An Exploration and Cross Cultural Comparison of Mental Health Outcomes Associated with Single Parenthood

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Dedication and Acknowledgements

When I boarded the plane to America on August 25th 2005, it was hard to imagine that all of my wildest dreams would come true. At every step of my journey I was lucky enough to be supported by extraordinary people who helped me meet my goals, and who accompanied me to this moment. My dissertation is dedicated to you!

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Abstract

Single parenthood is a risk factor for mental health deficits, an effect that may be exacerbated by circumstances such as socioeconomic status (SES) or number of children in the household. To date, however, there are no genetically informed studies examining the association between single parenthood and mental health, which may have non-causal, family-level confounds. The present study used male and female pairs of MZ and DZ twins from United States (the Washington State Twin Registry) and Swedish (the Study of Twin Adults: Genes and Environment) datasets to examine the causality of the relation between single parenthood and several mental health outcomes. We controlled for genetic and shared environmental confounds as we reexamined whether this association is consistent with a causal explanation. We then investigated whether this effect is modified by age, education level, income level, and number of children. We found a quasi-causal effect of single parenthood on depression and anxiety, such that in all analyses single mothers experienced higher levels of depression and anxiety than their partnered counterparts. This effect was mediated by SES in the US, whereas in Sweden it was present even after controlling for SES confounds. Single mothers were worse off than single fathers in all analyses. No effects of single parenthood on perceived stress or coping were found in our samples. Our results suggest a nuanced picture of the effect of single parenthood on mental health outcomes. Sociopolitical climates and economic differences do not fully account for mental health disparities between single and partnered mothers across cultures.

Introduction

General background

Since the 1950s the West has experienced a steady rise in the number of single parent families, influenced by the continued surge of divorce rates coupled with the increased prevalence of out of wedlock childbirth (Bianchi, 1994; Casper & Bianchi, 2002).While in 1970 81% of all family households in the United States were headed by two married parents, by 2012 the number dropped to about 66% (Vespa, Lewis, & Kreider, 2013). This trend is problematic because becoming a parent is not without its health challenges: single and partnered parents alike report more depression (Evenson & Simon, 2005; Malik & Irshad, 2012), anxiety (Propst, 1986), and disease severity (Frank, 1985) than nonparents. However, married or cohabiting (i.e. partnered) parents are able to share their burdens with their partners. Research has found that sharing stressors with a partner alleviates their emotional effect (Kiecolt-Glaser & Newton, 2001), and even holding a loved one's hand while experiencing physical pain renders the pain more manageable (Coan, Schaefer, & Davidson, 2006). In the absence of a spouse, a single parent is left facing challenges without the buffering effect that a partner might provide, which may lead to their experiencing enhanced versions of the negative effects of parenthood.

The present manuscript is a genetically informed, multi-level analysis of the ways in which single parenthood is associated with specific mental health symptoms within and across cultures. We begin by reviewing research that examines single parenthood and health outcomes. The literature review is heavily focused on single motherhood and depression, as there is comparatively little research on other mental health outcomes related to single motherhood, and even less on single fatherhood. We consider correlates of single parenthood and mechanisms through which single parenthood might be associated with detrimental health outcomes. Due to

the relative scarcity of research concerning outcomes other than depression, we will focus on depression as our primary outcome of interest. We will treat our analyses of other psychological health outcomes of interest as exploratory.

An association between single parenthood and negative health outcomes has been found in numerous studies using non-experimental data and traditional methods (i.e. regression). However, these methods cannot account for selection effects and thus do not fully explain the association between single parenthood and health outcomes such as depression or anxiety. Considering the possibility of nonrandom selection (i.e. an individual has certain characteristics that lead him to experience a negative health outcome and these characteristics also impact his remaining single or getting divorced) versus causal processes (i.e. remaining single or getting divorced has direct causal effects on health outcomes) as mechanisms through which the two phenotypes are connected is an integral component to the development of more effective preventative and support measures. Thus, we will introduce a powerful approach for studying behavioral data, and one that has never been used in the study of single parenthood: twin research.

The use of twin data controls for genetic and shared environmental confounds that cannot normally be controlled in cross-sectional data, and brings us closer to a causal interpretation of our results. For example, if a female MZ (i.e., identical, monozygotic) twin who is a single parent is more depressed than her married parent co-twin, that difference cannot be accounted for by genetic confounds, because identical twins share essentially all of their genes. This difference also cannot be explained by shared environmental influences, which are by definition *all* of the experiences that the twins share as children. Thus, any observed difference in their depression

levels is a result of nonshared environment, which is the totality of experiences they do not share, and includes their difference in parenthood status.

