Current Evolutionary Adaptiveness of Anxiety:

Extreme Phenotypes of Anxiety Predict Increased Fertility Across Multiple Generations

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Abstract

Objective: Although recent research has begun to examine the impact of elevated anxiety on evolutionary fitness, no prior research has examined anxiety across a continuum. Such research is important as the effect of traits across a continuum on fertility hold important implications for the levels and distribution of the traits in later generations. **Method**: In a three-generational sample ($N = 2{,}657$) the linear and quadratic relationship between anxiety and the number of children, grandchildren, and great-grandchildren 15 years later was examined. **Results**: The findings suggested that anxiety had a positive quadratic relationship with the number of children, grandchildren, and great-grandchildren 15 years later. These relationships were not significantly moderated by sex. Moreover, most of the variance between anxiety and the number of great-grandchildren was explained by anxiety's influence on the number of children and grandchildren, as opposed to anxiety having an independent direct impact on the number of great-grandchildren. **Conclusion:** These findings suggest that extreme values from the mean anxiety are associated with increased evolutionary fitness within the modern environment.

Keywords: adaptiveness, evolution, disorder, anxiety

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Recent research suggests that one in nine persons will experience an anxiety disorder in any given year around the world (Baxter et al., 2013). Anxiety symptoms and disorders are characterized by tension, worry, fear, and avoidance (Jacobson and Newman, 2014; Newman et al., 2013; Olatunji et al., 2010; Szkodny et al., 2014). Anxiety disorders are a risk factor for heart disease (Roest et al., 2010) and associated with increased health care cost (Simon et al., 1995). As clinical anxiety has broad societal consequences, it is important to study.

Evolutionary theory dictates that heritable traits which impact an organism's ability to reproduce will have a profound impact on the levels of a trait within a species (MacDonald, 1999). Although some evolutionary theorists posit that the impact of evolutionary change occurs slowly and gradually over millions of years (Dawkins, 1986), recent theories and research have suggested that heritable traits which impact one's fertility can cause fast and dramatic changes in the levels of a given trait within the population (Gould, 1972; Swanson and Vacquier, 2002; Yoshida et al., 2003). Note that although these studies refer to the possibility of adaptive change, which requires a very large number of generations at the phenotypic level (Hawks et al., 2007), it is important to study heritable traits that may affect one's ability to reproduce within the present population to facilitate the exploration of candidate traits that should be examined across a larger number of generations.

Anxiety is one such moderately heritable trait and thus may impact evolutionary fitness (Eley et al., 2003; Lau et al., 2006; Legrand et al., 1999; Ogliari et al., 2006). Recent evidence suggests that anxiety at pathological levels is related to producing a greater number of children at earlier ages, followed by having fewer numbers of children at older ages (Jacobson, 2016).

Although it is unknown whether anxiety is related to fertility along a normative continuum, clinical psychologists and some evolutionary theorists have long posited that anxiety is adaptive if it is expressed in moderate levels, but disadvantageous when there is too little or too much present (recommended reading: Barlow, 1988; Bergstrom and Meacham, 2016; Marks and Nesse, 1994; Nesse, 1999; Nesse, 1987, 2001). Consequently, more research should examine a normative range of anxiety levels, rather than just pathological levels, to arrive at a more complete understanding of anxiety's relationship to fertility.

Because the prior investigation of anxiety did not include a range of anxiety levels (Jacobson, 2016), it is unclear whether prior findings hold implications towards shifting levels of these traits across generations. To determine the implied influence of anxiety on the mean and variance of anxiety across generations, it is necessary to know the impact of anxiety on fertility across a continuum. In evolutionary studies, there are three primary selection forces that may shape a trait's levels across generations: (1) stabilizing selection, (2) disruptive selection, and (3) directional selection (Rueffler et al., 2006). Stabilizing selection is said to occur when values closest to the mean of a distribution are related to increased production of offspring (i.e. inverted U; Roff, 2012). Human birth weight is a classic example of stabilizing selection as an average birth weight is favored because a low birth weight can increase one's likelihood of mortality, and a high birth weight can decrease chances of a successful delivery through the pelvis; this suggests that average birth weight alone is unlikely to change across generations (Ulizzi et al., 1981). In contrast, disruptive selection suggests that extreme values away from the population mean are related to increased production of offspring (Rueffler et al., 2006). An example of disruptive selection is rabbits living in an area with white rocks and black rocks, wherein rabbits who are either highly white or highly black may be likely to blend in with the terrain, whereas

grey rabbits are likely to be targeted by predators; disruptive selection would result in a stable

mean of a trait across time, but increased polarization of a given trait (Seehausen et al., 2014).

Lastly, directional selection favors one end of a continuum of a given trait (Rueffler et al., 2006).

For example, finches with small beaks were effective in eating small seeds in an area

consistently in a wet climate, but, when droughts occurred, finches with larger beaks able to eat

larger seeds prospered due to a lack of availability of small seeds; thus, directional selection

would result in an increased or decreased level of a trait across time (Campbell and Reece,

2002).

Notably, as discussed at length by the seminal work of Keller and Miller (2006), the

relationship between traits and current fertility also has implications towards the genetic

ontology of psychiatric disorders. In seeming opposition to the theories by clinical psychologists

and some evolutionary theorists (Barlow, 1988; Bergstrom and Meacham, 2016), Keller and

Miller (2006) theorize that because natural selection often favors uniformity the variation found

across persons in anxiety is likely not species-typical behavior. In addition, Keller and Miller

(2006) posit that evolution, through directional selection, attempts to eliminate psychopathology

across generations, nevertheless mutations from polygenetic sources generate psychiatric

disorder in each generation (by inherited and de novo mutations). In particular, Penke et al.

(2007) theorize that personality traits do not decrease fitness, although personality may be

moderated by environmental heterogeneity, also suggesting that genetic variation between

personality traits and psychopathology are maintained by different evolutionary processes.

Evidence of the strength and distribution thus hold potential implications towards the complexity

and the types of genes that define anxiety.

The goal of the current study was to investigate the following research questions: whether anxiety predicted later fertility across a continuum. Based on these large gaps in the literature, we first examined the continuous impact of anxiety symptoms on fertility in predicting the number of children, grandchildren, and great grandchildren. Notably, the modeling approach allowed for the examination of both linear and quadratic relationships between anxiety at all levels and later fertility, which was essential to test whether anxiety is related to fertility rates across a continuum. Given that sex can lead to differential impacts of traits on fertility (Jacobson, 2016), the current study also explored whether these impacts differed across males and females. Our research questions were: (1) does anxiety predict the later number of children, and is this relationship linear or quadratic?; (2) does anxiety predict the later number of grandchildren, and is this relationship linear or quadratic?; (3) does anxiety predict the later number of great-grandchildren, and is the relationship linear or quadratic?; (4) is the relationship between anxiety and the number of grandchildren mediated by the number of children?; (5) is the relationship between anxiety and the number of great-grandchildren mediated by the number of children and the number of great-grandchildren?; and (6) are the associations between anxiety and children, grandchildren, and great-grandchildren moderated by sex?

Method

Participants

The participants were recruited from the Longitudinal Study of Generations (Silverstein and Bengtson, 2016). This sample included a total of 300 three-generation families with 553 persons in the first generation (49.5% female, mean age 80.48 during anxiety assessment, *SD* $_{age}$ = 6.56, age range = 58-104, 86.8% Caucasian), 851 persons in the second generation (54.0% female, mean age 57.31 during anxiety assessment, *SD* $_{age}$ = 5.739, age range = 34 – 81, 62.8%

Caucasian), and 1,253 persons in the third generation (51.7% female, mean age 33.22, *SD* $_{age}$ = 4.000, age range = 16-54, 76.8% Caucasian). Because this was a multigenerational study at its origin, all participants in generation 1 were required to have at least 1 child and at least 1 grandchild. Generations 2 and 3 were not required to have children or grandchildren to participate in the study. The participants were randomly sampled from a subscriber list of 840,000 persons who were members of a health maintenance organization in California.

Participants were originally recruited as part of the study in 1971. However, their level of anxiety was assessed in 1985, and the number of children, grandchildren, and great-grandchildren was assessed in 1985, 1988, 1991, 1994, 1997, and 2000 (the last date that is publicly available). By the time of the last assessment, participants in the first generation averaged 95.48 years, participants in the second generation averaged 72.31 years, and participants in the third generation averaged 48.22 years.

