Journal of Abnormal Psychology

0021-843X/16/$12.00 http://dx.doi.org/10.1037/abn0000185

Current Evolutionary Adaptiveness of Psychiatric Disorders: Fertility Rates, Parent−Child Relationship Quality, and Psychiatric Disorders Across the Lifespan

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This study sought to evaluate the current evolutionary adaptiveness of psychopathology by examining whether these disorders impact the quantity of offspring or the quality of the parent–child relationship across the life span. Using the National Comorbidity Survey, this study examined whether *DSM–III–R* anxiety, posttraumatic stress, depressive, bipolar, substance use, antisocial, and psychosis disorders predicted later fertility and the quality of parent–child relationships across the life span in a national sample ($N = 8{,}098$). Using latent variable and varying coefficient models, the results suggested that anxiety in males and bipolar pathology in males and females were associated with increased fertility at younger ages. The results suggested almost all other psychopathology was associated with decreased fertility in middle to late adulthood. The results further suggested that all types of psychopathology had negative impacts on the parent–child relationship quality (except for antisocial pathology in males). Nevertheless, for all disorders, the impact of psychopathology on both fertility and the parent–child relationship quality was affected by the age of the participant. The results also showed that anxiety pathology is associated with a high-quantity, low-quality parenting strategy followed by a low-quantity, low-quality parenting strategy. Further, the results suggest that bipolar pathology is associated with an early high-quantity and a continued low-quality parenting strategy. Posttraumatic stress, depression, substance use, antisocial personality, and psychosis pathology are each associated with a low-quantity, low-quality parenting strategy, particularly in mid to late adulthood. These findings suggest that the evolutionary impact of psychopathology depends on the developmental context.

General Scientific Summary
This study suggests that anxiety and bipolar psychopathology both have early positive impacts on fertility. The results further suggest that posttraumatic stress, depression, substance use, antisocial personality, and psychosis pathology were associated with low fertility in later adulthood. Thus, the evolutionary impact of psychopathology may depend on one's age.

Keywords: adaptiveness, evolution, psychiatric, anxiety, depression

Nearly half of the population will have met diagnostic criteria for a psychiatric disorder within their lifetime (Kessler et al., 2005). Anxiety, mood, externalizing, and psychotic disorders are associated with an increased risk of cardiovascular diseases (Osby, Correia, Brandt, Ekbom, & Sparen, 2000; Rehm et al., 2009; Suls & Bunde, 2005), increased risk of suicide (Hor & Taylor, 2010; Sareen et al., 2005; Verona, Sachs-Ericsson, & Joiner, 2004), low productivity (Kessler & Frank, 1997; Wittchen, Carter, Pfister, Montgomery, & Kessler, 2000), and lower quality of life (Black, Gunter, Loveless, Allen, & Sieleni, 2010; Hasson-Ohayon, Kravetz, Roe, David, & Weiser, 2006; Wittchen et al., 2000). Consequently, psychopathology merits investigation because of its broad impact.

Although many theories of psychopathology have emerged, most theories fundamentally involve a partial genetic predisposition (i.e., diathesis-stress models, differential susceptibility; Belsky & Pluess, 2009; Mineka, Watson, & Clark, 1998; Walker & Diforio, 1997). Supporting this view, previous research has broadly corroborated the partial heritability of psychopathology. Specifically, anxiety pathology tends to yield heritability estimates between 20% and 48%: Generalized anxiety disorder is 32% heritable (Hettema, Neale, & Kendler, 2001); panic disorder is 48% heritable (Hettema et al., 2001); social phobia is 28% heritable (Nelson et al., 2000); and specific phobia is 20% to 40% heritable (Hettema et al., 2001). Posttraumatic stress pathology has been found to be 30% to 71% heritable (Sartor et al., 2010; Sartor et al., 2012; Stein, Jang, Taylor, Vernon, & Livesley, 2002; True

The National Comorbidity Survey was funded by the National Institute of Mental Health (Grants R01 MH/DA46376 and R01 MH49098), the National Institute of Drug Abuse (through a supplement to R01 MH/DA46376), and the W. T. Grant Foundation (Grant 90135190). I thank Amanda Jacobson for her helpful review of my manuscript drafts.

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et al., 1993). Depression pathology has been found to be 29% to 71% heritable: Major depression is 29% to 42% heritable (Kendler, Gatz, Gardner, & Pedersen, 2006), and dysthymia is 45% to 46% heritable (Edvardsen et al., 2009). Bipolar pathology has been found to be 59% heritable (Lichtenstein et al., 2009). Substance use pathology has been found to be 49% to 80% heritable: 60% to 80% for most drug use disorders (Kendler, Karkowski, Neale, & Prescott, 2000), and 49% for alcohol use disorders (Rhee & Waldman, 2002). Antisocial personality pathology has been found to be 36% heritable (Rhee & Waldman, 2002). Likewise, psychotic pathology has been found to be 81% to 85% heritable: Schizophrenia is 81% heritable (Sullivan, Kendler, & Neale, 2003), and schizoaffective disorder is 85% heritable (Cardno et al., 1999). Thus, all the primary domains of psychopathology have been found to be partially heritable. These results suggest that a genetic predisposition represents a fundamental aspect of how psychiatric disorders manifest.

When a trait possesses a genetic component, it is possible that it will affect an organism's evolutionary fitness, thereby potentially impacting the prevalence of traits across generations (Raine, 1997). Consequently, evolutionary psychologists posit that all animals, including humans, have abundant psychological functions designed to continue their hereditary line (Marks & Nesse, 1997). Notably, unlike previous interpretations of evolutionary theory which suggest that maladaptive traits are eliminated at a constant gradual rate over hundreds of thousands of successive years (Dawkins, 1986), recent theories (Gould, 1972) and evidence suggest that many changes in evolution are not slow, but rather are fast and dramatic (Swanson & Vacquier, 2002; Yoshida, Jones, Ellner, Fussmann, & Hairston, 2003). Because natural selection can quickly eliminate traits that adversely affect one's ability to reproduce, this calls into question whether psychiatric disorders, as partially hereditable, affect one's ability to reproduce in the current environment (Stearns, Byars, Govindaraju, & Ewbank, 2010).

Indeed, because of their high prevalence in the current society, many authors have conjectured about the evolutionary adaptability of mental disorders (Keller, 2008; Keller & Miller, 2006; Marks & Nesse, 1997; Sloman, 2008). Although authors have speculated about the evolutionary adaptability or maladaptability of mental illnesses, there is a void in research regarding evaluations of these claims (Keller, 2008; Marks & Nesse, 1997). This research is needed as its findings have implications for the prevalence of psychiatric disorders over time.

Theories on evolutionary adaptiveness have largely focused on two major areas: the enhancement of an individual's personal and familial chances of reproduction (Hamilton, 1964; McGuire, Marks, Nesse, & Troisi, 1992). Whereas many authors have tested current specific evolved functions (e.g., if depression causes a person to abandon a hopeless argument earlier than it would have otherwise ended; Sloman, 2008), current broad reproductive success (e.g., if a disorder decreases an individual's or family's number of offspring) has rarely been examined across psychiatric disorders. Because it may have implications in regard to the future prevalence of these disorders, current reproductive success should be examined in a broad context to reveal the actual adaptiveness of each disorder in the present environment (McGuire et al., 1992; Rieseberg, Widmer, Arntz, & Burke, 2002).

Prior research has primarily investigated the broad reproductive adaptiveness of psychiatric disorders by examining the associations between lifetime psychiatric disorders and fertility rates. In particular, considerable evidence has amassed that schizophrenia (Bundy, Stahl, & MacCabe, 2011; Power et al., 2013) and bipolar disorder are associated with decreased fertility (Baron, Risch, & Mendlewicz, 1982; Jonsson, 1991; Power et al., 2013). Depression and substance abuse have also been found to be associated with decreased fertility when collapsed across the life span (Power et al., 2013). Across disorders, most studies have showed that these disorders are particularly associated with decreased fertility in males (Bundy et al., 2011; Power et al., 2013).

Although these patterns of fertility have amassed considerable evidence, the literature has broadly neglected anxiety, posttraumatic stress, alcohol use disorders, and antisocial personality pathology. Specifically, no research has examined the impact of anxiety disorders on fertility rates, except with studies using small samples or no control groups (Calzeroni, Conte, Terzi, Vita, & Sacchetti, 1989; Essock-Vitale & McGuire, 1989). Further, no studies have examined the impact of posttraumatic stress pathology on fertility. Although substance abuse has been examined, no research has examined either alcohol use disorders or antisocial personality disorders. Examining antisocial personality pathology more broadly is important given that criminality, a hallmark of antisocial personality pathology, has recently been found to be associated with increased fertility rates (Yao, Långström, Temrin, & Walum, 2014). Consequently, more research is needed to investigate whether anxiety disorders, posttraumatic stress, alcohol use disorders, and antisocial personality pathology affect fertility rates.