The first goal of the proposed study is to use twin data to determine the extent to which selection accounts for the association between single parenthood and depression. A secondary, related goal is to explore the relation between single parenthood and other specific negative mental health outcomes such as anxiety. An important contribution made by the present study is the inclusion of cohabiting parents in the “partnered” parent category. Prior research has focused on the distinction between marital and "non-marital" states (e.g., divorce or singlehood). Throughout the developed world, and increasingly in the U.S., cohabitation is becoming more common as a step towards—and in some cases an alternative to—legal marriage (Cherlin, 2010). It is critical to extend current findings by building a knowledge base that adequately reflects societal shifts in union formation.

A second question of interest is whether different national policy regimes reduce or enhance that relationship. A cross-cultural comparison will be conducted between the United States (US) and Sweden, two countries whose vastly different policies aimed at single parents are examined and contrasted. These differences will allow the study of "gene-culture" interactions (a basic form of gene-environment interaction) through the examination of how genetic influences on single parenthood differ across social, cultural, and policy contexts. The difference in environmental influences creates the exciting possibility of observing heritability estimate differences across cultures as well as within them. While several welfare regime comparisons have been conducted with the aim to assess the outcomes of single parents in various countries, this is the first of its kind, made unique by the use of twin data which allows for a more nuanced analysis and interpretation of the findings.

Research investigating single parenthood using traditional methods

Much research links family structure to individual psychological, social, and economic well-being (Afifi, Cox, & Enns, 2006; Crosier, Butterworth, & Rodgers, 2007; Robinson, Magee, & Caputi, 2014). Forty-one percent of children in the U.S. are born outside of marriage, and nearly half of all marriages end in divorce (Copen, Daniels, Vespa, & Mosher, 2012). While arrangements like joint custody are increasing, we know that 80% of children in single parent families are still raised primarily by their mothers (Grall, 2009). In 2012, 24% of children under the age of 17 were living with only their mothers, and 4% who were living with only their fathers (Mykyta & Macartney, 2012).

Compared with married mothers, single mothers report more mental health problems and lower well-being (Brown & Moran, 1997; Cairney, Boyle, Offord, & Racine, 2003; Davies, Avison, & Mcalpine, 1997; Wade, Veldhuizen, & Cairney, 2011), and more physical health issues (Baker & North, 1999; Broussard, Joseph, & Thompson, 2012; Floderus, Hagman, Aronsson, Marklund, & Wikman, 2008; Fritzell, Ringbäck Weitoft, Fritzell, & Burström, 2007; Sarfati & Scott, 2001; Young, Cunningham, & Buist, 2005). While demographically less common, single fathers also report increased emotional distress, more symptoms of depression and anxiety, and lower self-esteem than married fathers (Campbell & Pike, 2002; Clarke-Stewart & Bailey, 1989; Stewart, 1986). Given the prevalence of these findings, it is critical to better understand the mechanisms through which single parenthood is associated with mental health outcomes. It is noted that the studies reviewed here, as well as most current literature, compare single with married parents, whereas our study treats married and cohabiting parents equivalently for comparison purposes.

Health risks associated with single parenthood

Depression is by far the most commonly investigated mental health outcome associated with single motherhood. Research shows that women have an almost doubled risk of depression when compared to men (Atkins, 2010), even after controlling for age, education, and employment (Clarke-Stewart & Bailey, 1989; Collings, Jenkin, Carter, & Signal, 2013). Single mothers are at an even higher risk when compared to their married counterparts: they are twice (Wang, 2004) to three times (Cairney, Boyle, Lipman, & Racine, 2004; Cairney & Wade, 2002; Collings et al., 2013; Colton, Janzen, & Laverty, 2015) as likely to report depressive symptoms and/or clinical depression, a finding that has been replicated everywhere from the US to Europe, Canada, and Australia (Cairney et al., 2003; Colton et al., 2015; Crosier et al., 2007; Robinson et al., 2014; Sperlich, Arnhold-Kerri, & Geyer, 2011a; Wang, 2004). While less commonly reported in research, single mothers also endorse lower self-rated mental health (Colton et al., 2015), decreased perceived social support and increased parenting stress (Osborne, Berger, & Magnuson, 2012), and they have a higher likelihood of anxiety and substance use disorders (Crosier et al., 2007; Wang, 2004) that married mothers.