Measures

Hopkins Symptom Checklist Anxiety Scale. The Hopkins Symptom Checklist anxiety scale is a 9-item scale that asks respondents to report how "nervous or shaky", "faint or dizzy", "trouble getting your breath", "fearful or afraid", "hot and cold spells", "tense or keyed up", "mind go blank", "check and double check what you did", and "easily annoyed or irritated" the participants were within the past week (Derogatis et al., 1974; Silverstein and Bengtson, 2016). Note that some of the items have been used in later psychometric scales, including the Symptom Checklist 90 (SCL-9), and these items have shown high convergent validity as they have been found to be related to anxiety and interpersonal sensitivity (*r* range = 0.56 – 0.91) (Bech et al., 2014), which is are predominant components of anxiety (Jacobson et al., 2013; Jacobson and Newman, 2014; Jacobson and Newman, 2016; Newman et al., 2017). Participants in generations

1, 2, and 3 responded to this scale in 1985. Responses were scored on a 1 – 5 Likert scale from not at all to extremely. In the present sample, the scale showed good internal consistency (α = 0.82).

Number of children, grandchildren, and great-grandchildren. All participants in generations 1-3 were asked about their children, grandchildren, and great-grandchildren. Participants were asked to list all of their children (and the number of children was recorded) in 1971; how many children have you had in 1985; the number of living biological children in 1991; and the number of living children in 1994, 1997, and 2000. Similarly, the number of grandchildren they had was asked in 1985, 1988, and 1991; the number of living grandchildren was asked in 1994; and the number of grandchildren was asked in 1997 and 2000. Lastly, the participants were asked how many great-grandchildren they had in 1988 and 1991; how many living great-grandchildren they had in 1994; and how many great-grandchildren they had in 1997 and 2000. Given the nature of the longitudinal study, the total number of children, grandchildren, and great-grandchildren throughout these periods was calculated by taking the maximum of all assessment points for the number of children, grandchildren, and great-grandchildren, respectively. Note that the maximum of the measurements was taken as not all participants completed all measurement occasions, and some participants' children had died after reaching reproductive age during later assessments.

Control variables. In the present sample, we controlled for the influence of age and generation. We also examined moderation by sex. Note that race/ethnicity was not controlled for given that it was mostly homogenous.[1]

Planned Analyses

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[1] Note that all results presented do hold when race is added as a control (which could likely be in part due to the homogeneity of the sample).

Descriptive statistics were using partial correlations (note that zero-order correlation coefficients were not appropriate here given that the current hypotheses involve a quadratic transformation, and higher order polynomials should not be interpreted in the absence of first order polynomials). Analyses were conducted using structural equation modeling (SEM), using the "lavaan" package in R using robust maximum likelihood (Gibson and Ninness, 2005).[234] Given the large sample size, practical model fit indices were used to evaluate the model fit (CFI, TLI, and RMSEA). Good fit was suggested to occur at CFI $\geq$ 0.950, TLI $\geq$ 0.950, and RMSEA $\leq$ 0.050. However, acceptable fit was considered when CFI $\geq$ 0.900, TLI $\geq$ 0.900, and RMSEA $\leq$ 0.080 (Brown, 2014; Marsh et al., 2004; Sharma et al., 2005). All missing data were handled using random forest multiple imputation, which represents a gold standard in handling missing data for non-linear effects (Ji et al., 2018).

A single SEM was created to test the first five hypotheses. Two latent variables were created, one of which represented anxiety and the second representing anxiety squared. For the anxiety term, all nine items freely loaded onto the anxiety factor, and, for the anxiety squared factor, all nine items were squared first and then freely loaded onto the anxiety squared factor. Notably, quadratic terms cannot be artifacts of common method variance(Siemsen et al., 2009).

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[2] Note that multilevel structural equation modeling was also estimated using Mplus 7 to account for the non-independence of the observations within families. The intraclass correlation coefficients suggested that the vast majority of the variance occurred within persons (rather than within families), with 87-99% of the variance reflecting within-person effects, rather than within family effects. The results are not presented as bootstrapped indirect effects are not available using multilevel structural equation modeling (and this is important as this represents the gold standard). Nevertheless, as a sensitivity analysis, all of the primary results remained statistically significant when accounting for non-independence using these multilevel structural equation models.

[3] Although robust maximum likelihood can account for deviations from normality, several sensitivity analyses were conducted where the outcomes (number of children, grandchildren, and great-grandchildren) were modeled using generalized linear models Poisson distributions and quasi-Poisson distributions (which allow over-dispersion). Additionally, multilevel generalized linear models using Poisson distributions were also modeled (accounting for nesting within families; multilevel generalized linear models do not have the quasi-Poisson distribution in R's lmer package). All the primary results remained significant regardless of the modeling strategy.

[4] Note that a sensitivity analysis was conducted to test if outliers might have influenced the results. We identified multivariate normality outliers via Cook's Distance (using the standard convention of four times the mean distance)- and removed both outliers and re-ran the models, and all of the primary results remained consistent.

These two latent factors (anxiety and anxiety squared) were then regressed upon the following manifest items: (1) the number of children, (2) the number of grandchildren, and (3) the number of great-grandchildren. Additionally, the number of grandchildren was predicted by the number of children, and the number of great-grandchildren was predicted by the number of children and the number of great grandchildren. All analyses controlled for generation and age.[5] As is common practice within SEM (e.g., Jacobson et al., 2018; Jacobson et al., 2017), note that the residuals between a given item and itself squared was allowed to correlate.

To test the mediational hypotheses (hypotheses 4 and 5), bootstrapped indirect effects were created using 10,000 bootstrapped samples (Valente et al., 2016). Lastly, a series of multi-group SEMs were utilized to examine hypothesis 6 (i.e. whether sex moderates the relationship between anxiety and children, grandchildren, or great-grandchildren). To do this, the paths for males and females were estimated simultaneously, and first allowed to vary. Next, equality constraints were specified to first test measurement invariance of the factor loadings for the anxiety scales, such that the loadings of the males and the females were constrained to be equal to test if this led to a significant difference in fit. Lastly, a series of equality constraints was specified to test whether the relationship between anxiety and children, grandchildren, and great-grandchildren resulted in a significant difference in fit.

Results

Descriptive Statistics

Partial correlations between the items are presented in Table 1. Note that the partial correlations suggested that anxiety had negative linear relationships and positive quadratic

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[5] Note that although generation was highly correlated with age, the variance inflation factor was exceptionally small (i.e. lower than 2), suggesting that the two control variables are not collinear with one another despite this high correlation. Nevertheless, as a sensitivity analysis, generation was removed and the primary results remained significant.

relationships with the number of children. The number of children was positively correlated with the number of grandchildren, and the number of grandchildren was positively correlated with the number of great-grandchildren. Older generations were associated with having more grandchildren and great-grandchildren.

Hypotheses 1-5: Anxiety and Children, Grandchild, and Great-Grandchildren

As mentioned in the planned analyses, a model including anxiety and anxiety squared in 1985 predicting the number of children, grandchildren, and great-great grandchildren was included in one SEM. The fit indices of the model suggested that the model had acceptable fit to the data $\chi 2$ (211) = 2,439.656 95%, CI [2,125.716, 2,910.215]; p < .001; CFI = 0.932, 95% CI [0.922, 0.940]; TLI = 0.919, 95% CI [0.907, 0.928]; and RMSEA = 0.062, 95% CI [0.057, 0.069]. See Figure 1 for a graphical depiction of the standardized model estimates.

The bootstrapped standardized total effects indicated that there was a significant negative linear relationship between anxiety and the number of children (β = -1.280, 95% CI [-1.567, -1.031]), and a significant positive quadratic relationship between anxiety and children (β = 1.114, 95% CI [0.861, 1.418]). The linear and quadratic relationships between anxiety and the number of children are depicted in Figure 2. Those at either end of the anxiety continuum were associated with having a greater number of children, and the effect sizes of these estimates are large (Preacher and Kelley, 2011).

Similarly, the bootstrapped standardized total effects suggested that there was a significant negative linear relationship between anxiety and the number of grandchildren (β = -0.541, 95% CI [-0.745, -0.362]), and a significant positive quadratic relationship between anxiety and the number of grandchildren (β = 0.277, 95% CI [0.205, 0.368]). The linear and quadratic relationships between anxiety and the number of grandchildren are depicted in Figure 3. Those at

either end of the anxiety continuum were associated with having a greater number of grandchildren, and the effect sizes of these estimates are small to medium (Preacher and Kelley, 2011).

Likewise, the bootstrapped standardized total effects suggested that there was a significant negative linear relationship between anxiety and the number of great-grandchildren (β = -0.267, 95% CI [-0.476, -0.061]), and a significant positive quadratic relationship between anxiety and the number of great-grandchildren (β = 0.210, 95% CI [0.026, 0.406]). The linear and quadratic relationships between anxiety and the number of great-grandchildren are depicted in Figure 4. Those at either end of the anxiety continuum were associated with having a greater number of great-grandchildren, and the effect sizes of these estimates are small (Preacher and Kelley, 2011).

