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Are testosterone levels and depression risk linked based on partnering and parenting? Evidence from a large population-representative study of U.S. men and women

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ABSTRACT

Partnered adults tend to have lower risks of depression than do single individuals, while parents are more commonly depressed than non-parents. Low testosterone men, and possibly women, are also at greater risk of depression. A large body of research has shown that partnered parents have lower testosterone than single non-parents in some cultural settings, including the U.S. Here, we drew on a large (n = 2438), U.S.-population representative cohort of reproductive aged adults (age: 38.1 years ± 11.1 SD) to test hypotheses regarding the intersections between partnering and parenting, testosterone, socio-demographic characteristics, and depression outcomes. Men and women's depression prevalence did not vary based on testosterone. Partnered fathers had lower testosterone than single (never married, divorced) non-fathers, but were less commonly depressed than those single non-fathers. Partnered mothers had reduced testosterone compared to never married and partnered non-mothers. Never married mothers had higher depression prevalence and elevated depressive symptomology compared to partnered mothers; these differences were largely accounted for by key health-related covariates (e.g. cigarette smoking, BMI). We found significant three-way-interactions between socioeconomic status (SES), testosterone, and parenting for adults' depression risks. High testosterone, high SES fathers had the lowest prevalence of mild depression, whereas low testosterone, low SES non-fathers had the highest. Compared to other mothers, low SES, low testosterone mothers had elevated prevalence of mild depression. Overall, low SES, high testosterone non-mothers had substantially elevated depression risks compared to other women. We suggest that psychobiological profiles (e.g. a male with low testosterone) can emerge through variable psychosomatic and psychosocial pathways and the net effect of those profiles for depression are influenced by the social (e.g. partnering and parenting status; socioeconomic gradients), cultural (e.g. gender and family life domains), and ecological (e.g. the lived environment, particularly related to low SES and poverty) contexts in which individuals find themselves.

1. Introduction

Past research demonstrates that married individuals tend to have lower risks of depression compared to their single and divorced counterparts (Lamb et al., 2003; Pearlin and Johnson, 1977). However, parenthood, when modeled similarly as a longer-term social role, is not comparably protective against depression. Rather, evidence indicates that parents residing with dependent children experience more depression than individuals who are childless (Evenson and Simon, 2005). While parenting often comes with many emotional benefits, heightened depression risk among mothers and fathers likely occurs due to some combination of physical, emotional, and psychological stressors that also accompany parenthood. Such strains can include: reduced sleep or impaired sleep quality, role overload and psychosocial stress from dual domestic and professional responsibilities, economic worries, and decreased marital relationship satisfaction. These challenging parenting dynamics may be particularly exacerbated in contexts such as single parenthood or for impoverished families (Evenson and Simon, 2005; Durette et al., 2011; Gray and Anderson, 2010; Garfield et al., 2006, 2014; Kim and Swain, 2007; Gallo and

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Matthews, 2003).

Moreover, it is estimated that up to 19% of mothers experience post-partum depression in the months after their babies are born (O'Hara and McCabe, 2013), and recent research has also shown that post-partum depression among fathers is much more common than previously thought, with up to 10% of new fathers experiencing depressive symptoms in the U.S. (Garfield et al., 2006, 2014; Ramchandani et al., 2005; Kim and Swain, 2007). In light of the human toll that depressive symptoms and depression conditions exact through diminished quality of life and elevated mortality risks through suicide as well as multi-generational pathways via effects on child development (O'Hara and McCabe, 2013; Ramchandani et al., 2005), the social and biological pathways through which depression-protective effects or risk factors emerge remain a prominent public health concern.

A large number of studies have shown that partnered men and fathers have lower testosterone (T) than single non-fathers in many settings around the world. A smaller number of studies have found similar patterns among partnered women and mothers (Gettler, 2014; Gray and Anderson, 2010; Rilling, 2013; Trumble et al., 2015; van Anders et al., 2011; van Anders, 2013). Despite such results and the growing body of research on T and partnering-parenting, there has been little explicit research, to date, testing whether variation in T based on life history status has implications for mental health (Kim and Swain, 2007). In particular, this perspective has not been applied to prevalence of depression, which has been linked to low T in men and (preliminarily) in women, though results vary. This research gap is particularly noteworthy given the variation in depression risks between married and non-married individuals and parents versus non-parents as well as widespread research interest in parental post-partum depression and its impacts on child development.