Single fathers are less common as a group, and research studies usually have trouble gathering a large enough sample size to adequately describe them, even in large representative datasets (Chzhen & Bradshaw, 2012). Nevertheless, they are also reported to experience negative outcomes associated with their parenthood status. Compared to partnered fathers, single fathers report increased depression, anxiety, and lower self-esteem (Campbell & Pike, 2002; Clarke-Stewart & Bailey, 1989; Stewart, 1986). Being a single father predicts acute psychological distress after the birth of a child (Skari et al., 2002), and puts single fathers at greater risk for psychiatric and substance use disorders (Wade et al., 2011). Divorced fathers are more depressed

than their married counterparts (Bronte-Tinkew, Moore, Matthews, & Carrano, 2006; Spector, 2006), and if they are the nonresident parent they also report lower levels of emotional well-being (Bokker, Farley, & Bailey, 2006).

Possible explanations for outcome differences between single and partnered parents

There are many possible reasons why single parents may be at higher risk of experiencing negative health outcomes compared to their partnered counterparts. Research has especially focused on differences in socioeconomic status (SES) and demographic characteristics as explanations for the observed differences in health outcomes between single and partnered mothers. When it comes to single fathers, it appears that the mechanisms are slightly different: research does not find income or demographic characteristics to be significant predictors, and instead suggests social support and a sense of loss as possible explanations.

Economic hardships were found to be more common in single-parent families and the resulting strain was associated with an increased vulnerability to depression (Cooper et al., 2008; Crosier et al., 2007). This is explained by considering the resources that better financial status can provide, and that can enhance health and well-being: better housing, better nutrition, and better access to health services (Colton et al., 2015). However, the correlation between single parenthood and lower income applies more to mothers than to fathers. A full-time job leads to a higher income, which is beneficial to single mothers' mental health (Evenson & Simon, 2005), and staying employed can decrease their risk of depression by about 3% a month (Zabkiewicz, 2010). However, that is not enough, as even single mothers who are employed full time may be more likely to be in financial hardship that partnered mothers, and thus at higher risk for depression (Brown & Moran, 1997).

Age and race are correlates of the association between single motherhood and depression, with younger women being more predisposed to depression than older women, (J. D. Brown, Harris, Woods, Buman, & Cox, 2012; Horwitz, Briggs-Gowan, Storfer-Isser, & Carter, 2007), and single mothers of color having a higher risk of depression than white mothers (Wang, 2004). The number of children is an inconclusive predictor of maternal mental health: some researchers suggest that more children correspond to an increase in risk for depression (Horwitz et al., 2007; Sperlich, Arnhold-Kerri, & Geyer, 2011b), while others did not find number of children to be a predictor of depression (Hilton & Kopera-Frye, 2006).

Whereas financial strain is an often-quoted mechanism linking single motherhood and detrimental health outcomes, single fathers are better off in their employment situations than single mothers (Clarke-Stewart & Bailey, 1989), and are generally exposed to lower levels of socioeconomic deprivation than single mothers (Collings et al., 2013). Instead, their heightened risk for negative health outcomes is related to the loss of their partner, child, and former life.

As mentioned earlier, an overwhelming majority of mothers have custody over their children (Evenson & Simon, 2005). This means, in most cases, that the noncustodial father is deprived of contact with the child, compared to a custodial parent. Stewart et al. (1986) found that after a divorce, the noncustodial father suffers from a sense of loss, be it related to his partner, financial standing, social status, or child (Stewart, 1986). Having limited contact with their child increases the father's emotional distress, and not having custody is found to be detrimental to the father's mental health (Bokker et al., 2006; Campbell & Pike, 2002). While custodial fathers also report depressive symptoms and less happiness, some research suggests that they in fact experience positive adjustment after a divorce and the depression that manifests itself might stem from the loss of their partner, not from the strain of parenting (Stewart, 1986).

Indeed, custodial fathers, the majority of whom are employed, might garner additional self-confidence from mastering household tasks (Campbell & Pike, 2002; Hetherington, Cox, & Cox, 1976; Stewart, 1986). More research is needed to describe this understudied group, and to reveal the ways in which their parenthood status might affect their mental and physical health outcomes.

The methods used thus far in studying single parenthood suggest a variety of reasons why single parents might be at higher risk for negative health outcomes, especially depression. Factors such as socioeconomic status and demographic characteristics are important to consider, both as potential mediators or moderators of the association and because they may reveal selection effects. None of the studies presented so far can adequately differentiate between social selection and social causation, two competing explanations for observed differences between phenotypes. Thus, these methods cannot adequately explain the mechanisms behind their findings, as observed associations alone are causally ambiguous and it is, of course, impossible to randomly assign single parenthood.