Past examinations of the adaptiveness of psychopathology have uniformly examined the effect of single disorders on fertility rates. Nevertheless, psychiatric disorders are highly comorbid with one another; and consequently, when one does not account for this comorbidity, it may compromise the validity of prior findings. Consequently, greater research is needed to examine the impact of multiple disorders after examining the latent structure of these disorders.

In addition to limitations from the high co-occurrence of disorders, nearly every prior study examining the relationship between fertility and psychiatric disorder has collapsed across the life span. Consequently, few studies have accounted for the directionality between psychiatric disorders and fertility rates, meaning that these studies have been unable to establish whether psychiatric disorders are a cause or consequence of the low birth rates.

Additionally, by collapsing across the life span, these studies have largely been unable to account for the adaptiveness of these disorders across different developmental stages (Stearns et al., 2010). Given that earlier ages hold greater weight in determining the evolutionary adaptiveness of traits than later ages (Baltes, 1997), examining psychiatric disorders across different developmental stages is especially important. Nevertheless, a single study has reported the effects of age on the impact of psychopathology (bipolar) in predicting fertility and found that the impact of bipolar on fertility significantly depended on the age of the participants (Baron et al., 1982).

In the process of examining fertility in psychiatric disorders, past studies have also failed to account for other evolutionary strategies. In particular, another competing evolutionary strategy is to emphasize quality relationships over quantity of direct offspring (Newson, Postmes, Lea, & Webley, 2005). Specifically, the qual-

ity relationships approach emphasizes spending greater amounts of resources on fewer children to increase the likelihood of those children having children (Becker, 1960). As such, considering both the quantity of children and the quality of the relationship with those children is important from an evolutionary viewpoint.

Although past research has neglected to examine the quantity and quality of psychiatric disorders at different stages across the life span, theorists have hypothesized that internalizing (composed of anxiety, posttraumatic stress, depression, and bipolar pathology) and externalizing (composed of alcohol use, substance use, and antisocial personality pathology) problems would have different impacts on both the quantity of children and quality of parent–child relationships across the life span. In particular, prior theorists have suggested that internalizing problems may be associated with earlier mating behaviors for women, and externalizing behaviors are characterized by more frequent sexual experiences for men (Belsky, Steinberg, & Draper, 1991). Further, these theorists suggest that as persons with internalizing and externalizing problems age, they invest fewer resources in their children. This suggests that those with psychiatric disorders in their earlier adulthood would tend to be have higher fertility rates, whereas those with psychiatric disorders in their later adulthood would tend to have lower quality relationships with their offspring as compared with their peers (Belsky et al., 1991).

In the current study, an evaluation was performed to examine the impact of modern day psychopathology on later fertility. This study fills gaps in prior literature by (a) examining latent models of psychiatric disorders including anxiety, posttraumatic stress, depression, bipolar, substance use, antisocial personality, and psychotic disorders to account for high comorbidity patterns in the psychiatric disorders; (b) investigating whether psychopathology temporally precedes and predicts later fertility by utilizing a longitudinal method and controlling for baseline fertility; (c) exploring the impact of psychopathology on fertility across both sex and developmental stages; and (d) examining the impact of psychopathology on the evolutionary adaptiveness of both the quantity of offspring and quality of relationships with those offspring. This study used the National Comorbidity Surveys from 1990–1992 and the National Comorbidity Survey Follow-Up from 2001–2002, including a national survey taken by more than 8,000 adults residing in the United States.

On the basis of theories by Belsky et al. (1991), I hypothesized that sex and age would moderate the impact of psychopathology on fertility and parent–child relationship quality all psychopathology. In particular, on the basis of theories by Belsky et al. (1991) and paired with prior evidence that psychopathology predicts low fertility rates (Baron et al., 1982; Bundy et al., 2011; Jonsson, 1991; Power et al., 2013), I hypothesized that (a) for women but not men, anxiety, posttraumatic stress, depression, and bipolar pathology would predict increased fertility rates during late adolescence and early adulthood; (b) for men but not women, substance use pathology and antisocial personality pathology would predict increased fertility rates during late adolescence and early adulthood; (c) for men and women, all psychopathology (anxiety, posttraumatic stress, depression, bipolar, substance use, antisocial personality pathology, and psychosis pathology) would predict decreased fertility rates during middle and late adulthood; and (d) for men and women, all psychopathology (anxiety, posttraumatic stress, depression, bipolar, substance use, antisocial personality pathology, and psychosis pathology) would predict poor parent–child relationship quality in mid to late adulthood.

Method

Participants

Participants (N = 8,098; 47.3% Male, 75.13% White, 12.48% Black, 9.05% Hispanic, 3.33% other, 15 to 61 years old, average age 33.73) were drawn from the National Comorbidity Survey from 1990–1992, and the participants were re-interviewed approximately 9 to 12 years later (Kessler, 2008).

Measures

***DSM–III–R* codes.** Each participant completed the World Mental Health Survey's Composite International Diagnostic Interview (CIDI). The CIDI field trials suggested excellent interrater reliability (Wittchen et al., 1991), good retest reliability (Wittchen et al., 1990), and good concordance with clinical diagnoses (Janca, Robins, Cottler, & Early, 1992). The *DSM–III–R*/CIDI criteria included a lifetime diagnoses of (a) anxiety pathology, including social phobia, panic disorder, specific phobia, agoraphobia, and generalized anxiety disorder; (b) posttraumatic stress disorder; (c) depression pathology, including major depression and dysthymia; (d) bipolar disorder; (e) substance use pathology, including alcohol use disorder, including both alcohol abuse and alcohol dependence, and drug use disorder, including both drug abuse and drug dependence; (f) antisocial personality disorder; and (g) psychosis, including schizophrenia, schizophreniform disorder, schizoaffective disorder, delusional disorder, or psychosis not otherwise specified as a part of psychotic disorders diagnoses (Kendler, Gallagher, Abelson, & Kessler, 1996). Note that only one psychosis variable was used for multiple disorders as it was the only disorder variable in the public use dataset of the National Comorbidity Survey. The analyses were based on binary presence of lifetime diagnoses (1 = *presence of the disorder*, 0 = *absence of the disorder*).

Number of living biological children. At the follow-up, each participant was asked, "How many living biological children do you have, not counting step children, adopted children, or foster children?" This question was then used as one of the primary outcome measures.

Parent−child relationship quality. At the follow-up, each participant was asked, "On a scale from 0 to 10 where 0 means "the worst possible relationship" and 10 means "the best possible relationship" how would you rate your overall relationship with your (child/children) these days?" This question was used as the other primary outcome measure.

Moderating variables. Moderating variables included self-reported sex and age at the baseline.

Control variables. Self-reports of the number of biological children at baseline, marital status, household income, education, race, trauma exposure, and geographic region collected at baseline were used to control for confounding variables in this study. The number of living biological children at baseline was calculated based on the participant's self-reports of their biological children's ages at the follow-up. Age and education were continuous count variables. The household income was measured on a scale ranging

from 0 (*no income*) to 21 (*$100,000 or more*). Note that trauma was measured dichotomously and defined as exposure to direct combat experience, being in a life threatening accident, being involved in a flood fire or natural disaster; witnessing someone being badly hurt, injured, or killed; being raped; being sexually molested; being physically attacked or assaulted; being threatened with a weapon, held captive, or kidnapped; or having gone through any other terrible experience that most people never go through (i.e., 1 = *exposed to trauma*, 0 = *not exposed to trauma*, Bromet, Sonnega, & Kessler, 1998). Note that the average age of the children was also controlled when predicting the parent–child relationship quality.

Planned analyses. The goals of the analyses were to examine the latent structure of pathology and then to examine how latent psychopathology predicted fertility and parent–child relationships across the life span. Given this, the results were analyzed in two major stages: (a) performing a confirmatory factory analysis to obtain the factor scores of the latent psychopathology variables, and (b) running varying-coefficient models to examine the effects of the latent psychopathology constructs on fertility and parent–child relationship quality across sex and age.[1]

In performing a confirmatory factor analysis, four primary confirmatory models were considered. The first confirmatory model was a four factor model comprised of fear (social phobia, panic disorder, simple phobia, agoraphobia), anxious misery (posttraumatic stress, generalized anxiety, major depression, dysthymia, and bipolar), externalizing (drug use, alcohol use, and antisocial personality), and psychosis (schizophrenia, schizophreniform disorder, schizoaffective disorder, delusional disorder, or psychosis not otherwise specified; note that this was only included by one variable). The structure of fear, anxious misery, and externalizing disorders was based on Cox, Clara, and Enns (2002) and Krueger (1999), and psychosis was used as a separate factor given that prior evidence has found that it is distinct from other disorders (Kotov et al., 2010). The second confirmatory model closely resembled the first model, except that a second higher order factor included both fear and anxious misery disorders (Krueger, 1999).

