Evolution and Human Behavior xxx (2016) xxx–xxx

Contents lists available at ScienceDirect

Evolution and Human Behavior

journal homepage: www.ehbonline.org

Original Article

# Genetic confounding of the relationship between father absence and age at menarche

Nicole Barbaro [a,*], Brian B. Boutwell [b,c], J.C. Barnes [d], Todd K. Shackelford [a]

[a] Oakland University, Department of Psychology, 112 Pryale Hall, Rochester, MI, 48309
[b] Saint Louis University, Criminology and Criminal Justice, School of Social Work, St Louis, MO, 63103
[c] Saint Louis University, Department of Epidemiology, St Louis, MO, 63103
[d] University of Cincinnati, School of Criminal Justice, Cincinnati, OH, 45221

ARTICLE INFO

*Article history:*
Initial receipt 26 February 2016
6 November 2016
Final revision received 28 November 2016
Available online xxxx

*Keywords:*
Life history theory
Age at menarche
Father absence
Behavioral genetics
Simulation

ABSTRACT

Research in evolutionary psychology, and life history theory in particular, has yielded important insights into the developmental processes that underpin variation in growth, psychological functioning, and behavioral outcomes across individuals. Yet, there are methodological concerns that limit the ability to draw causal inferences about human development and psychological functioning within a life history framework. The current study used a simulation-based modeling approach to estimate the degree of genetic confounding in tests of a well-researched life history hypothesis: that father absence (*X*) is associated with earlier age at menarche (*Y*). The results demonstrate that the genetic correlation between *X* and *Y* can confound the phenotypic association between the two variables, even if the genetic correlation is small—suggesting that failure to control for the genetic correlation between *X* and *Y* could produce a spurious phenotypic correlation. We discuss the implications of these results for research on human life history, and highlight the utility of incorporating genetically sensitive tests into future life history research.



## 1. Introduction

Researchers in the behavioral sciences have made theoretical and empirical advances by leveraging life history theory to better understand human psychology and behavior (e.g., Brumbach, Figueredo, & Ellis, 2009; Chisholm, 1993, 1999; Del Giudice, 2009; Figueredo et al., 2006). Within this framework, life history theorists argue that aspects of the familial environment during childhood are associated with conditional adjustments of an individual's life history strategy. In particular, research has advanced the hypothesis that father absence in childhood is associated with earlier age at menarche (Belsky, Steinberg, & Draper, 1991; Ellis, 2004; Ellis, Figueredo, Brumbach, & Schlomer, 2009; Mul, Oostdijk, & Drop, 2002; Parent et al., 2003). In the current article, we propose an alternative hypothesis: that the relationship between father absence and age at menarche is confounded by the genetic correlation between the two phenotypic traits. We suggest that the association between father absence and age at menarche may not reflect a causal association, but instead could result from a shared genetic variation. We provide support for this argument by employing simulation-based modeling to demonstrate the degree to which the phenotypic correlation ($r_p$) between father absence (*X*) and age at menarche (*Y*) might be confounded by genetic variation. We do so by drawing on estimates of $r_p$ and on heritability coefficients for both *X* and *Y* that have been previously published.

### 1.1. Human life history

Life history theory provides a framework for addressing how and why organisms, including humans, allocate resources to conflicting life tasks (Kaplan & Gangestad, 2005; Roff, 2002; Stearns, 1992). Allocating resources to these conflicting life tasks involves tradeoffs, because the available resources are finite and limited. Resources must be strategically allocated so that the organism can grow, maintain homeostasis, and reproduce. Resource allocation "decisions" are made throughout the lifespan and are reflected in an individual's behaviors (Kenrick, Griskevicius, Neuberg, & Schaller, 2010; Simpson, Griskevicius, & Kim, 2011). Allocation strategies that optimized the use of resources throughout the lifespan were selected over human evolutionary history (Ellis et al., 2009). Ancestrally adaptive resource allocation strategies vary with species-typical evolutionary history, individual differences, and local ecology. Life history theorists argue that the solutions to life history tradeoffs arise via combination of genetic variation and environmental inputs experienced by the organism (Ellis et al., 2009), whereby psychological and physiological mechanisms "decide" how to allocate resources that enhanced ancestral survival and reproduction (Chisholm, 1999).