Methodological issues in parenthood research: social selection vs. social causation

Saying that single parenthood causes negative health outcomes is inaccurate if all we have to go on is correlational research, since there are two possible mechanisms for processes that could explain the observed association: social selection and social causation (Carr & Springer, 2010). Selection implies that people with positive traits or who exhibit behaviors influenced by their early rearing environments or genotypes tend to select into partnered parenthood (the term "partnered" will be used instead of "married" in order to account for committed non-marital relationships), whereas individuals with more negative traits are at a higher risk of remaining single or getting divorced. Causal pathways imply that intrinsic aspects

of partnered parenthood are protective to those who enter into the parenthood state, through mechanisms such as economic advantage, the normative aspect of a relationship, and increased emotional and social support (Dinescu et al., 2016; Dinescu, Haney-Claus, Turkheimer, & Emery, in press; Emery, Horn, & Beam, 2012; Turkheimer & Harden, 2013). Researchers continue to debate the extent to which causation or selection processes are at play in observed associations between social determinants and health outcomes, and the problem arises in trying to tease out correlation from causation. We are concerned with issues of correlation versus causation in the relationship between single parenthood and parents' health, both scientifically and because effective social programs need to target true causes of health issues.

An additional concern is that of potential gene-environment correlation. Someone's genetic makeup is correlated with their environmental experiences, creating a potential extraneous factor that could explain the correlation between single parenthood and, for instance, depression. There are two ways in which genetic selection might explain the effects of parenthood status on depression (Scarr & McCartney, 1983). The evocative gene-environment correlation occurs when certain genotypes evoke certain responses from the environment. For instance, pleasant and cheerful adults may be more likely to evoke positive reactions from their partners and perpetuate a positive relationship, whereas adults who are predisposed to depressive feelings and morose dispositions may be more likely to remain single or get divorced. The active gene-environment correlation can be conceptualized as niche-picking and occurs when people seek out environments that they find stimulating and compatible. For instance, people who are more steadfast or optimistic may select themselves into partnered parenthood, whereas people who have neurotic or depressive tendencies might select themselves out of it. In the absence of random assignment, causal inferences are difficult to accomplish. Single parents differ from

partnered parents in many ways, some measurable and others impossible to measure. Genetic and environmental selection factors may account for an observed association between single parenthood and health indicators.

Prior research has tried to untangle cause and correlation by examining the confounding, mediating and moderating effects of measured variables (Cunningham & Knoester, 2010; Samuels-Dennis, 2006) or by using longitudinal designs (Avison, Ali, & Walters, 2007; Brown & Moran, 1997). However, the use of cross-sectional data makes selection effects a plausible hypothesis in non-experimental social science, and longitudinal analyses are not able to control for genetic or unmeasured shared environmental confounds. We are proposing a study of single parenthood and health outcomes that uses a type of data uniquely positioned to help answer the question of whether selection or causation mechanisms are at play in an observed association between our variables of interest: twin data. Twin research is a robust tool that allows us to account for the presence of possible selection factors and perform a quasi-experimental analysis of correlation and causation. This method has been used extensively in investigations of observed social and behavioral outcomes, from marriage and family research (Beam et al., 2011; Emery et al., 2012; Horn, Xu, Beam, Turkheimer, & Emery, 2013) to BMI and alcohol use (Dinescu, Horn, Duncan, & Turkheimer, 2015; Kaprio, 2015).

Twins as quasi-experiments

Genetically informed research offers the most robust tool available in the analysis of quasi-experimental data. Behavioral genetic research uses twins, who by definition are matched for family and cultural background and genetic predisposition, and can help enormously to answer causal questions by controlling for genetic and shared environmental selection. Comparing identical twins discordant for single parenthood controls for genetic differences,

while also controlling for race, neighborhood, family background, and for the totality of familial environmental experience. In fact, twin studies control for measured and *unmeasured* genetic and shared environmental selection, including aspects of family life that we can assess only imperfectly (e.g., family dynamics) or not at all (e.g., the entirety of shared childhood experiences).

Twin studies offer the best available methodology for distinguishing cause from correlation in cross sectional data short of a randomized trial, which is impossible to conduct for outcomes such as parenthood. However, while this methodology controls for confounds that are shared by pairs of twins who were raised together, it cannot control for *all* possible confounds of a causal relationship. Associations within twin pairs do not conclusively prove causation, because without randomization it is ultimately not possible to control all potential confounds, but because twinships control for genetic and shared environmental confounds at the level of families, they are a step closer than non-genetic research to a causal interpretation of the results. Therefore, the twin design allows us to establish a *quasi-causal effect* of single parenthood on mental health outcomes (Turkheimer & Harden, 2013). We use the term quasi-causal to refer to an association between uncontrolled variables that survives testing with a quasi-experimental design in which genetic and shared environmental confounds are controlled.