The third model was based on the *DSM–5* organization of psychopathology, including the (a) anxiety pathology, including social phobia, panic disorder, specific phobia, agoraphobia, and generalized anxiety disorder; (b) trauma and stressor-related pathology, including only posttraumatic stress disorder; (c) depression pathology, including major depression and dysthymia; (d) bipolar and related pathology, including only bipolar disorder; (e) substance-related pathology, including alcohol use disorder (including both alcohol abuse and alcohol dependence) and drug use disorder (including both drug abuse and drug dependence); (f) personality pathology, including only antisocial personality disorder; and (g) the schizophrenia spectrum pathology (including schizophrenia, schizophreniform disorder, schizoaffective disorder, delusional disorder, or psychosis not otherwise specified as a part of psychotic disorders diagnoses, but only being made up of one single item; Kendler et al., 1996). The last model was based on a hierarchical *DSM–5* organization, nesting anxiety, posttraumatic stress, depression, and bipolar pathology within a higher order internalizing factor and substance use and antisocial personality pathology into a higher order externalizing factor (see Figure 1 for a diagram of each of the factor models).

Because of the influence of sample size on chi-squared statistics, the current model examined the practical indices of model fit: root mean square error of approximation (RMSEA), comparative fit index (CFI), Tucker−Lewis Index (TLI), and standardized root-mean-square residual (SRMR). Additionally, given that the different models had differed in terms of complexity, the Akaike's information criterion (AIC) and the Bayesian information criterion (BIC) were also considered to ensure that the confirmatory model balanced model fit with model parsimony. Both the AIC and the BIC penalize for model complexity, and, as such, both indices would only favor more complex models if they showed substantially better fit (Song & Belin, 2008). Note that this analysis used stratified weights to ensure that the results were representative of the United States population, using the packages lavaan and lavaan.survey (Oberski, 2014; Rosseel, 2012). A diagonally weighted least squares estimator was used to account for the non-normality in the data structure.

Following the confirmatory factor analysis, a varying-coefficient model was estimated to examine how the psychopathology factor scores predicted (a) the number of children in the follow-up and (b) quality of the parent–child relationship. As recommended by Stearns et al. (2010), these varying-coefficient models were tested using Generalized Additive Models (GAMs) and the following equations:

$$\begin{aligned} Offspring = {} & \gamma_0 + \gamma_1 * BaselineOffspring + \gamma_2 * MaritalStatus \\ & + \gamma_3 * Income + \gamma_4 * Race + \gamma_5 * Education \\ & + \gamma_6 * Region + \gamma_7 * Trauma + \gamma_8 * Sex \\ & + \gamma_9 * Psychopathology + f_1(Age) \\ & + f_2(Age)Psychopathology \\ & + f_3(Age)Psychopathology * Sex \end{aligned}$$

The notation *Offspring* represents the number of living biological children at the follow-up (or the outcome variable), γ_0 denotes the model intercept for the number of offspring, γ_1 denotes the number of biological children at the baseline, γ_2 denotes the participant's marital status (via dummy coded variables), γ_3 denotes the participant's income, γ_4 denotes the participant's race (via dummy coded variables), γ_5 denotes the participant's education (in years), γ_6 denotes the participant's geographical region (via dummy coded variables), γ_7 denotes the self-reports of a participant's exposure to trauma in his or her lifetime, γ_8 denotes the participant's sex, and γ_9 denotes the main effect of the psychopathology of interest. The terms *f* denote thin-plate regression smoothing splines, and terms in the parenthesis can have a linear or nonlinear trajectory. As such, the term f_1 denotes the main effect and effect of age on the number of children, allowed to be a linear or nonlinear trajectory. Note that the primary terms of interest are the last two terms, f_2 (*Age*) *Psychopathology* and f_3 (*Age*) *Psychopathology* Sex*, which represent varying-coefficients of the impact of the linear effects of psychopathology factor of interest on the dependent variable, based on a nonlinear moderation of age, for each respective sex. These terms allow the investigation of how

[1] Note that two stages were required due to current software limitations, wherein current software is unable to perform generalized additive structural equation models.

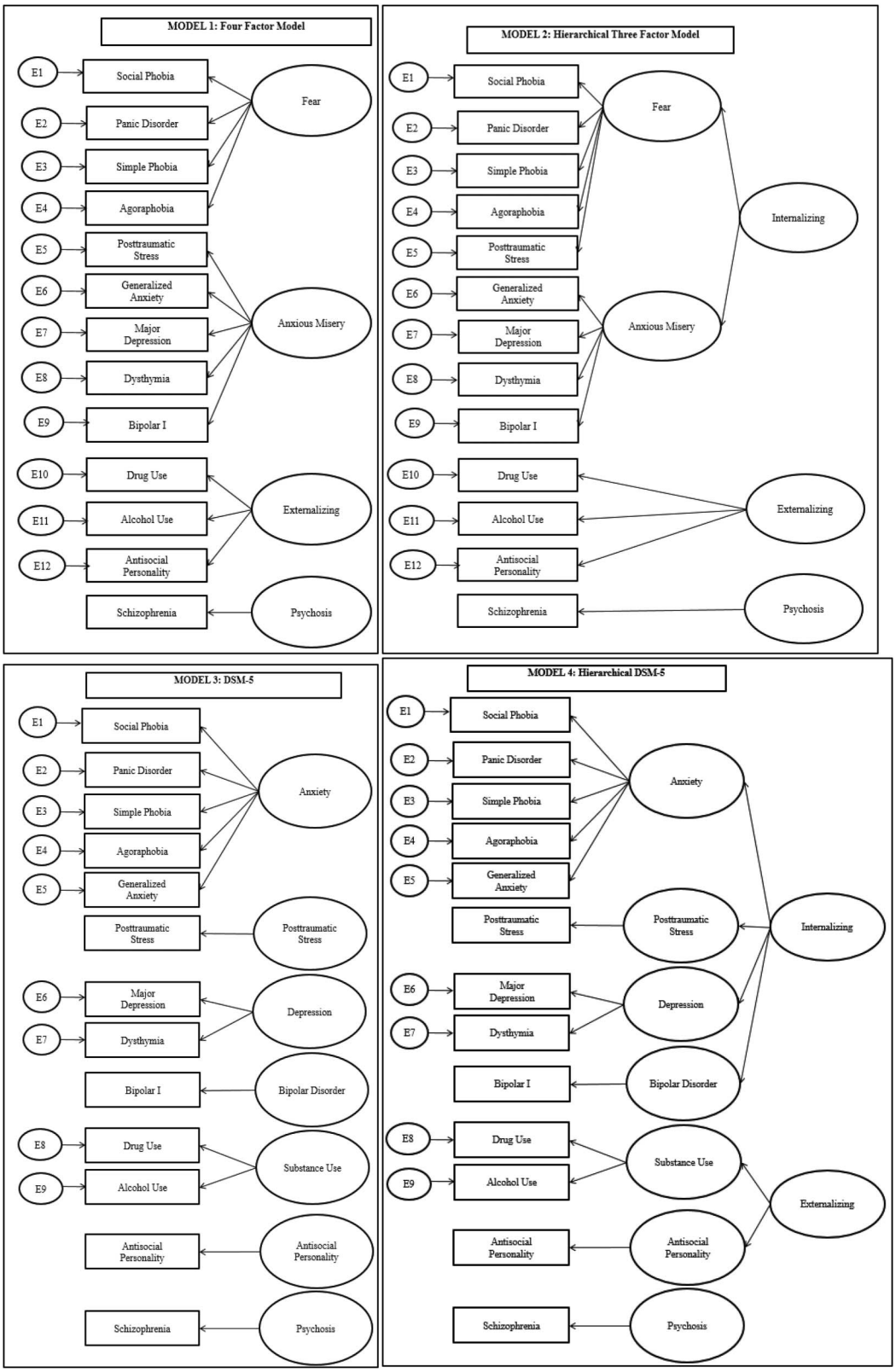

Figure 1. This figure depicts the four models being tested. Note covariances among latent factors are not depicted.

sex and age moderate the effect of psychopathology on the outcome. Importantly, this allows for the estimation of how each psychopathology construct changes across the life span. These coefficients allow for the impact of each psychopathology factor to be constant over age, change linearly, or have peak periods of impact. Separate models were run for each latent psychopathology of interest. For all analyses, the analyses used stratified weights to ensure that the results were representative of the United States population. The GAMs were estimated using the package mgcv (Wood, 2014).