The results of the bootstrapped standardized indirect effects also suggested that the relationship between anxiety and the number of grandchildren was mediated by the number of children, retaining both the significant mediational effect of the linear (β = -0.606, 95% CI [-0.754, -0.479]) and quadratic effects (β = 0.528, 95% CI [0.402, 0.679]). The results suggested that the mediational effect of the number of children explained 90.6% of the total variance between linear anxiety and the number of grandchildren. Likewise, the number of children explained 87.8% of the total variance between quadratic anxiety and the number of grandchildren.

The bootstrapped indirect effects of the two-path mediation also suggested that the relationship between anxiety and the number of great-grandchildren was mediated by both the number of children and the number of grandchildren for both the linear (β = -0.318, 95% CI [-0.411, -0.243]) and quadratic terms (β = 0.277, 95% CI [0.205, 0.369]). The results suggested

that the double-mediational pathway of both the number of children and the number of

grandchildren explained 86.3% of the total variance between the linear effect of anxiety and the

number of great-grandchildren. The double-mediational pathway between both the number of

children and the number of grandchildren explained 80.6% of the total variance between the

quadratic effect of anxiety on the number of great grandchildren.

Hypothesis 6: Moderation by Sex

Next, we tested the same structural model used to test hypotheses 1-5, but utilized a

multi-group estimation, such that males and females were estimated simultaneously, but the

model coefficients were allowed to freely vary separately for both males and females.

Moderation was then tested by constraining the male and female coefficients to be equal.

To test this, we first wanted to ensure that the factor loadings for anxiety did not differ

significantly between groups, and, consequently, we constrained all factor loadings to be equal

between the male and female groups. The fit indices suggested that the model with the

constrained loadings (model 2) did not fit significantly ($\chi2$ (18) = 19.729; p = .348) differently

from the model with the unconstrained loadings (model 1), suggesting that the factor loadings

did not significantly vary between males and females. Next, we similarly constrained all

regression paths to be equal across groups, and the results suggested that the model with the

constrained loadings (model 2) fit significantly ($\chi2$ (11) = 29.049; p = .002) better than the model

with all regression coefficients to be constrained across males and females (model 3).

Consequently, we then compared the unconstrained model systematically with one regression

coefficient at a time to find the regression coefficients that were significantly different between

males and females.

Constraining the linear effect of anxiety on the number of children (model 4) did not result in significantly ($\chi2$ (1) = 0.114; p = .736) different fit from the model with the constrained factor loadings but free regression coefficients (model 2), suggesting that the linear effect of anxiety on children did not differ significantly between males and females. We added an additional constraint to the prior model (model 4) and tested whether constraining the quadratic term of anxiety on the number of children to be equal between males and females would result in a difference in fit (model 5). Likewise, we found that there was no significant difference in fit ($\chi2$ (1) = 0.409; p = .522), suggesting that the quadratic effect of anxiety on the number of children did not vary between males and females.

We next constrained the regression on the number of children predicting the number of grandchildren to be equal across males and females (model 6) and found that there was no significant ($\chi2$ (1) = 0.011; p = .916) difference in fit compared to the prior model (model 5). We added an additional constraint, where the effect of the number of grandchildren on the number of great-grandchildren was constrained to be equal (model 7), and we found no significant ($\chi2$ (1) = 0.122; p = .727) reduction in model fit compared to the prior model (model 6). We then constrained the effect of date of birth on the number of children to be equal (model 8) and found a significant ($\chi2$ (1) = 8.643; p = .003) difference between males and females compared to the prior model (model 7), with the results suggesting that earlier dates of birth were significantly more positive for females than for males. Thus, we left this path unconstrained (model 7) and next tested the effect of generation on the number of children (model 9) and likewise found a significant ($\chi2$ (1) = 8.643; p = .003) difference in fit, with the results suggesting that the relationship between generation and the number of children was significantly more positive for males than females.

Thus, we also left this pathway unconstrained and next constrained the direct effect of anxiety on grandchildren to be equal across males and females (model 10) and found no significant ($\chi2$ (1) = 3.175; p = .075) difference in fit compared to the prior model (model 7). We subsequently constrained the direct quadratic effect of anxiety on the number of grandchildren to be equal across males and females (model 11) and found no significant ($\chi2$ (1) = 0.039; p = .843) difference in fit compared to the prior model (model 10). We added the direct effect of anxiety on the number of great-grandchildren to be equal across males and females (model 12) and found no significant ($\chi2$ (1) = 0.008; p = .929) difference in model fit compared to the prior model (model 11). We constrained the direct quadratic effect of anxiety on the number of great-grandchildren to be equal across males and females (model 13) and found no significant ($\chi2$ (1) = 3.305; p = .069) difference in model fit compared to the prior model (model 12).

We then constrained the direct effect of date of birth on the number of grandchildren between males and females (model 14) and found no significant ($\chi2$ (1) = -0.055; p > .999) difference in model fit compared to the prior model (model 13). We constrained the direct effect of the generation on the number of grandchildren to be equal between males and females (model 15) and found no significant ($\chi2$ (1) = 2.311; p = .128) difference in model fit compared to the prior model (model 14). We then constrained the direct effect of the date of birth on the number of great-grandchildren to be equal between males and females (model 16) and found no significant ($\chi2$ (1) = 0.000; p > .999) difference in fit compared to the prior model (model 15). We next constrained the direct effect of generation on the number of great-grandchildren between males and females (model 17) and found a significant ($\chi2$ (1) = 5.534; p = .019) difference in model fit compared to the prior model (model 16), where males had a significantly more positive association between the generation and the number of great-grandchildren than did

females. Lastly, we constrained the effects of the number of children predicting the number of

great-grandchildren to be equal between males and females (model 18) and found no significant

($\chi2$ (1) = 0.369; p = .544) difference in model fit compared to the prior model (model 16).

In sum, the multi-group models showed that there were no significant differences

between males and females in any of the substantive relationships of interest. The results showed

comparable effects of linear and quadratic effects of anxiety predicting the number of children,

number of grandchildren, and the number of great-grandchildren.

Discussion

In direct opposition to findings showing that anxiety is *psychologically* adaptive at mean

levels, long characterized as the Yerkes-Dodson law of arousal (Teigen, 1994), these findings

suggest that increases in the extremes in anxiety result in increased evolutionary fitness, whereas

values closer to the mean of anxiety result in decreased *evolutionary* adaptiveness. These

findings provide initial contrast to long-held speculations of clinical psychologists, who have

suggested that anxiety itself is also *evolutionarily* adaptive near mean levels (Barlow, 1988;

Bergstrom and Meacham, 2016; Cohen, 2011), whereby it acts as a stabilizing selection—

meaning normative levels are related to increased reproductive fitness and that the levels of a

given trait would remain stable across time (Schmalhausen, 1949). In contrast, these results

showed that values close to the mean in anxiety were associated with decreased evolutionary

fitness, and that values farther away from the mean resulted in increased fitness, providing initial

support for disruptive selection within an evolutionary context which would be associated with

the variance of a given trait changing across time.

Building on recent findings of a few studies indicating that disruptive selection may be

occurring regarding neuroticism, education, and fluid intelligence in humans (Eaves et al., 1990;

Sanjak et al., 2018) (note that other studies have failed to observe these relationships; e.g., Merz and Liefbroer, 2018; Penke and Jokela, 2016; Woodley of Menie et al., 2016), the current research supports that anxiety is undergoing disruptive selection within the present sample, and the findings were consistent across several generations. Notably, although these findings were robust across several generations which is a strong early evidence, this evidence should be considered preliminary as it often takes many generations to establish adaptive change at the phenotypic level (Hawks et al., 2007). Nevertheless, these findings may suggest an explanation for one of the potential conundrums regarding the paradox of seemingly common harmful levels of anxiety (Keller and Miller, 2006), that although these behaviors appear to be maladaptive in terms of their level of distress and social impairment (Wakefield, 1999), values particularly far from the mean are characteristic of increased fitness. This also seems to have implications towards some of the potential reasons that anxiety appears to vary considerably across persons within the population: the environment seems to be directly selecting for greater degrees of variability in anxiety.