Sophisticated, multivariate analyses allow us to partition covariances between targeted family experiences and the outcomes they cause into genetic, shared, and nonshared environmental components. It is conceivable that the same factors that contribute to someone becoming a single parent might also contribute to higher levels of depression, stress, or anxiety. For example, poverty might increase someone's chances of never marrying, or getting divorced after having a child, while also contributing to a higher level of depression or anxiety compared

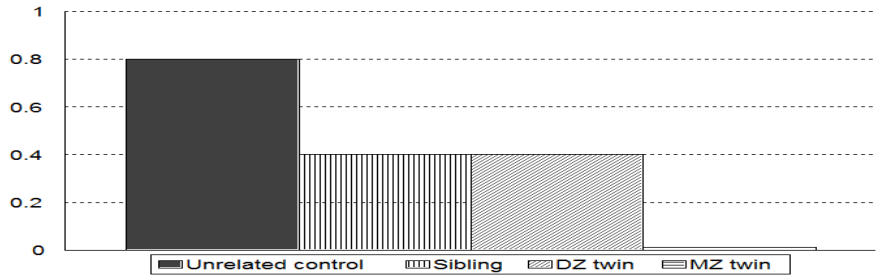
to someone with higher income. On the other hand, single parenthood may directly cause the negative outcomes. The twin studies we propose will allow us to distinguish selection from causation in our analyses, and then test specific social causation hypotheses using data unbiased by genetic selection or by measured and unmeasured shared environmental selection.

The utility of twin research in the study of behavioral data

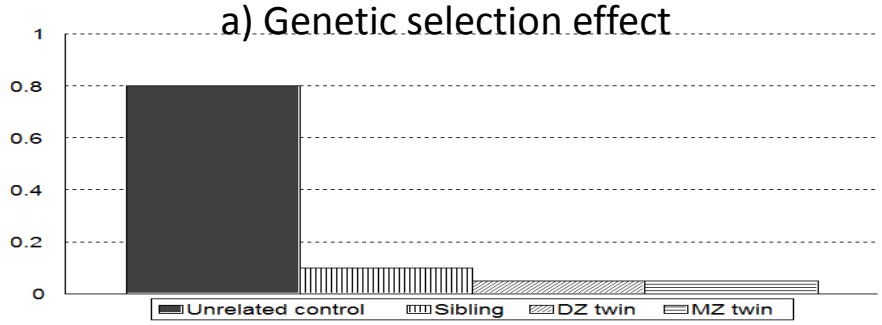
We explain the utility of the twin design below, using the example of single motherhood and depression. To the extent that single motherhood causes differences in depression levels, when we look at twin pairs we should observe this relationship within pairs (i.e. between the two twins in a pair) as well as between them (i.e. comparing one twin pair to another). Within pairs of identical (i.e. monozygotic, or MZ) twins who differ in "motherhood status" (i.e. single vs. partnered mothers), a causal hypothesis implies that the partnered member of the pair will display different depression levels than the single twin. This might be because partnered mothers have more opportunities to take breaks from the stress of parenting, or because they get more emotional support from their partner, or simply because they get to share the burden of parenting and responsibility with someone else. An association between single motherhood and depression within pairs of MZ twins controls for selection factors arising in either genotype or rearing environment, because the twins are genetically identical and were raised in the same home. In contrast, if the association between single motherhood and depression is the result of non-causal genetic or environmental confounds, the association would be observed between pairs (families in which mothers tend to be partnered tend to have positive outcomes) but not within them.

The advantage of studying twins can be made even clearer by illustrating it graphically in Figure 1 (below).

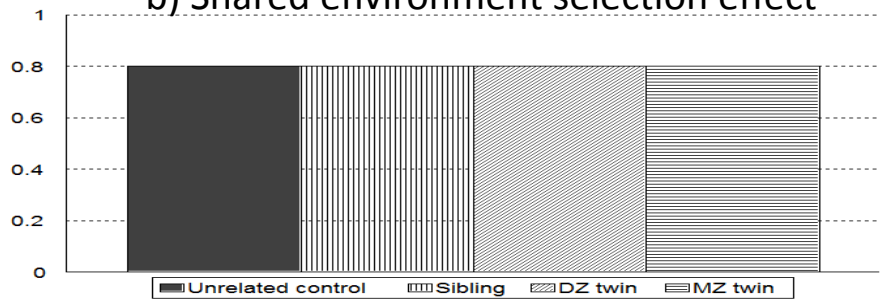
Figure 1. The illustration and interpretation of differences in selection effects obtained by studying unrelated individuals, siblings, and twins