Note that the number of offspring and the parent–child relationship quality were run in separate models. For the parent–child relationship quality, an additional term $\gamma_{10}{}^{*}$ *Child-Age* was added to control for the age of the participant's children. The number of offspring was modeled using a Poisson distribution, and the relationship quality was modeled using a Gaussian distribution.

Note for varying-coefficient models, the primary output comes in the form of continuous smooth curves, best represented by graphs (Tan, Shiyko, Li, Li, & Dierker, 2012). Note that both significance and nonsignificance at particular ages is depicted in graphs, and only text entries of the highest and lowest regression coefficients of the peak periods of interests are reported.

Replicability is tantamount in scientific findings. Given the complexity of the current results, all estimates from the generalized additive models were derived from bootstrapped estimation (with 1,000 bootstrap resamples). Bootstrapping involves resampling the study's data to ensure that the results are stable across different configurations and is one of the primary methods of improving replicability of results in the psychological science without sacrificing power (Funder et al., 2014; Thompson, 1995).

Missing data was handled through random forest multiple imputation using the missForest package (Stekhoven & Bühlmann, 2012), which has shown greater accuracy than other multiple imputation methods. Note that all analyses were analyzed in the R program. Cohen's *d* was calculated for all regression weights using the following formula: $d = Z * 2/\sqrt{N}$ (Wolf, 1986).

Results

Factor Structure of Psychopathology

In comparing the fit of the four different confirmatory factor models, the results suggested that the *DSM–5* structure without the hierarchical factors (i.e., Model 3) provided the best fit to the data ($\chi^2 = 147.201$, $p < .001$, TLI = 0.936, CFI = 0.896, RMSEA = 0.016, SRMR = 0.023), and fit the data significantly better than Model 1 (the four-factor model of psychopathology including fear, anxious misery, externalizing, and psychosis), Model 2 (the three factor model of psychopathology), or Model 4 (a hierarchical *DSM–5* factor model of psychopathology; see Table 1). Additionally, both the AIC and the BIC favored the *DSM–5* structure of psychopathology, suggesting that even when penalizing for model complexity, the *DSM–5* structure of psychopathology provided better fit than other models (see Figure 2). Based on these results, the factor scores of the *DSM–5* organization of psychopathology were obtained and used for subsequent analyses including: anxiety, posttraumatic stress, depression, bipolar, substance use, antisocial personality, and psychosis pathology.

Psychiatric Disorders and Future Fertility

With respect to the anxious pathology predicting the number of offspring, there was a significant three-way interaction among sex, age, and anxiety pathology in predicting the number of offspring ($\chi^2 = 39.88$, $p < .001$; see Figure 3). Not supporting my first hypothesis, the results showed that males between ages 15 to 18 at baseline had significant positive associations with children (peak regression coefficient = 1.776, CI = [0.033, −3.518], $Z = 1.996$, $p = .046$, $d = 0.463$), suggesting that males with high anxiety pathology at ages 15 to 18 had approximately 1.766 more children in the subsequent 9 to 12 years at the follow-up compared with those with low anxiety pathology. In contrast and in support of my third hypothesis, the results also showed that males had a significant negative association between anxiety pathology and the number of children between ages 32 to 52 at baseline (peak regression coefficient = −0.478, CI = [−0.684, 0.273], $Z = -4.563$, $p < .001$,

Table 1
Structure of Pathology Model Fit

Model	χ^2	CFI	TLI	RMSEA	SRMR	AIC	BIC	χ^2 difference
1. Four-factor model	336.349 (60)*	.887	.853	.024	.040	−17,553	−17,385	189.148 (12)*
2. Hierarchical three-factor model	318.177 (61)*	.847	.804	.023	.038	−19,442	−19,278	170.796 (13)*
3. *DSM-5*[a]	147.201 (48)*	.936	.896	.016	.023	−24,206	−23,992	
4. Hierarchical *DSM-5*	246.253 (60)*	.880	.844	.020	.032	−23,733	−23,565	99.052 (12)*

Note. This table includes the fit statistics of the four different structural models of psychopathology. Model 4 had the lowest AIC and lowest BIC values, suggesting that it had best fit while accounting for model complexity. The chi-square difference test reflects the difference in fit between the best-fitting model (Model 4) and the model being evaluated. Each chi-square difference tests suggested that Model 4 provided significantly better fit to the data. CFI = comparative fit index; TLI = Tucker−Lewis Index; RMSEA = root mean square error of approximation; SRMR = standardized root-mean-square residual; AIC = Akaike's information criterion; BIC = Bayesian information criterion.

[a] This row designates the best-fitting model, based on each of the chi-square statistics and each of the practical indices of fit.

* $p < .05$.

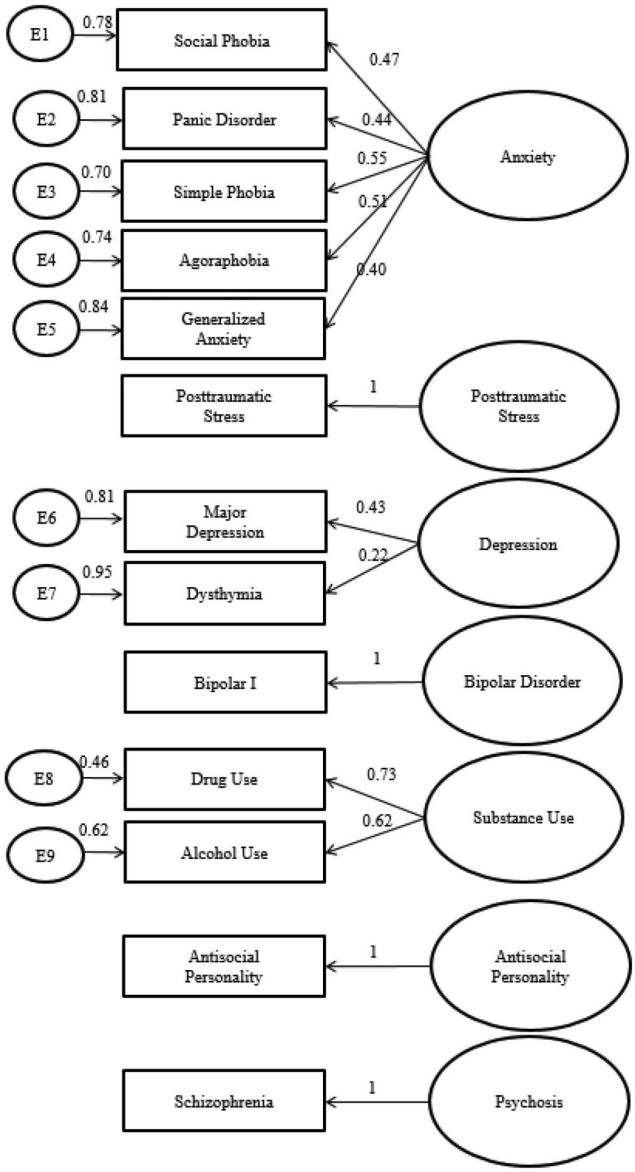

Figure 2. This figure represents the confirmatory factor analysis results of psychiatric disorders. Note covariances among latent factors are not depicted. The model fit indices suggested a good fit to the data (χ^2 = 147.201, $p < .001$, TLI = 0.936, CFI = 0.896, RMSEA = 0.0016, SRMR = 0.023).

$d = -0.816$). This suggests that males with high anxiety pathology between ages 32 and 52 at baseline had approximately 0.478 less children in the subsequent 9 to 12 years compared with males with low anxiety pathology. In contrast, females did not have any positive relationship between anxiety and fertility, which did not support my first hypothesis. Nevertheless, supporting my third hypothesis, females had a significant negative relationship between anxiety pathology and fertility between ages 41 to 51 (peak regression coefficient = −0.421, CI = [−0.701, −0.131], $Z = -2.846$, $p = .004$, $d = -0.676$). This suggests that females with high anxiety pathology between age 41 to 51 at baseline had 0.421 fewer children in the subsequent 9 to 12 years compared with females with low anxiety pathology.