* Corresponding author.
*E-mail addresses:* nmbarbar@oakland.edu (N. Barbaro), boutwellb@slu.edu (B.B. Boutwell), jc.barnes@uc.edu (J.C. Barnes), shackelf@oakland.edu (T.K. Shackelford).

http://dx.doi.org/10.1016/j.evolhumbehav.2016.11.007
1090-5138/

Natural selection has produced a species-typical life history strategy in humans characterized by lengthy pregnancy, extended childhood, high parental investment, and low fecundity (Rushton, 2000). The question then becomes: what causes individual variation in resource allocation decisions and in the speed of life history strategies? Within-species variation in life history strategies is recognized across human populations and across diverse animal taxa (Gross, 1996; Promislow & Harvey, 1990; Roff, 2002; Stearns, 1992; West-Eberhard, 2003). Individual differences in life history strategies are facilitated by psychological and physiological mechanisms, underpinned by genes that are responsive to the local ecology and that afford conditional adjustments within the constraints of the species-typical life history strategy (Ellis et al., 2009).

Applications of life history theory to developmental psychology, in particular, center on the role of childhood environments calibrating life history traits that manifest across an individual's lifespan. Belsky et al. (1991) argue that aspects of the family environment in early childhood influence the reproductive strategies of females, in particular. Certain indicators of familial functioning, such as father absence, are hypothesized to result in earlier pubertal maturation, and specifically an earlier age at menarche in girls. Earlier age at menarche is thought to be indicative of a faster life history strategy in which greater resources are allocated to mating effort via the ability to begin reproduction at an earlier age (Belsky et al., 1991; Ellis, 2004). However, there remain two perspectives regarding the determinants of menarche timing: (1) Selection has favored psychological mechanisms that are sensitive to, for example, specific familial environmental inputs, which afford conditional adjustments of pubertal maturation and life history strategies, or (2) genetic variation accounts for individual differences in pubertal maturation and life history strategies (i.e., the life history of an individual's genetic relatives is more indicative of the individual's life history strategy).

Age at menarche is a well-investigated life history trait (Ellis, 2004; Ellis, McFadyen-Ketchum, Dodge, Pettit, & Bates, 1999; Mul et al., 2002; Parent et al., 2003), with the prevailing hypothesis being that father absence – an aspect of the familial environment – results in earlier age at menarche, thereby facilitating the execution of a faster life history strategy. In accordance with the first perspective above, age at menarche is more strongly determined by environmental risk during development, such that earlier age at menarche may facilitate earlier reproduction and greater mating effort. In accordance with the second perspective above, however, age at menarche is heritable (Dick, Rose, Pulkkinen, & Kaprio, 2001; Kirk et al., 2001; Rowe, 2002), suggesting that individual variation in age at menarche may be more strongly influenced by one's genes, independent of father absence, specifically.

The degree to which environmental conditions or genetic variation is influential in determining menarche timing remains a topic of debate among life history theorists. If the second perspective is accurate – that is, genetic influences account for individual variation in age at menarche, independent of the early familial environment – it would not diminish the importance of the environment. Empirical evidence in accordance with the second perspective would, however, (1) necessitate research designs capable of holding genetic variation constant so that environmental influences on pubertal maturation in girls can be accurately investigated (see generally Barnes, Boutwell, Beaver, Gibson, & Wright, 2014), and (2) suggest that for age at menarche, specifically, father absence may not be as influential as previously suggested (see Ellis, 2004).