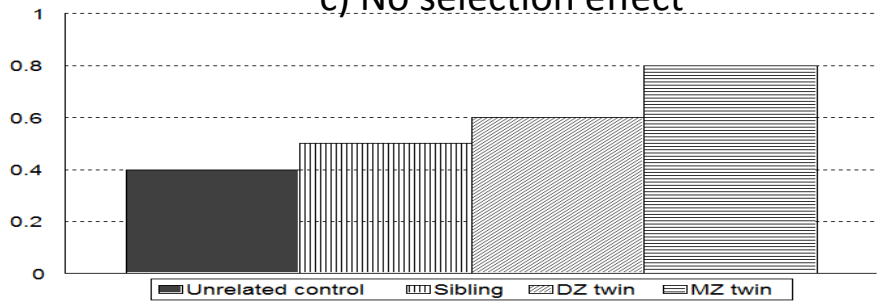
a) Genetic selection effect



b) Shared environment selection effect



c) No selection effect



d) Suppression effect

Figure 1 presents the difference in selection confounds controlled in analyses involving unrelated adults (no control for genetic or shared environmental confounds), siblings (they share 50% of their genes, on average, and grow up in the same family at different times), DZ twins (they share 50% of their genes on average, and are reared together simultaneously), and MZ twins (they share 100% of their genes and are reared together simultaneously). Selection effects are increasingly evident with each successive comparison; to return to our example, the difference in depression levels between single and partnered mothers would become smaller as we compare different demographic groups. The results are not always straightforward: it is possible to encounter various outcomes, based on the selection effects that might be involved.

If there is evidence of genetic selection, we will notice a 50% decrease in effect sizes for siblings and DZ twins, and a further decrease to 0 for MZ twins (Figure 1a). If the differences are accounted for by shared environmental selection, the effects will be small for siblings, and will disappear in DZ and MZ twin analyses (Figure 1b). If there are no significant genetic or shared environmental selection confounds, the effect sizes should not differ between the groups (Figure 1c). Lastly, a suppression effect could mask an effect that does in fact exist. A suppression effect would appear in cases where an effect was observable when examined within twin pairs – in other words, if hypothetically single motherhood did negatively impact women's depression levels. However, when we sampled the general population we would not be able to observe this effect because in this genetically unrelated sample, women who are at low risk for depression select themselves into single motherhood, thus *suppressing* the existing effect. So while in an MZ pair of twins, who have equal genetic predispositions for depression, we would see a difference between depression levels of the single versus partnered twin, we would not observe the same difference in the general population.

In the general population individuals have different genetic propensities for depression, and the hypothetical effect would get suppressed by non-random selection into single parenthood of women with low genetic risk for depression. If a suppression effect was present, the effect sizes would be larger when examining siblings and twins (Figure 1d). The magnitude of effect size differences would depend on whether the selection effects were genetically- or environmentally-based, or both.

Thus, twin data and genetically informed analyses are useful tools for differentiating between selection effects and causal mechanisms, and thus deepening our understanding of the mechanisms through which single parenthood is associated with health outcomes. Our primary, present focus is to investigate the mental health effects of single parenthood for adults using two large, representative twin data sets. Our central goal is determining the extent to which selection accounts for the observed effects of single parenthood. After controlling for selection we propose, where data allow, to analyze estimates of any effects of single parenthood by testing causal hypotheses using: (a) different parenthood statuses, and (b) different mental health outcomes (i.e., depression, anxiety, perceived stress, coping).

The role of policy in single parents' outcomes

A second focus of the current study is to conduct a cross-cultural comparison of societal influences on mental health outcomes of single parents, as research suggests that policy regimes may have a significant impact on citizens' health and behavior. Welfare regime theory is increasingly used in research on the associations between social determinants and health outcomes. This theory argues that population health should be better in social democratic regimes, or in our case, that supportive policy measures would help alleviate single parents' financial strain – and through it, the risk for negative mental health outcomes.

The different societal contexts between Sweden and the US allow for testing of "gene-culture" interactions by examining whether genetic influences on the association between single parenthood and mental health outcomes differ between the two countries. The United States and Sweden, two countries with radically different social systems and policy measures impacting single parents, offer a natural experiment within which to test our hypotheses.

The proposed study will be the first cross cultural analysis of single parenthood using twin data. Our aim is to use their differing policy contexts to draw further conclusions about the role of the environment on health outcomes.