Given that prior theories regarding the maintenance of psychopathology have suggested that psychopathology is likely either maintained by normative adaptation (Penke et al., 2007) or negative directional selection paired with mutations (Keller and Miller, 2006), the current findings suggest that in contrast extremes in anxiety may be under selective pressure within the current evolutionary environment. The mechanism underlying these associations nevertheless remains unknown. One plausible mechanism that could explain this attitude is the potential that extremes in anxiety could be related to having more children at younger ages. This notion is supported by some evidence given that extremely high levels of anxiety is associated with having more offspring at a younger age (Jacobson, 2016), characteristic of fast-spectrum behaviors (Del

Giudice, 2014). However, research has not yet examined whether this might also be characteristic of those with values that are extremely lower than the mean levels of anxiety. Nevertheless, given that psychopathy is associated with the inability to feel anxiety (Anestis et al., 2017; Lilienfeld et al., 2012; Sandvik et al., 2015), this would be exceptionally relevant to the extension of the current findings as psychopathy and related traits have been found to be positively related to fitness (Međedović et al., 2017; Neumann et al., 2012; Yao et al., 2014) and have been theorized to be related to having early sexual experiences at early ages (see Glenn et al., 2011 for greater detail). Thus, future work should also examine the impact of the inability to feel anxiety, a characteristic of psychopathy.

This research also has implications towards the classification of psychopathology itself. One of the most important conceptual distinctions that should be made is a vastly differing definition of "disorder" offered by evolutionary medicine, in contrast to the one posited by modern psychiatry. Modern psychiatry espouses the notion that psychiatric disorders are defined as a constellation of symptom characteristics (i.e. "syndrome") characterized by a "clinically significant disturbance in an individual's cognition, emotion regulation, or behavior that reflects a dysfunction in the psychological, biological, or developmental processes underlying mental functioning" (American Psychiatric Association, 2013). In contrast, modern evolutionary medicine offers a definition of disorder as a heritable trait that directly impairs one's ability to reproduce and one's offspring to reproduce (Abed and St John-Smith, 2016). Note that this quantitative conceptualization of a disorder is solely based on reproduction, rather than health, distress, or wellness, and contrasts the popular interpretation of evolutionary disease as a harmful dysfunction based on a qualitative disturbance of their "intended function" within the ancestral environment (Wakefield, 1999). Importantly, given that extreme levels of anxiety were found to

be positively related to reproductive success, and anxiety disorders are defined by atypically high levels of anxiety (American Psychiatric Association, 2013), this necessarily disputes the notion that these reflect evolutionary disorders from an evolutionary characterization of disease.

There are many plausible explanations for the present findings. In particular, given that neuroticism, education, and fluid intelligence both appear to have a moderate positive correlation with anxiety (Coplan et al., 2011; Scheier et al., 1994) and experimental evidence that progressive muscle relaxation inductions appear to be related to small decreases in one's ability to learn new information (Jacobson et al., 2011; Jacobson et al., 2013), it is plausible that the disruptive selective effects of anxiety on the number of children in later generations is in part due to the impact of neuroticism, education, and fluid intelligence (i.e. pleitropy) (Keller and Miller, 2006; Sanjak et al., 2018). Nevertheless, the construct of neuroticism is a combination of anxiety, depression, and anger; consequently suggesting that neuroticism somehow undermines the study of anxiety as a third variable relies on circular reasoning (see Jacobson and Newman, 2017 for a discussion of the problems of neuroticism as a confound to anxiety). Rather, neuroticism's relationship with fertility appears to be a partial corroboration of the impact of anxiety across another study. Future work should examine anxiety and both education and fluid intelligence simultaneously so as to determine which or all of the present variables both replicate with later generational effects and which present variables drive the disruptive selective pressures. Nevertheless, given that other studies have failed to find quadratic effects of neuroticism, intelligence, and education, more work is needed to determine if pleitropy reflects a plausible explanation of the current findings (Merz and Liefbroer, 2018; Penke and Jokela, 2016; Woodley of Menie et al., 2016).

An alternative explanation for the present findings may be a result of different

reproduction strategies based on one's life history. Those with high anxiety are more likely to

have poorer quality relationships with their parents (Manson, 2015) and poorer social

relationships with their peers (Jacobson et al., 2017; Jacobson and Newman, 2016; Manson,

2015). Consequently, those with high anxiety surrounding relationships may adopt a low-status

position based on poorer levels of functioning (Roche et al., 2018) and try to expend greater

energy on planning to have children at an earlier age. Supporting this position, evidence suggests

that rather than looking for casual relationships, persons with anxiety are often searching for

committed interpersonal relationships (Del Giudice and Belsky, 2010; Jackson and Kirkpatrick,

2007). Moreover, high anxiety is thought to lead to greater fertility planning (Morgan and King,

2001). Those with high anxiety are thought to adapt a fast-spectrum strategy (Del Giudice,

2018), and this may explain why those with high anxiety have more children at earlier ages

(Jacobson, 2016). Supporting the impact of the life history (Del Giudice, 2014), emotional

corrective experiences and increased awareness may lower anxiety and improve relationship-

quality (Newman et al., 2015; Roche et al., 2016b), and poor interpersonal relationship patterns

and even transient moments of high anxiety can maintain anxiety (Jacobson et al., 2016;

Newman et al., 2018). Thus, one's life history may impact the adoption of a fast-spectrum

planned strategy to have children at earlier ages among those with high anxiety, and this may be

in part motivated by some persons desiring to have children at earlier ages to lessen their own

anxiety (Lalos et al., 1985).

The influence of reproduction strategies may only be compounded by research on mate

choice, given that those with high anxiety perceive anxious partners as more similar to

themselves (Gee et al., 2012; Heimberg et al., 1985). Moreover, anxious persons are more

attracted to anxious partners, and persons with little anxiety may be attracted to persons with little anxiety (Frazier et al., 1996; Poire et al., 1997). Thus, paired with the prior discussed research, those with high anxiety may more often choose highly anxious mates and thereby increase the effects of any early reproduction strategies.

Although this early work points towards a continuum of disruptive selection which has implications towards the prevalence and distributions of these traits (Campbell and Reece, 2002; Rueffler et al., 2006), the implications of these selection effects on later prevalence and distribution of these traits across later generations remain unknown. Future studies should examine whether these selection advantages will lead to changing variability of anxiety in generations to come.

Although this study contains many strengths, it is not without limitations. Firstly, given the use of archival data, the study utilized a dated anxiety sub-scale. Nevertheless, the scale held high internal consistency, the items on the scale have shown high convergent and face validity, and some of the scale items continue to be used in modern measures today (Bech et al., 2014). Secondly, although this sample studied many generations, the first generation was required to have at least one child and grandchild at the origin of the study. Thus, the data from the first generation may necessarily decrease the potential range in the variance related to reproductive success given this demand characteristic. Future work should attempt to examine the continuous relationship between anxiety and fertility in a sample without such demand characteristics. Given that anxiety fluctuates within daily life and responds to one's environment (Frank et al., 2017; Roche and Jacobson, 2018; Roche et al., 2016a), future work should also examine the levels of anxiety across longer time periods to ensure that the observations are a result of trait, rather than state anxiety.

In sum, these early findings suggest that extreme values away from the mean are related to increased production of offspring. Moreover, the results suggest that beyond the immediate effect on one generation alone, the same quadratic relationship of anxiety on the production of offspring exists for the number of children, grandchildren, and great-grandchildren. This work suggests that the impact of anxiety on fertility may have implications for the distribution of anxiety in generations to come.

References

Abed, R., St John-Smith, P., 2016. Evolutionary psychiatry: A new College special interest group. BJPsych Bulletin 40(5), 233-236.

American Psychiatric Association, A.P.A., 2013. Diagnostic and Statistical Manual of Mental Disorders (DSM-5). American Psychiatric Association, Arlington, Virginia.

Anestis, J.C., Harrop, T.M., Green, B.A., Anestis, M.D., 2017. Psychopathic personality traits as protective factors against the development of post-traumatic stress disorder symptoms in a sample of national guard combat veterans. J. Psychopathol. Behav. Assess. 39(2), 220-229.

Barlow, D.H., 1988. Anxiety and its disorders: The nature and treatment of anxiety and panic. Guilford Press, New York, NY, US.

Baxter, A.J., Scott, K.M., Vos, T., Whiteford, H.A., 2013. Global prevalence of anxiety disorders: A systematic review and meta-regression. Psychol. Med. 43(5), 897-910.

Bech, P., Bille, J., Moller, S.B., Hellstrom, L.C., Ostergaard, S.D., 2014. Psychometric validation of the Hopkins Symptom Checklist (SCL-90) subscales for depression, anxiety, and interpersonal sensitivity. J. Affect. Disord. 160, 98-103.

Bergstrom, C.T., Meacham, F., 2016. Depression and anxiety: Maladaptive byproducts of adaptive mechanisms. Evolution, Medicine, and Public Health 2016(1), 214-218.

Brown, T.A., 2014. Confirmatory factor analysis for applied research. Guilford Publications, New York, NY.

Campbell, N.A., Reece, J.B., 2002. Biology. Benjamin Cummings.

Cohen, R.A., 2011. Yerkes–Dodson Law, in: Kreutzer, J.S., DeLuca, J., Caplan, B. (Eds.), Encyclopedia of Clinical Neuropsychology. Springer New York, New York, NY, pp. 2737-2738.