In contrast to anxiety pathology, for posttraumatic stress pathology, sex did not significantly moderate the relationship between age, posttraumatic stress pathology, and fertility ($\chi^2 = 0.000$, $p = .765$), in contrast to my first hypothesis. Nevertheless, age significantly moderated the relationship between posttraumatic stress pathology and fertility ($\chi^2 = 13.570$, $p = .004$). The results indicated that posttraumatic stress pathology significantly predicted lower fertility rates when participants' ages were between 34 to 53 at baseline (peak regression coefficient = −0.156, CI = [−0.227, −0.086], $Z = -4.328$, $p < .001$, $d = -0.680$), supporting my third hypothesis. These results indicate that those with high posttraumatic stress pathology between the ages of 45 to 53 at baseline had 0.156 fewer children 9 to 12 years later compared with those with low posttraumatic stress pathology for both males and females.

For depression pathology, sex did significantly moderate the relationship between age, depression pathology, and fertility ($\chi^2 = 43.070$, $p < .001$). In particular, males had a significant negative relationship between depression pathology and fertility from ages 30 to 52 (peak regression coefficient = −0.729, CI = [−0.999, −0.458], $Z = -5.281$, $p < .001$, $d = -1.012$), supporting my third hypothesis. Likewise supporting my third hypothesis, females had a significant negative relationship between depression pathology and fertility from ages 38 to 52 (peak regression coefficient = −0.829, CI = [−1.209, −0.449], $Z = -4.274$, $p < .001$, $d = -1.014$). This suggests that males with high depression pathology between the ages of 30 to 52 at baseline had 0.729 fewer children 9 to 12 years later compared with males with low depression pathology. Similarly, the results suggested that females with high depression pathology between the ages of 38 to 52 at baseline had 0.828 fewer children 9 to 12 years later compared with females with low depression pathology.

In regard to bipolar pathology, sex did not significantly moderate the relationship between age, bipolar pathology, and fertility ($\chi^2 = 0.562$, $p = .186$), contrary to my first hypothesis. Nevertheless, age significantly moderated the relationship between bipolar pathology and fertility ($\chi^2 = 10.650$, $p = .019$). The results suggest that bipolar pathology was significantly positively related to fertility between ages 16 to 18 (peak regression coefficient = 0.774, CI = [0.080, 1.469], $Z = 2.186$, $p = .029$, $d = 0.337$), supporting my first hypothesis. These results suggest that those with high bipolar pathology from age 16 to 18 had 0.774 more children 9 to 12 years later compared with those with low bipolar pathology for both males and females.

In regard to substance use pathology, sex did not significantly moderate the relationship between age, substance use pathology, and fertility ($\chi^2 = 0.840$, $p = .078$), contrary to my second hypothesis. However, age did moderate the relationship between substance use pathology and fertility ($\chi^2 = 19.180$, $p < .001$). The results suggested that substance use pathology was significantly negatively associated with fertility between ages 33 to 36 (peak regression coefficient = −0.115, CI = [−0.223, −0.115], $Z = -2.097$, $p = .036$, $d = -0.252$), supporting my third hypothesis. These results suggest that those with high substance use pathology from age 33 to 36 had 0.115 fewer children 9 to 12 years later compared with those with low substance use pathology.

For antisocial personality pathology, sex did not significantly moderate the relationship between age, antisocial personality pa-

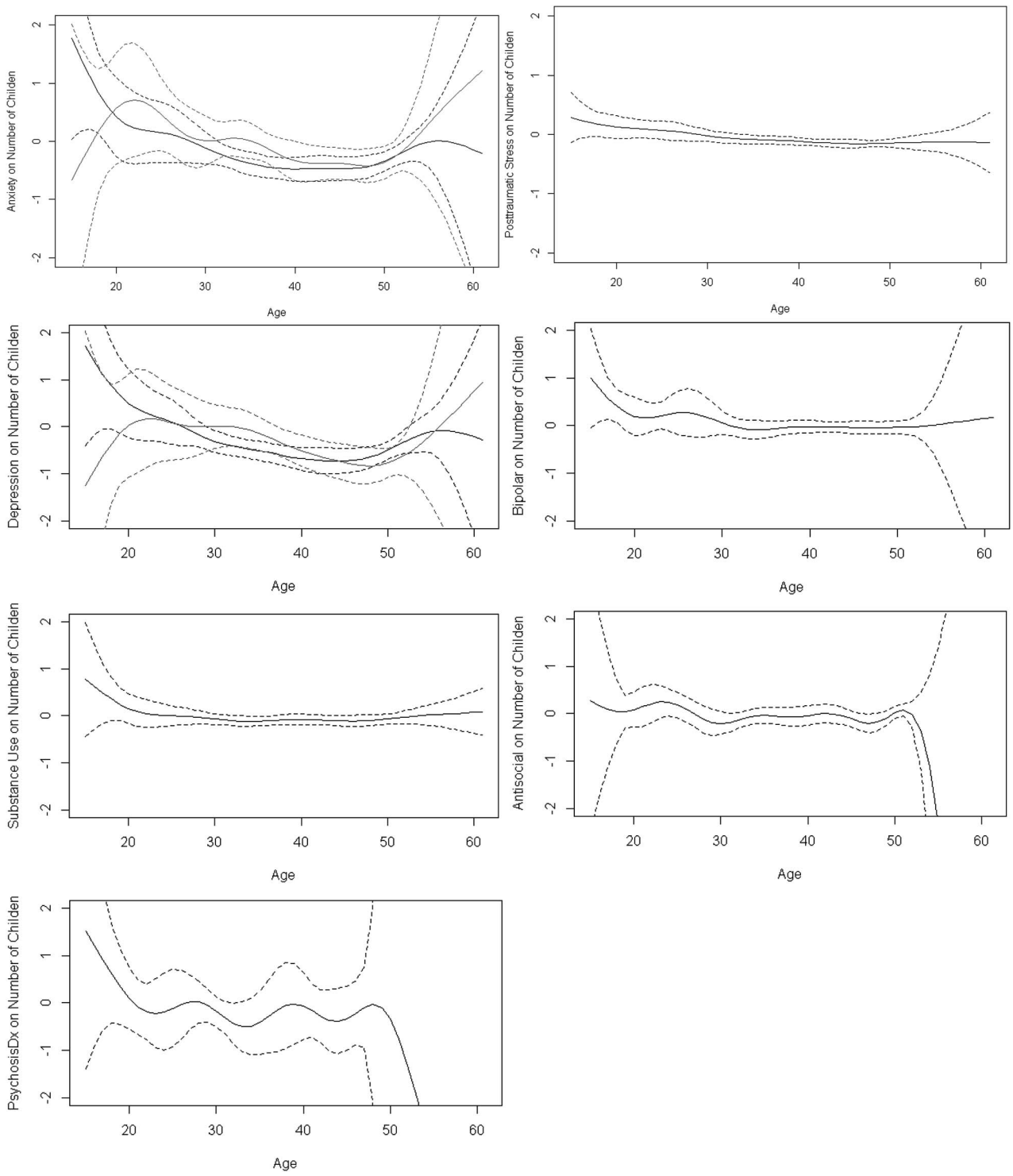

Figure 3. This figure depicts seven graphs: anxiety predicting fertility across age and sex, posttraumatic stress pathology predicting fertility across age, depression pathology predicting fertility across age and sex, bipolar pathology predicting fertility across age, substance use pathology predicting fertility across age, antisocial personality pathology predicting fertility across age, and psychosis pathology predicting fertility across age. Note that the black lines represent the estimates of both males and females for the posttraumatic stress, bipolar, substance use, antisocial personality, and psychosis pathology. For the anxiety and depression pathology graph, the black lines represent the males and the gray lines represent the females. The dashed lines represent the 95% bootstrapped confidence intervals for the estimates.

thology, and fertility ($\chi^2 = 0.000$, $p = .919$), contrary to my second hypothesis. On the other hand, age did significantly moderate the relationship between antisocial personality pathology and fertility ($\chi^2 = 21.520$, $p = .018$). The results suggested that antisocial personality pathology was negatively associated with fertility at the age of 47 (regression coefficient = −0.202, CI = [−0.398, −0.006], $Z = -2.020$, $p = .043$, $d = -0.323$), supporting my third hypothesis. These results suggest that those with high antisocial personality pathology at age 47 had 0.202 fewer children in the following 9 to 12 years compared with those with low antisocial personality pathology.

For psychosis pathology, sex did not significantly moderate the relationship between age, psychosis pathology, and fertility ($\chi^2 = 0.000$, $p = .840$). Nevertheless, age did significantly moderate the relationship between psychosis and fertility ($\chi^2 = 12.640$, $p = .027$). The results suggested that psychosis pathology was negatively associated with fertility at the age of 32 (regression coefficient = −0.436, CI = [−0.870, −0.003], $Z = -1.973$, $p = .048$, $d = -0.235$), supporting my third hypothesis. This suggests that individuals with high psychosis pathology at the age of 32 were associated with having 0.436 fewer children than those with low psychosis pathology.