Research on the impact of welfare regimes on outcome differences for single parents

Prior research is inconclusive as to whether different policy regimes have an impact on the association between single parenthood and mental health outcomes. A comparative analysis between Canada and Norway suggests that Canadian single parents have a lower health status than partnered parents, whereas the same is not found in Norway (Curtis & Phipps, 2004). This result is interpreted as having to do with the more generous social benefits in Norway. Similarly, other studies reveal more of a struggle to meet basic needs in countries with less support, such as Russia and the US, compared to countries with more support, such as Norway (Brown, 2008), and find that single mothers are at higher risk for depressive feelings in some welfare regimes versus others (Van de Velde, Bambra, Van der Bracht, Eikemo, & Bracke, 2014). On the other hand, some studies find that single parents have significantly worse health and more financial hardship than coupled parents regardless of policy regimes when comparing Italy, Sweden, and Britain (Burstrom et al., 2010; Fritzell et al., 2012), or more generally, that the type of welfare regime does not have a significant impact on health disparities in the population (Brennenstuhl,

Quesnel-Vallée, & McDonough, 2012). There is no available research on the impact on policy regimes on single fathers' experience.

Given the inconclusive results of existing investigations, more research is needed in order to understand the complex ways in which social support measures may impact the association between single parenthood and health outcomes. We propose to conduct a cross-cultural comparison using Sweden and the US, two countries with vastly different welfare regimes, and different social policy measures aimed at single parents. These differences create significant environmental variation between and within cultures. Variation between cultures means that single parents experience vastly different conditions and support depending on the country they live in, and the policy measures they benefit from. Variation within cultures means that the differences between single and partnered parents are smaller in a culture with more support for single parents, and larger in a culture with less support measures for them. Below we describe the current state of policy measures in our countries of interest.

Current policy landscape: Sweden

Sweden has three major policies that target family economic security, gender equality, voluntary parenthood, and children's rights (Haas, 1996). Sweden 1) supplies allowances for each child, 2) has an extremely generous parental leave policy after the birth of a child, and 3) has a free public childcare program. These policies are universal, and all families are guaranteed certain rights (Haas, 1996). These programs do not depend on the income of the child's parents, nor are they short-term or temporary. The creation of these policies is meant to encourage all parents to take on the dual roles of working and child-rearing, with the idea that the laws in place should prevent as many difficulties that might arise from that duality as possible. The laws aim for a horizontal equality among families, regardless of family structure (i.e. single parent families

headed by single mothers or fathers), number of children, racial or socioeconomic background (Ozawa, 2004).

In order to gain this horizontal equality, Sweden's laws aim to keep as many families as possible above the poverty line. This is especially crucial when talking about single parents. Oftentimes single parents must provide all care-giving and economic support for their children. In countries without supportive family policies, many single parents are forced to only work part time in order to care for their child due to high childcare prices. For instance, when adequate government policies are not in place to offer support, single mothers are at a greater risk for poverty (Brady, 2006; Misra, Moller, & Budig, 2007). In Sweden, however, single mothers have less than a 2% risk of poverty, as compared to single mothers in the United States who have a risk of over 35% (Misra, Budig, & Moller, 2007).

Sweden's success in assisting parents, single parents in particular, comes from a few key policies. The first of such policies is the child allowance policy. The benefit, as of March 2014, is split equally between the child's guardians if the child is under joint custody, while single parents get the whole allowance of about $150 USD per month until the child turns 16 ("Nordic Social Insurance Portal-Family Benefits," n.d.). This policy displays gender equality as well as the encouragement for women's economic independence, even when in a heterosexual cohabitation. Research has found that when governments give families this type of assistance, poverty is more easily eradicated (Christopher, 2002; Christopher, England, Smeeding, & Phillips, 2002; Kenworthy, 1999; Misra, Budig, et al., 2007; Smeeding, 2005).

A second important policy is the parental leave policy that is in place after the birth of a child. A substantial leave policy has been shown to help all parents, but single parents benefit more. In fact, a moderate to strongly generous leave brings single mothers down to an almost

equal risk for poverty as partnered mothers (Misra, Moller, Strader, & Wemlinger, 2012). In Sweden, parents share a maximum of 480 days of parental leave, with two months exclusively for each parent in order to encourage fathers to spend more time at home with their child. The first 390 days are compensated based on income. If the parent has a low income or no income at all, a basic flat rate is paid. The other 90 days are paid a low flat rate. Altogether, parents are eligible to receive up to 80% of their income while on leave (Wells & Bergnehr, 2014). Leave is designed to allow women to have children, but still keep their competitive and higher paid jobs. 81% of mothers with children under the age of 18 are employed (Barnombudsmannen, 2010), and Sweden's wage gap is lower than the OECD average (OECD, n.d.).

Swedish subsidized childcare also keeps women from having to choose between having a child and having economic stability. It has been shown that universal childcare helps to increase both women's participation in the labor market as well as women's wages (Pettit & Hook, 2005). One of the greatest deterrents that prevent single mothers from going back to work is lack of affordable childcare: providing childcare for children of ages 0-2 was significant in helping reduce the risk of poverty in single parents (Burstrom et al., 2010). Thus, Sweden's policy is a dual investment; by supplementing childcare, parents do not have to cut back to a part-time job, and children start out in a stimulating, safe, and educational environment.