Coplan, J.D., Hodulik, S., Mathew, S.J., Mao, X., Hof, P.R., Gorman, J.M., Shungu, D.C., 2011. The Relationship between Intelligence and Anxiety: An Association with Subcortical White Matter Metabolism. Frontiers in Evolutionary Neuroscience 3, 8.

Dawkins, R., 1986. Puncturing punctuationism, in: Dawkins, R. (Ed.) The blind watchmaker: Why the evidence of evolution reveals a universe without design. WW Norton & Company, New York, NY, pp. 224-252.

Del Giudice, M., 2014. An evolutionary life history framework for psychopathology. Psychological Inquiry 25(3-4), 261-300.

Del Giudice, M., 2018. Life History Strategies, in: Del Guidice, M. (Ed.) Evolutionary Psychopathology: A Unified Approach. Oxford University Press, New York, NY, pp. 95-129.

Del Giudice, M., Belsky, J., 2010. The development of life history strategies: Toward a multi-stage theory, in: Buss, D.M., Hawley, P.H. (Eds.), The Evolution of Personality and Individual Differences. Oxford University Press.

Derogatis, L.R., Lipman, R.S., Rickels, K., Uhlenhuth, E.H., Covi, L., 1974. The Hopkins Symptom Checklist (HSCL): A self-report symptom inventory. Behav. Sci. 19(1), 1-15.

Eaves, L.J., Martin, N.G., Heath, A.C., Hewitt, J.K., Neale, M.C., 1990. Personality and reproductive fitness. Behav. Genet. 20(5), 563-568.

Eley, T.C., Bolton, D., O'Connor, T.G., Perrin, S., Smith, P., Plomin, R., 2003. A twin study of anxiety-related behaviours in pre-school children. Journal of Child Psychology and Psychiatry 44(7), 945-960.

Frank, B., Jacobson, N.C., Hurley, L., McKay, D., 2017. A theoretical and empirical modeling of anxiety integrated with RDoC and temporal dynamics. J. Anxiety Disord. 51, 39-46.

Frazier, P.A., Byer, A.L., Fischer, A.R., Wright, D.M., Debord, K.A., 1996. Adult attachment style and partner choice: Correlational and experimental findings. Personal Relationships 3(2), 117-136.

Gee, B.A., Antony, M.M., Koerner, N., Aiken, A., 2012. Appearing anxious leads to negative judgments by others. J. Clin. Psychol. 68(3), 304-318.

Gibson, S., Ninness, B., 2005. Robust maximum-likelihood estimation of multivariable dynamic systems. Automatica 41(10), 1667-1682.

Glenn, A.L., Kurzban, R., Raine, A., 2011. Evolutionary theory and psychopathy. Aggression and Violent Behavior 16(5), 371-380.

Gould, N.E.-S.J., 1972. Punctuated equilibria: an alternative to phyletic gradualism, in: Schopf, T. (Ed.) Models in Paleobiology. Princeton University Press, San Francisco, CA.

Hawks, J., Wang, E.T., Cochran, G.M., Harpending, H.C., Moyzis, R.K., 2007. Recent acceleration of human adaptive evolution. Proceedings of the National Academy of Sciences 104(52), 20753.

Heimberg, R.G., Acerra, M.C., Holstein, A., 1985. Partner similarity mediates interpersonal anxiety. Cognitive Therapy and Research 9(4), 443-453.

Jackson, J.J., Kirkpatrick, L.A., 2007. The structure and measurement of human mating strategies: toward a multidimensional model of sociosexuality. Evolution and Human Behavior 28(6), 382-391.

Jacobson, N.C., 2016. Current evolutionary adaptiveness of psychiatric disorders: Fertility rates, parent-child relationship quality, and psychiatric disorders across the lifespan. J. Abnorm. Psychol. 125(6), 824-839.

Jacobson, N.C., Chow, S.M., Newman, M.G., 2018. The Differential Time-Varying Effect

Model (DTVEM): A tool for diagnosing and modeling time lags in intensive longitudinal data.

Behav. Res. Methods.

Jacobson, N.C., Kramer, S., Tharp, A., Costa, S., Hawley, P., 2011. The effects of encoding in

hypnosis and post-hypnotic Suggestion on academic performance. Am. J. Clin. Hypn. 53, 247-

254.

Jacobson, N.C., Kramer, S.L., Tharp, A.G., Harmon, K.A., Cejas, G.P., Costa, S.C., 2013.

Deficits of encoding in hypnosis: A result of altered state of awareness. Am. J. Clin. Hypn.

55(4), 360-369.

Jacobson, N.C., Lord, K.A., Newman, M.G., 2017. Perceived emotional social support in

bereaved spouses mediates the relationship between anxiety and depression. J. Affect. Disord.

211, 83-91.

Jacobson, N.C., Newman, M.G., 2014. Avoidance mediates the relationship between anxiety and

depression over a decade later. J. Anxiety Disord. 28(5), 437-445.

Jacobson, N.C., Newman, M.G., 2016. Perceptions of close and group relationships mediate the

relationship between anxiety and depression over a decade later. Depress. Anxiety 33(1), 66-74.

Jacobson, N.C., Newman, M.G., 2017. Anxiety and depression as bidirectional risk factors for

one another: A meta-analysis of longitudinal studies. Psychol. Bull. 143, 1155-1200.

Jacobson, N.C., Newman, M.G., Goldfried, M.R., 2016. Clinical feedback about empirically

supported treatments for obsessive-compulsive disorder. Behav. Ther. 47, 75-90.

Ji, L., Chow, S.-M., Schermerhorn, A.C., Jacobson, N.C., Cummings, E.M., 2018. Handling

missing data in the modeling of intensive longitudinal data. Structural Equation Modeling.

Keller, M.C., Miller, G., 2006. Resolving the paradox of common, harmful, heritable mental

disorders: Which evolutionary genetic models work best? Behav. Brain Sci. 29(4), 385-404;

discussion 405-352.

Lalos, A., Jacobsson, L., Lalos, O., Schoultz, B., 1985. The wish to have a child. Acta Psychiatr.

Scand. 72(5), 476-481.

Lau, J.Y., Eley, T.C., Stevenson, J., 2006. Examining the state-trait anxiety relationship: a

behavioural genetic approach. J. Abnorm. Child Psychol. 34(1), 19-27.

Legrand, L.N., McGue, M., Iacono, W.G., 1999. A twin study of state and trait anxiety in

childhood and adolescence. The Journal of Child Psychology and Psychiatry and Allied

Disciplines 40(6), 953-958.

Lilienfeld, S.O., Patrick, C.J., Benning, S.D., Berg, J., Sellbom, M., Edens, J.F., 2012. The role

of fearless dominance in psychopathy: confusions, controversies, and clarifications. Personal

Disord 3(3), 327-340.

MacDonald, K., 1999. An evolutionary perspective on human fertility. Population and

Environment 21(2), 223-246.

Manson, J.H., 2015. Life history strategy and the HEXACO personality dimensions. Evol

Psychol 13(1), 48-66.

Marks, I.f., Nesse, R.M., 1994. Fear and fitness: An evolutionary analysis of anxiety disorders.

Ethology and Sociobiology 15(5), 247-261.

Marsh, H.W., Hau, K.-T., Wen, Z., 2004. In Search of Golden Rules: Comment on Hypothesis-

Testing Approaches to Setting Cutoff Values for Fit Indexes and Dangers in Overgeneralizing

Hu and Bentler's (1999) Findings. Structural Equation Modeling 11(3), 320-341.

Međedović, J., Petrović, B., Želeskov-Đorić, J., Savić, M., 2017. Interpersonal and affective

psychopathy traits can enhance human fitness. Evolutionary Psychological Science 3(4), 306-

315.

Merz, E.M., Liefbroer, A.C., 2018. Cross-national differences in the association between

educational attainment and completed fertility. Do welfare regimes matter? . Vienna Yearbook of

Population Research, 95-120.

Morgan, S.P., King, R.B., 2001. Why have children in the 21st century? Biological

predisposition, social coercion, rational choice. European Journal of Population 17(1), 3-20.

Nesse, R., 1999. Proximate and evolutionary studies of anxiety, stress and depression: Synergy at

the interface. Neurosci. Biobehav. Rev. 23(7), 895-903.

Nesse, R.M., 1987. An evolutionary perspective on panic disorder and agoraphobia. Evolution

and Human Behavior 8, 73-83.

Nesse, R.M., 2001. The smoke detector principle. Ann. N. Y. Acad. Sci. 935(1), 75-85.

Neumann, C.S., Schmitt, D.S., Carter, R., Embley, I., Hare, R.D., 2012. Psychopathic traits in

females and males across the globe. Behav. Sci. Law 30(5), 557-574.