In sum, the only positive relationships between psychopathology and fertility were (a) in anxiety pathology for males between the ages of 15 to 18 and (b) in bipolar pathology for both males and females between the ages of 16 to 18. All other associations between psychopathology and fertility were negative, with the pattern of results suggesting the primary impact of psychopathology on fertility occurred in participants' 30s and 40s.

Psychiatric Disorders and Future Parent−Child Relationship Quality

For anxiety pathology predicting the parent–child relationship quality, sex significantly moderated the relationship between age, anxiety pathology, and parent–child relationship quality ($F = 7.421$, $p < .001$, see Figure 4). For males, the results indicated anxiety pathology was significantly negatively associated with parent–child relationship between 18 to 21 (peak regression coefficient = −1.310, CI = [−2.294, −0.328], $Z = -2.614$, $p = .009$, $d = -0.604$), 31 to 35 (peak regression coefficient = −1.478, CI = [−2.893, −0.062], $Z = -2.046$, $p = .041$, $d = -0.348$), and 40 to 42 (peak regression coefficient = −1.002, CI = [−1.889, −0.115], $Z = -2.215$, $p = .027$, $d = -0.457$), supporting my fourth hypothesis. These results suggest that males with high anxiety pathology between the ages of 18 to 21, 31 to 35, and 40 to 42 at baseline had poorer parent–child relationship quality 9 to 12 years later compared with those with low anxiety pathology. Similarly, for females, the results suggested that anxiety pathology was significantly negatively related to parent–child relationship quality between 15 to 36 (peak regression coefficient = −5.951, CI = [−8.767, −3.134], $Z = -4.141$, $p < .001$, $d = -0.915$) and 43 to 53 (peak regression coefficient = −4.121, CI = [−6.407, −1.837], $Z = -3.535$, $p < .001$, $d = -0.962$), supporting my fourth hypothesis. These results suggest that females with high anxiety pathology between the ages of 15 to 36 and 43 to 53 at baseline have poorer parent–child relationship quality 9 to 12 years later compared with those with low anxiety pathology.

For posttraumatic stress pathology, the results also showed that sex significantly moderated the relationship between age, posttraumatic stress pathology, and parent–child relationship quality, $F = 7.661$, $p < .001$. For males, the results showed that posttraumatic stress pathology was significantly negatively related to parent–child relationship quality between ages 32 to 38 (peak regression coefficient = −0.810, CI = [−1.281, −0.338], $Z = -3.366$, $p < .001$, $d = -0.654$), supporting my fourth hypothesis. These results suggest that males with high posttraumatic stress pathology between the ages of 32 to 38 at baseline had poorer parent–child relationship quality than those with low posttraumatic stress pathology. Likewise, for females the results showed that posttraumatic stress pathology was significantly negatively related to parent–child relationship quality between ages 26 to 36 (peak regression coefficient = −1.003, CI = [−1.607, −0.400], $Z = -3.260$, $p = .001$, $d = -0.559$) and 47 to 50 (peak regression coefficient = −1.499, CI = [−2.931, −0.066], $Z = -2.051$, $p = .040$, $d = -0.497$), supporting my fourth hypothesis. These results suggest that females with high posttraumatic stress pathology between the ages of 26 to 36 and 47 to 50 at baseline had poorer parent–child relationship quality than those with low posttraumatic stress pathology.

Likewise, for depression pathology, the results also showed that sex significantly moderated the relationship between age, depression pathology, and parent–child relationship quality ($F = 13.665$, $p < .001$). For males, depression pathology was significantly negatively associated with parent–child relationship quality between ages 31 to 38 (peak regression coefficient = −1.785, CI = [−3.048, −0.522], $Z = -2.770$, $p = .005$, $d = -0.458$), supporting my fourth hypothesis. These results suggest that males with high depression pathology between the ages of 31 to 38 at baseline had poorer parent–child relationship quality 9 to 12 years later than those with low depression pathology. For females, depression pathology was significantly negatively associated with parent–child relationship quality between ages 15 to 18 (peak regression coefficient = −8.500, CI = [−12.807, −4.193], $Z = -3.868$, $p < .001$, $d = -0.854$), 23 to 26 (peak regression coefficient = −3.050, CI = [−5.803, −0.297], $Z = -2.172$, $p = .030$, $d = -0.453$), 32 to 36 (peak regression coefficient = −2.970, CI = [−4.937, −1.004], $Z = -2.961$, $p = .003$, $d = -0.517$), and 46 to 53 (peak regression coefficient = −5.517, CI = [−8.722, −2.312], $Z = -3.374$, $p < .001$, $d = -0.818$), supporting my fourth hypothesis. These results suggest that females with high depression pathology between the ages of 15 to 18, 23 to 26, 32 to 36, and 46 to 53 at baseline had poorer parent–child relationship quality 9 to 12 years later compared with those with low depression pathology.

Similarly, for bipolar pathology, sex significantly moderated the relationship between age, bipolar pathology, and parent–child relationship quality ($F = 10.886$, $p < .001$). For males, the results showed that bipolar pathology was significantly negatively related to parent–child relationship quality between the ages of 20 to 21 (peak regression coefficient = −1.054, CI = [−2.021, −0.089], $Z = -2.141$, $p = .032$, $d = -0.393$), 32 (peak regression coefficient = −1.296, CI = [−2.517, −0.075], $Z = -2.080$, $p = .038$, $d = -0.344$), 41 (peak regression coefficient = −1.087, CI = [−1.928, −0.246], $Z = -2.533$, $p = .011$, $d = -0.479$), and 51 to 58 (peak regression coefficient = −23.603, CI = [−47.046, −0.160], $Z = -1.973$, $p = .048$, $d = -0.438$), supporting my fourth hypothesis. These results

