days	10.944	.362			
Times had 5+ drinks on same occasion (past mo.)	18.686	.347			
Emotional problems from drinking (12 mo.)	.526	.468			
1+ month much time drinking (12 mo.)	.039	.844			
Had to drink more to get effects (12 mo.)	.261	.610			
Alcohol problem (12 mo.)	.018	.894			
# times alcohol more than intended (12 mo.)	2.656	.753			
# times alcohol effects at work (12 mo.)	5.186	.394			

** p< .05*
Notes: *t* = t-statistic; SMD = Standardized mean difference; F-statistics used to test equality of the variance. SES = Socioeconomic status; mo. = month. SMD omitted if test was statistically non-significant.