Newman, M.G., Castonguay, L.G., Jacobson, N.C., Moore, G., 2015. Adult attachment as a

moderator of treatment outcome for GAD: Comparison between CBT plus supportive listening

and CBT plus interpersonal and emotional processing therapy. J. Consult. Clin. Psychol. 83, 915-

925.

Newman, M.G., Jacobson, N.C., Castonguay, L., 2013. Interpersonal and Emotion-Focused

Processing Psychotherapy for Generalized Anxiety Disorder, in: Emmelkamp, P.M., Ehring, T.

(Eds.), International Handbook of Anxiety Disorders: Theory, Research and Practice, Volume II.

Wiley Blackwell.

Newman, M.G., Jacobson, N.C., Erickson, T.M., Fisher, A.J., 2017. Interpersonal problems predict differential response to cognitive versus behavioral treatment in a randomized controlled trial. Behav. Ther. 48(1), 56-68.

Newman, M.G., Lafreniere, L.S., Jacobson, N.C., 2018. Relaxation-induced anxiety: Effects of peak and trajectories of change on treatment outcome for generalized anxiety disorder. Psychotherapy Research 68, 616-629.

Ogliari, A., Citterio, A., Zanoni, A., Fagnani, C., Patriarca, V., Cirrincione, R., Stazi, M.A., Battaglia, M., 2006. Genetic and environmental influences on anxiety dimensions in Italian twins evaluated with the SCARED questionnaire. J. Anxiety Disord. 20(6), 760-777.

Olatunji, B.O., Cisler, J.M., Deacon, B.J., 2010. Efficacy of cognitive behavioral therapy for anxiety disorders: A review of meta-analytic findings. Psychiatr. Clin. North Am. 33(3), 557-577.

Penke, L., Denissen Jaap, J.A., Miller Geoffrey, F., 2007. The evolutionary genetics of personality. European Journal of Personality 21(5), 549-587.

Penke, L., Jokela, M., 2016. The evolutionary genetics of personality revisited. Current Opinion in Psychology 7, 104-109.

Poire, B.A., Haynes, J., Driscoll, J., Driver, B.N., Wheelis, T.F., Hyde, M.K., Prochaska, M., Ramos, L., 1997. Attachment as a function of parental and partner approach-avoidance tendencies. Human Communication Research 23(3), 413-441.

Preacher, K.J., Kelley, K., 2011. Effect size measures for mediation models: quantitative strategies for communicating indirect effects. Psychol. Methods 16(2), 93-115.

Roche, M.J., Jacobson, N.C., 2018. Elections have consequences for student mental health: An accidental daily diary study. Psychol. Rep., 0033294118767365.

Roche, M.J., Jacobson, N.C., Phillips, J.J., 2018. Expanding the validity of the Level of Personality Functioning Scale Observer Report and Self-Report versions across psychodynamic and interpersonal pparadigms. J. Pers. Assess., 1-10.

Roche, M.J., Jacobson, N.C., Pincus, A.L., 2016a. Using repeated daily assessments to uncover oscillating patterns and temporally-dynamic triggers in structures of psychopathology: Applications to the DSM–5 alternative model of personality disorders. J. Abnorm. Psychol. 125(8), 1090-1102.

Roche, M.J., Jacobson, N.C., Roche, C.A., 2016b. Teaching strategies for personality assessment at the undergraduate level. J. Pers. Assess., 1-9.

Roest, A.M., Martens, E.J., de Jonge, P., Denollet, J., 2010. Anxiety and risk of incident coronary heart disease: A meta-analysis. J. Am. Coll. Cardiol. 56(1), 38-46.

Roff, D., 2012. Evolutionary quantitative genetics. Springer Science & Business Media, Montreal, Quebec, Canada.

Rueffler, C., Van Dooren, T.J., Leimar, O., Abrams, P.A., 2006. Disruptive selection and then what? Trends Ecol Evol 21(5), 238-245.

Sandvik, A.M., Hansen, A.L., Hystad, S.W., Johnsen, B.H., Bartone, P.T., 2015. Psychopathy, anxiety, and resiliency – Psychological hardiness as a mediator of the psychopathy–anxiety relationship in a prison setting. Personality and Individual Differences 72, 30-34.

Sanjak, J.S., Sidorenko, J., Robinson, M.R., Thornton, K.R., Visscher, P.M., 2018. Evidence of directional and stabilizing selection in contemporary humans. Proceedings of the National Academy of Sciences 115(1), 151.

Scheier, M.F., Carver, C.S., Bridges, M.W., 1994. Distinguishing optimism from neuroticism (and trait anxiety, self-mastery, and self-esteem): A reevaluation of the Life Orientation Test. J. Pers. Soc. Psychol. 67(6), 1063-1078.

Schmalhausen, I.I., 1949. Factors of evolution: the theory of stabilizing selection. Blakiston, Oxford, England.

Seehausen, O., Butlin, R.K., Keller, I., Wagner, C.E., Boughman, J.W., Hohenlohe, P.A., Peichel, C.L., Saetre, G.-P., Bank, C., Brannstrom, A., Brelsford, A., Clarkson, C.S., Eroukhmanoff, F., Feder, J.L., Fischer, M.C., Foote, A.D., Franchini, P., Jiggins, C.D., Jones, F.C., Lindholm, A.K., Lucek, K., Maan, M.E., Marques, D.A., Martin, S.H., Matthews, B., Meier, J.I., Most, M., Nachman, M.W., Nonaka, E., Rennison, D.J., Schwarzer, J., Watson, E.T., Westram, A.M., Widmer, A., 2014. Genomics and the origin of species. Nat. Rev. Genet. 15(3), 176-192.

Sharma, S., Mukherjee, S., Kumar, A., Dillon, W.R., 2005. A simulation study to investigate the use of cutoff values for assessing model fit in covariance structure models. Journal of Business Research 58(7), 935-943.

Siemsen, E., Roth, A., Oliveira, P., 2009. Common method bias in regression models with linear, quadratic, and interaction effects. Organizational Research Methods 13(3), 456-476.

Silverstein, M., Bengtson, V.L., 2016. Longitudinal Study of Generations, 1971, 1985, 1988, 1991, 1994, 1997, 2000, 2005 [California]. Inter-university Consortium for Political and Social Research (ICPSR) [distributor].

Simon, G., Ormel, J., VonKorff, M., Barlow, W., 1995. Health care costs associated with depressive and anxiety disorders in primary care. Am. J. Psychiatry 152(3), 352-357.

Swanson, W.J., Vacquier, V.D., 2002. The rapid evolution of reproductive proteins. Nat. Rev. Genet. 3(2), 137-144.

Szkodny, L.E., Jacobson, N.C., Newman, M.G., 2014. Generalized Anxiety Disorders, in: Gabbard, G.O. (Ed.) Gabbard's Treatments of Psychiatric Disorders, 5th Edition. American Psychiatric Publishing, Arlington, VA.

Teigen, K.H., 1994. Yerkes-Dodson: A law for all seasons. Theory & Psychology 4(4), 525-547.

Ulizzi, L., Gravina, M.F., Terrenato, L., 1981. Natural selection associated with birth weight II. Stabilizing and directional components. Ann. Hum. Genet. 45(2), 207-212.

Valente, M.J., Gonzalez, O., Miočević, M., MacKinnon, D.P., 2016. A note on testing mediated effects in structural equation models: Reconciling past and current research on the performance of the test of joint significance. Educational and Psychological Measurement 76(6), 889-911.

Wakefield, J.C., 1999. Evolutionary versus prototype analyses of the concept of disorder. J. Abnorm. Psychol. 108(3), 374-399.

Woodley of Menie, M.A., Reeve, C.L., Kanazawa, S., Meisenberg, G., Fernandes, H.B.F., Cabeza de Baca, T., 2016. Contemporary phenotypic selection on intelligence is (mostly) directional: An analysis of three, population representative samples. Intelligence 59, 109-114.

Yao, S., Långström, N., Temrin, H., Walum, H., 2014. Criminal offending as part of an alternative reproductive strategy: investigating evolutionary hypotheses using Swedish total population data. Evolution and Human Behavior 35(6), 481-488.

Yoshida, T., Jones, L.E., Ellner, S.P., Fussmann, G.F., Hairston, N.G., 2003. Rapid evolution drives ecological dynamics in a predator-prey system. Nature 424(6946), 303-306.