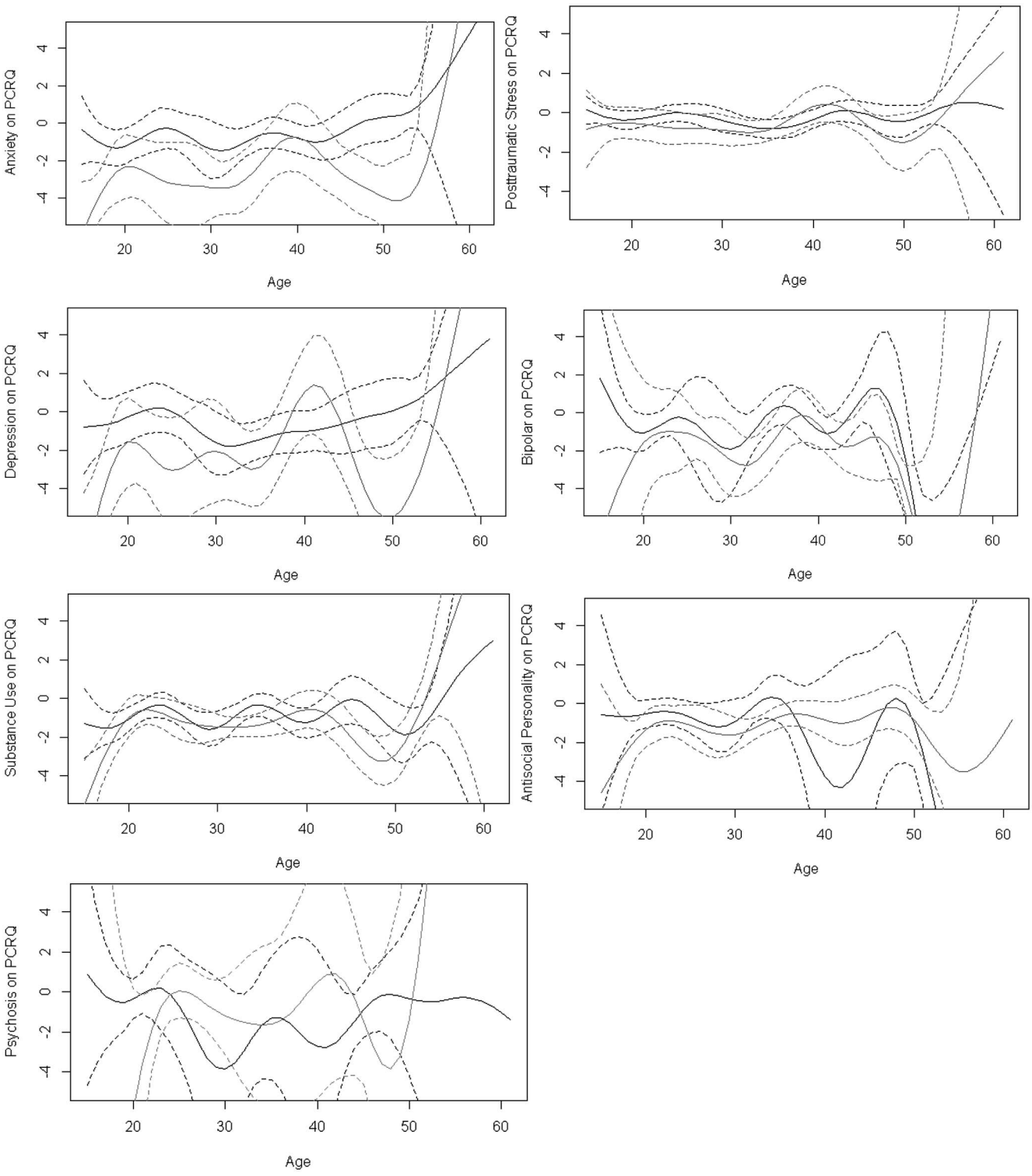

Figure 4. This figure depicts seven graphs, where anxiety, posttraumatic stress, depression, bipolar, substance use, antisocial personality, and psychosis pathology each predict parent–child relationship quality across both age and sex. The black lines depict the regression coefficient estimates for the males across each age. The gray lines depict the regression coefficient estimates for the females across each age. The dashed lines represent the 95% bootstrapped confidence intervals for the estimates.

suggest that males with high bipolar pathology between the ages of 20 to 21, 32, 41, and 51 to 58 have poorer quality parent–child relationships compared with those with low bipolar pathology. For females, the results showed that bipolar pathology was significantly negatively related to parent–child relationship quality between ages 26 to 35 (peak regression coefficient = −2.751, CI = [−4.148, −1.353], $Z = -3.858$, $p < .001$, $d = -0.662$), 41 to 44 (peak regression coefficient = −1.765, CI = [−3.026, −0.504], $Z = -2.743$, $p = .006$, $d = -0.563$), and 49 to 53 (peak regression coefficient = −8.378, CI = [−15.486, −1.269], $Z = -2.310$, $p = .021$, $d = -0.667$), supporting my fourth hypothesis. These results suggest that females with high bipolar pathology between the ages of 26 to 35, 41 to 44, and 49 to 53 at baseline had poorer parent–child relationships 9 to 12 years later compared with those with low bipolar pathology.

Likewise, sex significantly moderated the relationship between age, substance use pathology, and parent–child relationship quality ($F = 27.478$, $p < .001$). For males, substance use pathology significantly negatively predicted parent–child relationship quality between ages 16 to 21 (peak regression coefficient = −1.513, CI = [−2.450, −0.577], $Z = -3.167$, $p = .002$, $d = -0.746$), 26 to 33 (peak regression coefficient = −1.587, CI = [−2.461, −0.713], $Z = -3.560$, $p < .001$, $d = -0.629$), 37 to 41 (peak regression coefficient = −1.251, CI = [−2.030, −0.472], $Z = -3.147$, $p = .002$, $d = -0.563$), and 50 to 53 (peak regression coefficient = −1.859, CI = [−3.315, −0.404], $Z = -2.504$, $p = .012$, $d = -0.621$), supporting my fourth hypothesis. These results suggest that males with high substance use pathology between ages 16 to 21, 26 to 33, 37 to 41, and 50 to 53 had poorer parent–child relationships 9 to 12 years later compared with those with low substance use pathology. For females, substance use pathology significantly negatively predicted parent–child relationship quality between age 15 to 19 (peak regression coefficient = −5.467, CI = [−7.760, −3.174], $Z = -4.673$, $p < .001$, $d = -1.032$), 24 to 37 (peak regression coefficient = −1.459, CI = [−1.945, −0.973], $Z = -5.887$, $p < .001$, $d = -1.010$), and 44 to 52 (peak regression coefficient = −3.198, CI = [−4.493, −1.903], $Z = -4.842$, $p < .001$, $d = -1.240$), supporting my fourth hypothesis. These results indicate that females with high substance use pathology between ages 15 to 19, 24 to 37, and 44 to 52 had poorer parent–child relationship quality 9 to 12 years later compared with those with low substance use pathology.

Sex also significantly moderated the relationship between age, antisocial personality pathology, and parent–child relationship quality ($F = 17.14$, $p < .001$). Interestingly, for males, antisocial personality pathology did not significantly predict parent–child relationship quality at any time, contrary to my fourth hypothesis. In contrast, for females, antisocial personality pathology significantly negatively predicted poorer parent–child relationships between ages 17 to 35 (peak regression coefficient = −3.028, CI = [−5.541, −0.515], $Z = -2.362$, $p = .018$, $d = -0.549$) and 51 to 54 (peak regression coefficient = −2.727, CI = [−5.034, −0.419], $Z = -2.316$, $p = .021$, $d = -0.669$), supporting my fourth hypothesis. These results suggest that females with high antisocial personality pathology between the ages of 17 to 35 and 51 to 54 had poorer parent–child relationship quality 9 to 12 years later compared with those with low antisocial personality pathology.

Similarly, sex also significantly moderated the relationship between age, psychosis pathology, and parent–child relationship quality ($F = 8.003$, $p < .001$). For males, psychosis pathology significantly negatively predicted parent–child relationship quality between ages 31 to 32 (peak regression coefficient = −3.533, CI = [−7.062, −0.005], $Z = -1.963$, $p = .049$, $d = -0.334$) and 43 to 44 (peak regression coefficient = −2.065, CI = [−4.084, −0.046], $Z = -2.005$, $p = .045$, $d = -0.430$), supporting my fourth hypothesis. These results suggest that males with high psychosis pathology between ages 31 to 32 and 43 to 44 had poorer quality parent–child relationships 9 to 12 years later compared with those with low psychosis pathology. For females, psychosis pathology significantly negatively predicted parent–child relationship quality at age 21 (peak regression coefficient = −3.578, CI = [−6.974, −0.181], $Z = -2.065$, $p = .039$, $d = -0.440$), in contrast to my fourth hypothesis. This suggests that females with high psychosis pathology at age 21 had lower parent–child relationship quality 9 to 12 years later compared with those with low psychosis pathology.

In sum, psychopathology did not significantly positively predict parent–child relationship quality across any pathology or any age. With the exception of males with antisocial personality pathology, any higher psychopathology predicted significantly poorer parent–child relationships. In regard to sex differences, for almost all types of psychopathology (with the exception of psychosis), the impact of psychopathology on parent–child relationship quality occurred over a larger age range for females compared with males. With the exception of posttraumatic stress psychopathology, the effect sizes of the psychopathology on parent–child relationship quality was more deleterious for females than it was for males. With the exception of bipolar pathology, the impact of psychopathology on parent–child relationship quality occurred at earlier ages for females than for males.

Discussion

These results hold important implications for the study of current evolutionary adaptiveness. In particular, the findings demonstrated that the evolutionary impact of psychopathology depended upon the sex of the participant, the type of psychopathology, and the participant's developmental context. In particular, for males, anxiety pathology was associated with increased fertility in late adolescence, contrary to my first hypothesis, followed by the production of fewer offspring in one's 30s and 40s, supporting my third hypothesis. For females, anxiety pathology was only associated with decreased fertility in one's 40s, contrary to my first hypothesis and supporting my third hypothesis. For posttraumatic stress and substance use pathology, there were no differences between the sexes, contrary to my first hypothesis, but the results showed decreases in fertility rates in one's 30s and 40s in both sexes, supporting my third hypothesis. Depression pathology was also associated with decreased fertility rates in one's 30s and 40s, with males experiencing an earlier impact of depression pathology on fertility than females, contrary to my first hypothesis and supporting my third hypothesis. Antisocial personality and psychosis pathology were consistent across both males and females, but the impact of antisocial personality pathology and psychosis pathology on decreased fertility only occurred in a single year (age 47 for antisocial personality pathology and age 32 for psychosis pathology), supporting my third hypothesis. Last, for both males and females, bipolar pathology was the only psychopathology that was unidirectionally associated with increased fertility, and the positive associations occurred in late adolescence, partially sup-

porting my first hypothesis. With the exception of the findings regarding increased fertility in early adolescence for bipolar pathology in males and females and anxiety pathology in males, these results suggest that psychopathology is uniquely associated with decreased fertility rates, and its impact on decreased fertility primary occurs within one's 30s to 40s.