### 1.2. Genetic influences on pubertal maturation in girls

Although discussion and investigation of genetic factors contributing to individual variation in life history strategies are limited, researchers have called for an integration of life history theory and behavioral genetic methodologies (Ellis et al., 2009; Figueredo, Vásquez, Brumbach, & Schneider, 2004; Harkness, 2014; Hofmann, 2003; MacDonald, 1997; Scarr, 1995). Behavioral genetics provides insight into the genetic and environmental influences on human traits, which affords estimates of the phenotypic variation explained by genetic factors and environmental factors (Plomin, DeFries, Knopik, & Neiderhiser, 2013). Phenotypic variance is composed of three factors: heritability ($h^2$), shared environment ($c^2$), and nonshared environment ($e^2$). The $h^2$ component refers to phenotypic variance accounted for by variance in genes.[1] The environmental components ($c^2$ and $e^2$) refer to phenotypic variance accounted for by environmental experiences shared with others (i.e., aspects of the environment that make people similar to one another, $c^2$) and environmental experiences not shared with others (i.e., aspects of the environment that make people dissimilar, $e^2$).

A growing number of studies have employed genetically sensitive research designs to address issues of genetic confounding when testing life history hypotheses (Dick et al., 2001; D'Onofrio et al., 2006; Ellis, Schlomer, Tilley, & Butler, 2012; Kirk et al., 2001; Mendle et al., 2006, 2009; Rowe, 2002; Tither & Ellis, 2008). The results have revealed that various traits directly related to life history strategies are moderately to highly heritable (e.g., age at menarche; Dick et al., 2001; Kirk et al., 2001), as are indirect measures of life history strategies (e.g., mother relationship quality; Figueredo et al., 2004). These findings present life history researchers with an important issue that requires consideration in future research.

Specifically, to the extent that genetic factors exert influence on population variance in life history strategies, ignoring genetic influences may bias research aimed at examining the environmental influences on human life history. Put differently, genetic variance underpinning phenotypic traits may be conflated with environmental variance when estimating the relationship between two phenotypes if that genetic variance is not held constant. Moreover, environmental experiences hypothesized to influence the manifestation of life history strategies do not occur at random (Scarr, 1995). Selection effects, underpinned by genes, exert an influence on the environments that humans experience via gene–environment correlation (*r*GE) (Barnes et al., 2014; Kendler & Baker, 2007; Scarr & McCartney, 1983). Individuals seek out environments and experiences that correspond to their genotype, making it possible that environmental experiences are heritable due to an indirect effect of the genes on the environment that is mediated by individual decision making.[2] Failing to account for the influence of genetic variation on environmental conditions may impede the ability to draw accurate conclusions about which aspects of the environment *are* influential to individual variation in age at menarche. Because the influence of environmental factors on adolescent and adult sexual psychological and behavioral outcomes is a linchpin of life history theory, appropriate steps need to be taken to account for potential genetic confounding.

Research utilizing behavioral genetic methodologies has estimated the contribution of genetic variation and environmental inputs on pubertal maturation and reproductive traits, including age at menarche (D'Onofrio et al., 2006; Tither & Ellis, 2008), age at first reproduction (Kirk et al., 2001; Rowe, 2002), pubertal development (Dick et al., 2001), sexual debut (Mendle et al., 2009), and sexual behavior (D'Onofrio et al., 2006; Ellis et al., 2012). The results of these studies (reviewed below) demonstrate significant genetic contributions to the aforementioned life history traits — highlighting the potential for genetic confounding when life history traits are analyzed as outcome variables.

[1] Additive variance ($V_A$) is often indexed by $h^2$, so it is more accurate to describe this as evidence for "narrow-sense" heritability. With that in mind, Hill, Goddard, and Visscher (2008) provided evidence suggesting that additive variance accounts for the majority of genetic variance in many traits. Thus, the focus on additive variance is unlikely to bias $h^2$ estimates for many (if not most) phenotypes.

[2] Imagine a personality trait *P* emerges largely due to genetic factors. To the extent that *P* influences individuals' choices to engage in environmental situations, then those environmental factors will appear to be heritable due to the mediating role of *P* between the genes and the environment.

Please cite this article as: Barbaro, N., et al., Genetic confounding of the relationship between father absence and age at menarche, *Evolution and Human Behavior* (2016), http://dx.doi.org/10.1016/j.evolhumbehav.2016.11.007