Table 1

Partial Correlations Matrix of Study Variables

Variable	1.	2.	3.	4.	5.	6.	7.
1. Anxiety	1.000						
2. Anxiety^2	0.978*	1.000					
3. Number of children	-0.258*	0.234*	1.000				
4. Number of grandchildren	0.016	-0.017	0.561*	1.000			
5. Number of great-grandchildren	0.006	-0.009	-0.171*	0.459*	1.000		
6. Year of Birth	-0.061*	0.060*	0.029	-0.017	-0.007	1.000	
7. Generation	0.016	-0.020	-0.015	-0.094*	-0.156*	0.918*	1.000
M	0.000	0.000	2.731	4.301	2.330	1934.300	2.263
SD	0.789	0.789	1.889	4.729	4.505	19.118	0.781

Note. This table contains the partial correlations, means, and standard deviations of the primary study variables. Anxiety and anxiety^2 represent the saved factor scores of the latent variables. Because the quadratic term should not be interpreted in the absence of a linear effect in the model, partial correlations, rather than zero-order correlations have been presented.
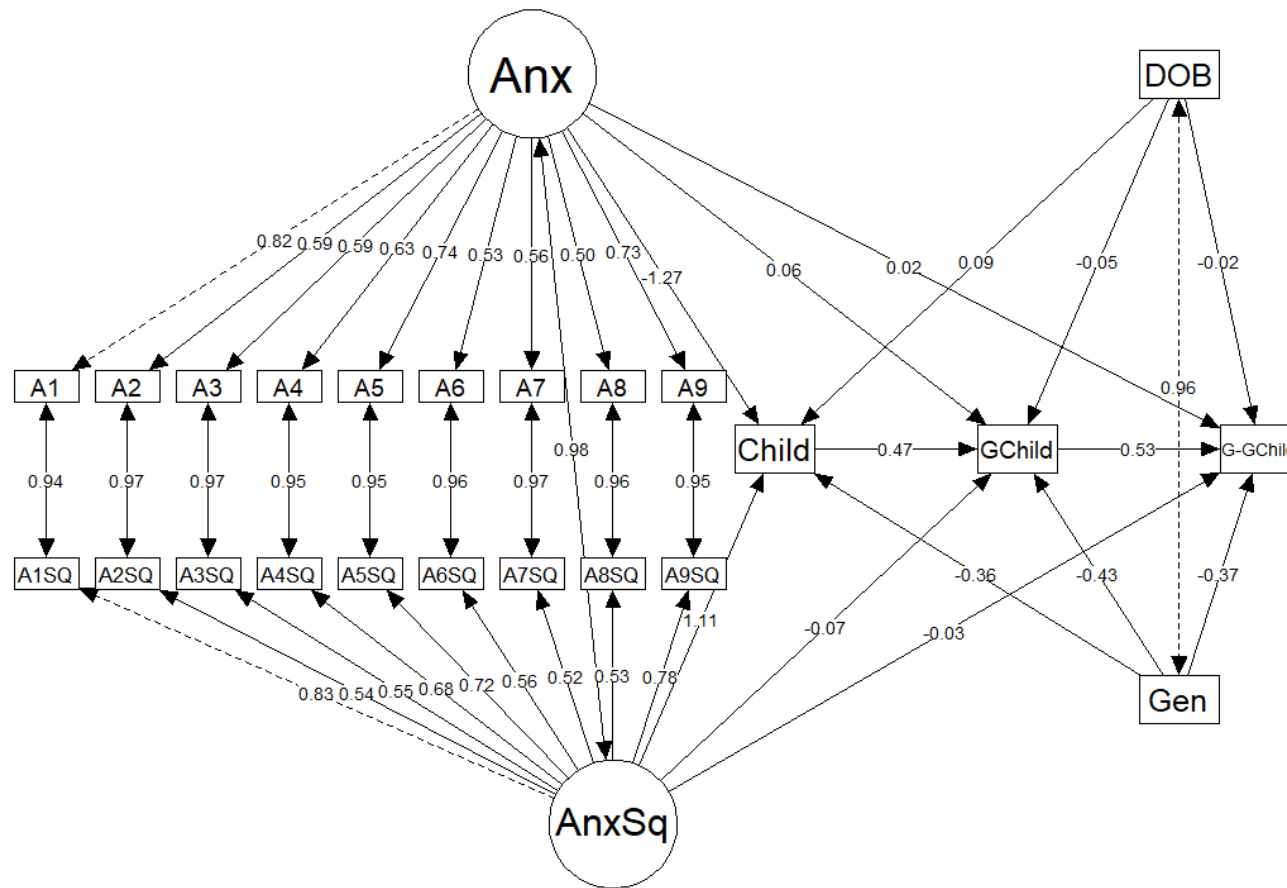
Figure 1. These graphs depict the standardized estimates of the primary structural equation model. Note that residual variances and intercepts in the model are not depicted due to spatial constraints. The terms Anx and AnxSq represent anxiety and anxiety^2 measured in 1985, respectively. Similarly, the terms Child, GChild, and G-GChild represent the total number of children, grandchildren, and greatgrandchildren up until the year 2000, respectively. Note that terms DOB and Gen represent the control variables of the date of birth and the generation, respectively. Solid lines represent the significant associations, and dashed lines represent the non-significant (or fixed) associations.