Much of the psychobiological research on partnering, parenting, and T is grounded in integrative perspectives that bring to bear evolutionary, cultural, and family system principles to frame questions regarding how human biological plasticity interfaces with social context. Neuroendocrine and behavioral research indicates that elevated T contributes to reduced prosociality and heightened competitive behaviors as well as elevated attention to threat and dominance challenges and (potentially) reactive aggression (Carré and Olmstead, 2015; Gettler, 2014; Gray and Anderson, 2010; Rilling, 2013; Trumble et al., 2015; van Anders et al., 2011; van Anders, 2013). Thus, in many cultural contexts in which partnering and parenting include nurturant, sensitive bonding and interactions, those relationships are associated with lower T, on average (Gettler, 2010, 2014; Gray and Anderson, 2010; Rilling, 2013; van Anders et al., 2011; van Anders, 2013). While reduced T among men and women might contribute to nurturant, sensitive parenting and empathetic social relationships in some settings, multiple studies have found that men with low T are at increased risk of depression (reviewed in Ebinger et al., 2009), which may have implications for paternal post-partum depression (Kim and Swain, 2007). While connections between depression and T have been less thoroughly examined in females, there is some preliminary evidence indicating that low T women might likewise have heightened depression risk (Giltay et al., 2012; cf. Weber et al., 2000).

An anthropological perspective on the intersections between these socio-demographic and psychobiological mediators of mental health and familial function will prove highly informative. Complementing existing psychobiological frameworks (van Anders et al., 2011; van Anders, 2013), an anthropological lens will shed light on the social and structural experiences that can affect biological function (i.e. the hypothalamic-pituitary-gonadal [HPG] axis that produces T), leading to the observation that the pathways that contribute to lower T or higher T production are context-dependent (Gettler, 2014, 2016; Gray and Anderson, 2010; Trumble et al., 2015; van Anders et al., 2011; van Anders, 2013). Alongside these biocultural perspectives and prior social science-based observations that depression risks based on partnering and parenting are variable and dependent on factors such as age and class, models that incorporate aspects of gender socialization (e.g. masculinity concepts) and gender norms (e.g. disproportionate parenting burdens falling on mothers) will make novel contributions to our understandings of the etiology of depression risks within- and across-populations.

Drawing on such a perspective, we analyzed cross-sectional data from a sample of reproductive aged men and women (mean age: 38.1 years ± 11.1 [SD]) who participated in the 2011–2012 National Health and Nutrition Examination Survey (NHANES), which is representative of the U.S. population, to shed further light on the intersections between life history status, T, and depression risks (n = 2438; 1505 = male). We tested hypotheses regarding the correlations between adult men and women's T and depressive symptoms (respectively) based on partnering and parenting status. Based on past U.S.-based results, we hypothesized that partnered parents would have lower T than single individuals. We hypothesized that partnered parents would report higher levels of depressive symptoms compared to partnered non-parents, but lower levels than single adults.

We then tested whether T acted as a moderating variable for links between partnering/parenting and depression. We specifically hypothesized that single adults who were not partnered or parents but who had low T would report higher levels of depression, whereas low T would not be a risk factor for depression in the context of partnering or parenting. Finally, based on the idea that the etiology or pathways to low T (and hence the implications for depression) are different for partnered adults and parents (e.g. compared to other single non-parents) and that social support (such as partnering) can buffer against adverse effects of deprivation and psychosocial stress, we tested for three-way interactions between partnering/parenting, T, and two socio-demographic variables (socioeconomic status [education level] and age). These factors, which affect depression risk and relate to variable T production, could contribute to divergent depression profiles in parents versus non-parents and partnered versus single adults.

2. Methods

2.1. NHANES 2011–2012

The NHANES studies are an ongoing series of data collections through the U.S. Center for Disease Control (CDC) that aim to assess health outcomes for a sample that is representative of the civilian, non-institutionalized U.S. population. Here, we focus on the 2011–2012 cross-sectional wave of the NHANES continuous data collections. We analyzed data from U.S. men and women between the ages of 20 and 60 years. This age range spans the period from young adulthood to the age at which the overwhelming majority of U.S. men and women have ceased having biological children (Martin et al., 2015). In total, 2438 subjects (1505 = male; 933 = female [non-pregnant, non-menopausal]) had full demographic, health, and hormonal data for inclusion in our analyses. We present descriptive statistics for the sample, stratified for males and females, in Table 1. See the Supplemental Methods file for further details on the NHANES study design and our selection criteria for men and women in this analysis.

2.2. Depression symptomology

For subjects 18 years of age and older, NHANES administers the