Moving from the quantity of children to parent–child relationship quality, the results showed that no psychopathology positively predicted parent–child relationship quality across any age. In contrast, higher psychopathology of all kinds was associated with poorer parent–child relationships (except for antisocial personality pathology in males), broadly supporting my fourth hypothesis. There was large variation between different types of psychopathology in regard to the ages in which psychopathology had a primary impact, but every type of psychopathology predicted poorer parent–child relationships in one's 30s. The results showed a consistent pattern of sex differences in the impact of psychopathology on the parent–child relationship quality. In particular, the impact of psychopathology on parent–child relationship quality occurred over a larger age range for females than for males (except for psychosis pathology), and the impact of the psychopathology on parent–child relationship quality was more deleterious for females than males (except for posttraumatic stress psychopathology).

For findings of anxiety pathology in males, the observed pattern of results for fertility fits a process called antagonistic pleitropy, wherein opposing effects are observed at different ages (Williams, 1957). In line with the findings of anxiety pathology, evolutionists have suggested that those genes that have stronger influences on sexual selection early in life and deleterious effects in later life will accumulate in populations; this pattern has been observed in many diseases in biology (i.e., sickle cell disease; Carter & Nguyen, 2011).

The results partially support the predictions about fertility made by Belsky et al. (1991), who emphasized that psychopathology led to a quantity over quality approach in early adulthood with sex-specific relationships for men and women. The results support that anxiety pathology is associated with a quantity over quality approach in early adulthood for males and linked to decreased quantity and quality of children, as well as poor relationships in middle to late adulthood for males and females. However, Belsky et al. (1991) theorized the relationship would only occur with females. The results also partially support the predictions made by Belsky et al. (1991) with regard to bipolar pathology, wherein the quantity over quality approach was found in early adulthood for both men and women, but the results were not sex-specific as theorized by Belsky et al. The results of early pathology for other types of psychopathology did not support the theorists' predictions, but the results did support the theorists' notion that psychopathology would predict poor parent–child relationships in mid to late adulthood (except for antisocial personality pathology). Thus, the results broadly supported the harmful impact of psychopathology on parent–child relationships as theorized and mostly refuted the predicted adaptive role for psychopathology in late adolescence and early adulthood.

These findings contrast prior studies that have found that psychopathology is only associated with decreased fertility (Bundy et al., 2011; Calzeroni et al., 1989; Essock-Vitale & McGuire, 1989; Power et al., 2013), with the results suggesting that anxiety pathology has an early positive impact on fertility in late adolescence in males, and the results suggest that bipolar pathology is uniquely associated with increased fertility in late adolescence for both males and females.

Nevertheless, the results support prior findings that psychosis pathology is associated with decreased fertility (Bundy et al., 2011; Power et al., 2013) and extends these findings by demonstrating that the impact of the psychosis on fertility occurs within the person's 30s. Likewise, the current results also support prior findings that depression and substance abuse are associated with decreased fertility (Power et al., 2013) and suggest that the impact of fertility occurs between one's 30s to 40s in depression pathology and in participants' 30s in substance use pathology.

The results suggesting that bipolar pathology is only positively associated with fertility contrast prior research, which suggests that bipolar disorders are associated with decreased fertility when collapsed across the life span (Jonsson, 1991; Power et al., 2013). Further, the current results suggesting that bipolar pathology is associated with increased fertility in late adolescence partially conflict with a prior study which suggested that bipolar pathology is associated with decreased fertility in late adolescence (Baron et al., 1982). This conflicting result is likely due to the prior study's results being misleading as the prior study only had a single participant with bipolar pathology within this age range. Nevertheless, the current results and the prior study both found no relationship between bipolar disorder and fertility in middle to late adulthood (Baron et al., 1982). The findings of antisocial personality pathology differ from previous results regarding criminality. In particular, past research demonstrated a positive relationship between criminality and fertility (Yao et al., 2014), whereas the current results only showed a negative relationship between antisocial personality pathology and fertility.

Further, the current results extend previous research which reported people with high anxiety, high depression, high substance use, and psychosis pathology have poor parent-children relationship quality with their parents (Branje, Hale, Frijns, & Meeus, 2010; Kim & Cicchetti, 2004; Schiffman et al., 2002; Shelton & Van Den Bree, 2010) and suggests that persons with high psychopathology across any type also is associated with poorer relationships with their children.

It is important to note the magnitude of the associations between psychopathology and quantity of children. In particular, the results showed that the positive impact of high anxiety pathology in males and high bipolar pathology in males and females were strongly associated with an increase in approximately one to two more children than those with low pathology during late adolescence and early adulthood. The impact of high depression on fertility occurred in mid to late adulthood and was associated with a decrease of approximately one child. The negative high anxiety pathology and high psychosis pathology were also moderately strong, being associated with the production of one half of a child fewer during mid to late adulthood. The impact of high antisocial personality pathology and posttraumatic stress pathology was also weaker, being associated with only a decrease in one fifth of a child in mid to late adulthood. Thus, the impact of psychopathology on fertility was mostly moderate to strong, which is particularly notable given that even small effects can be quite impactful in evolutionary contexts (Perfeito, Fernandes, Mota, & Gordo, 2007).

The magnitude of the associations in the parent–child relationship quality was particularly impactful in females. In particular, the results showed that high anxiety, high depression, and high substance use pathology in females were associated with large deficits in the parent–child relationship quality (minimum ds < -0.80). The effects of high anxiety in males, high posttraumatic stress pathology in males and females, high bipolar pathology in females, high substance use pathology in males, and high antisocial personality in females were each associated with moderate deficits of parent–child relationships (minimum ds < -0.50). Last, the impact of high depression in males, high bipolar pathology in males, and high psychosis pathology in males and females were associated with small deficits in the parent–child relationships (minimum ds < -0.20). Thus, the strength of the impact of psychopathology on poor parent–child relationship tended to be moderate to strongly impactful for most psychopathology.

Although these results represent the first largescale investigation of whether anxiety, posttraumatic stress, depression, bipolar, substance use, antisocial personality, and psychosis pathology affect fertility rates and parent–child relationship quality, limitations of the current study prevent definitive conclusions. The current study used the *DSM–III–R* diagnoses, whereas the most current psychiatric diagnostic manual is the *DSM–5*. Nevertheless, the diagnostic criteria for these disorders are generally consistent across the *DSM–III–R* to the *DSM–5*, and it is unlikely that many of these changes would impact the findings. For example, many changes occurred to redefine single items within diagnoses (e.g., separate categories of sections in schizophrenia symptoms, when the current results collapse across all psychotic disorders; Tandon et al., 2013). Additionally, some diagnoses have removed or retained a single diagnostic criterion (e.g., the removal of the single diagnostic criterion, autonomic hyperarousal, from generalized anxiety disorder [Andrews et al., 2010] and the inclusion of the single diagnostic criterion, fear of showing anxiety symptoms, for social phobia or social anxiety disorder [Bogels et al., 2010]). Consequently, although the results do not use the current diagnostic assessment, it is likely that the results would generalize to the *DSM–5* disorders.

Another limitation of the current work is the lack of comparison between genetic and environmental influences in phenotypic expression. Although this study controlled for exposure to traumatic events, this study did not explicitly examine nongenetic components, which may impact both fertility and parent–child relationship quality. This is a notable limitation of the current study given that the environment contributes between 15% and 80% of the variation depending on the pathology. Not accounting for the other influences in the environment, such as the impact of pharmacotherapy and psychotherapy, could also impact the influence of psychopathology on fertility. Further, this study did not take into account other genetic influences, like epigenetics. Thus, future studies should consider examining selective pressures using a twin-study design to examine the direct effects of environment and heredity on the prediction of evolutionary adaptiveness.

Additionally, although the present study examined the parent–child relationship quality, more research is needed to determine if there are other ways that psychiatric disorders may positively affect reproductive success (i.e., if parental worries about children result in medical treatment and prevention of medical illnesses or death in children). Further, the metric of the parent–child relationship summarized the relationship for all children, rather than including a quality rating for the relationship of each child, and, consequently, more research is needed to see if the results would be consistent if ratings of the relationship were obtained for each child. Additionally, given that the impact of psychopathology on evolution may also be tied to siblings' fertility (Power et al., 2013), future work should also consider the impact of psychiatric disorders on fertility in siblings. Another limitation of this study is that it only looked across two generations, and hence more research is needed to determine if these effects would be consistent across greater numbers of generations. Moreover, as evolutionary fitness is impacted by changes in the selection environment, more research is needed to determine whether psychiatric disorders affect evolutionary adaptiveness in other cultures. Last and most important, because of the changing environment, it should be noted that the current findings do not reflect the adaptiveness of psychiatric disorders in the ancestral environment, but rather the current findings only apply to the adaptiveness of psychiatric disorders in the modern environment (Stearns et al., 2010).

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Received September 14, 2015
Revision received May 23, 2016
Accepted May 25, 2016 ■