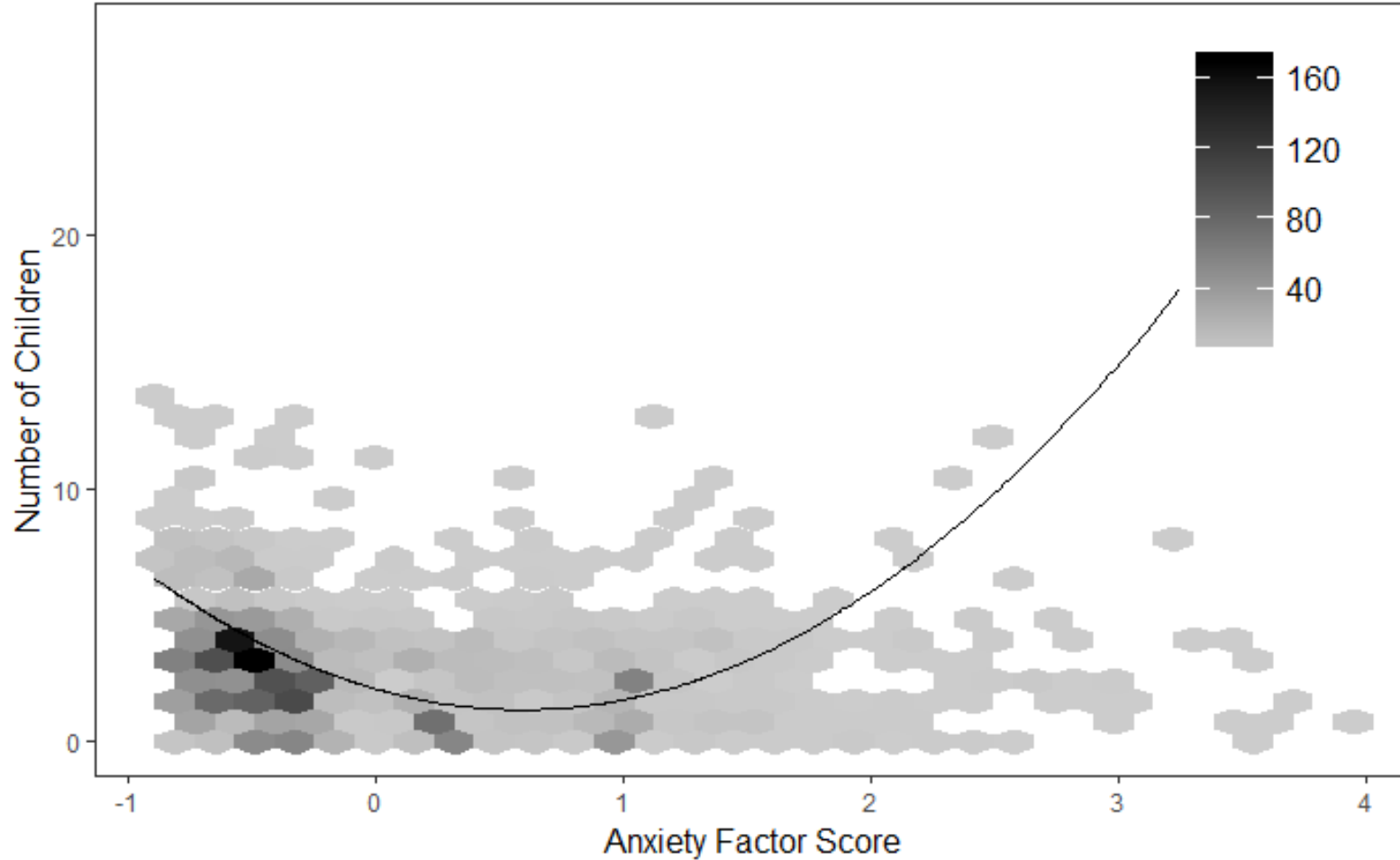
Figure 2. These graphs show both the hex-binned scatterplot between the anxiety factor score measured in 1985 and the number of children up until the year 2000, with the estimated linear and quadratic relationship based on the model estimates. As demonstrated in the model fit statistics, those at either extreme in the continuous level of anxiety are associated with increased numbers of children.
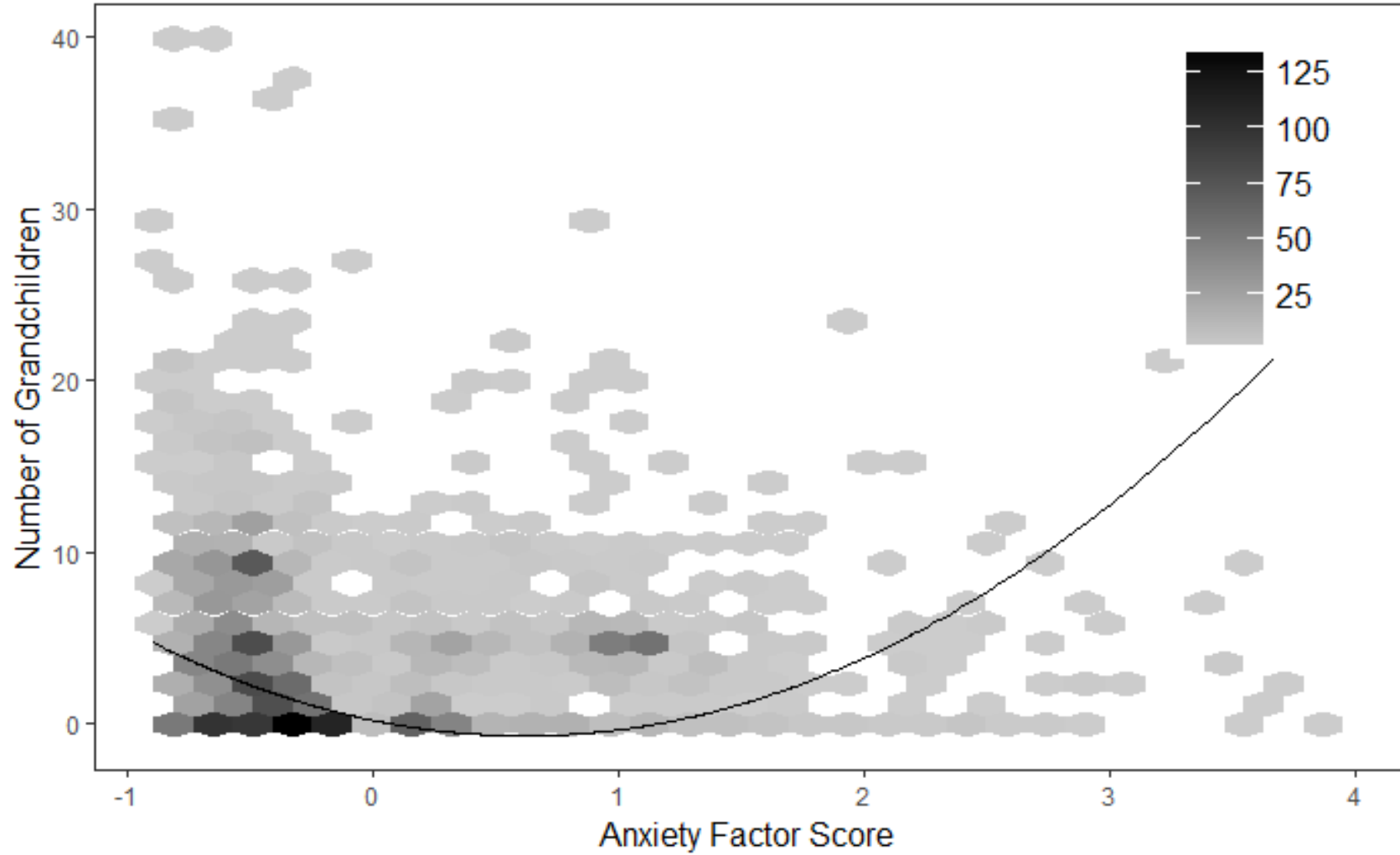
Figure 3. These graphs show both the hex-binned scatterplot between the anxiety factor score measured in 1985 and the number of grandchildren up until the year 2000, with the estimated linear and quadratic relationship based on the model estimates. As demonstrated in the model fit statistics, those at either extreme in the continuous level of anxiety are associated with increased numbers of grandchildren.
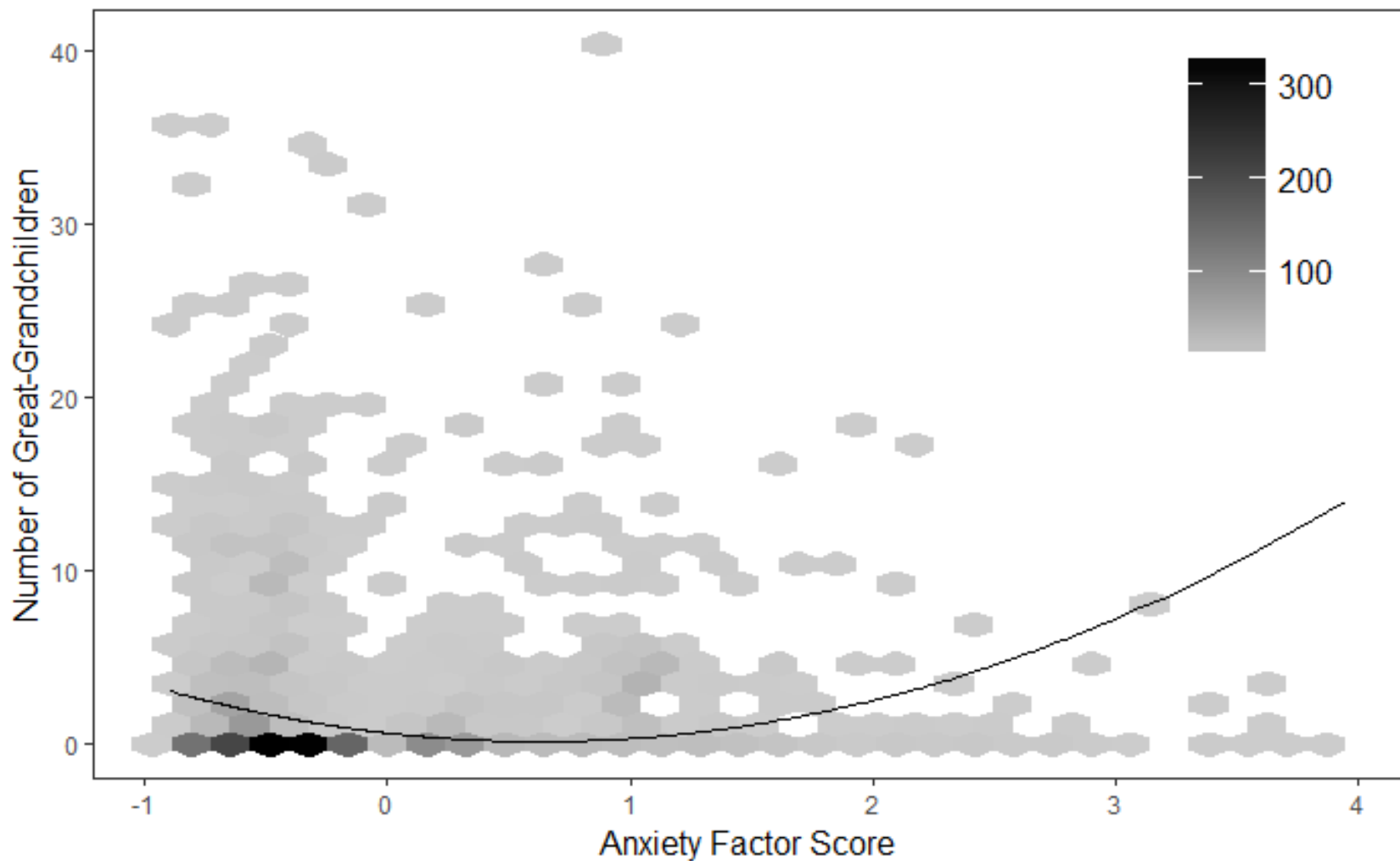
Figure 4. These graphs show both the hex-binned scatterplot between the anxiety factor score measured in 1985 and the number of great-grandchildren up until the year 2000, with the estimated linear and quadratic relationship based on the model estimates. As demonstrated in the model fit statistics, those at either extreme in the continuous level of anxiety are associated with increased numbers of great-grandchildren.