Current policy landscape: The United States

The United States has a different approach to assisting families, with few national programs. These programs are temporary, not universal, and eligibility is determined at the state level. America's welfare programs are not preventative measures, but rather attempt to help families recover once they have reached a level of need, as income level is one of the main criteria for eligibility of welfare assistance. Compared to Sweden, the United States does not

provide any type of child allowance, parental leave is sparse and without a specific national mandate, and unregulated childcare is more prominent here than in most other developed nations (G. Olsen, 2007). Financially, the United States offers a Child Tax Credit, which is a reduction in taxes of up to $1000 per child. However, this is not similar to an allowance, as the amount of credit depends on the amount of the income tax owed ("Ten Facts about the Child Tax Credit | National Tax Reports," n.d.).

In the US, parental leave fits under the Family and Medical Leave Act, which also covers leave for severe illness, temporary disability, or care for a child or spouse with a serious health condition. Under this act, companies must allow employees 12 weeks of unpaid leave unless the company is less than 50 employees, the person in question has been employed for less than a year, or if the employee has an income that is in the top 10% of wages within the company. These welfare programs were not designed to provide special assistance to mothers, and if a single mother does take time off, employers have the option of offering paid maternity benefits, or a certain number of weeks covered by temporary disability insurance. A study done in the mid 1990s showed that only 43% of women were given paid leave under these conditions, and only 7% of men (Han, Ruhn, & Waldfogel, 2009). Lack of leave and other social policies specific to working parents forces many fathers and mothers to choose between time with their infants and economic security (Gornick & Meyers, 2004).

Assistance programs in United States include Temporary Assistance for Needy Families (TANF), Supplemental Nutrition Assistance Program (SNAP), and The Special Supplemental Nutrition Program for Women, Infants, and Children (WIC). TANF is a grant that each state receives for their individual welfare programs ("Temporary Assistance for Needy Families (TANF) - Virginia Department of Social Services," n.d.). All recipients must find work within

two years of receiving aid to ensure that people are not remaining unemployed to collect benefits. SNAP (formerly Food Stamps) is meant to help low-income households purchase food and make educated nutrition choices ("Supplemental Nutrition Assistance Program (SNAP) | Food and Nutrition Service," n.d.). WIC is more focused on helping low-income pregnant women, mothers, and children under the age of five with healthcare and nutrition ("Women, Infants, and Children (WIC) | Food and Nutrition Service," n.d.). These programs are temporary, must be applied for, and do not focus on combining employment with childcare to prevent poverty. This makes them difficult to access by single parents: they have to apply and wait to be found eligible for assistance they will get only after having reached poor economic status, and the programs do nothing to encourage employment.

Understanding the effect of policy regimes on the relationship between single parenthood and health outcomes can help policy makers devise effective strategies for prevention and support. The environmental effects created by policy regimes may impact both the heritability estimates in the two cultures, as well as directly affect individuals' behavior and health outcomes.

Summary and hypotheses

Single parenthood is increasingly prevalent, and impacts communities in tangible ways. Economically, single parents have fewer resources and need to work more to maintain the same level of income as partnered parents. Socially, single parents benefit from less social support, maybe because do not have the time to contribute as much to a community as parents in a two-partner home. Emotionally, single parents may have greater needs, which may lead to alienation of their social support network and create a vicious cycle of lack of support and possible psychopathology. Despite the significance of this issue, relatively little is known about the nature of the mental health outcomes of single parents, about the causal mechanisms behind these

outcomes, and, consequently, about possible interventions. There are no investigations to date into this topic using genetically informed methods. Furthermore, the project has the unique advantage of being the first cross-cultural analysis of single parenthood using twin data. These characteristics make the present project a truly novel contribution to the field, and the beginning of a deeper investigation into the processes behind single parenthood.

The present project explores the association between single parenthood and health outcomes through a series of four studies.

Study 1 is a descriptive analysis of single parenthood and health outcomes.

H1. Single parents will exhibit higher mean levels of mental health outcome measures.

H2. The mean differences between single and partnered fathers will be smaller than the mean differences between single and partnered mothers.

Study 2 aims to eliminate nonrandom selection factors and greatly strengthen causal likelihood estimates of the association between single parenthood and depression.

H1. When examined in the whole sample (equivalent to the general population), single parents will exhibit significantly higher depression levels than partnered parents.

H2. The association between single parenthood and depression remains significant when controlling for genetic and shared environmental confounds, as well as for theoretically informed confounds of this association such as demographic variables or socioeconomic status.

Study 3 aims to conduct a cross-cultural comparison between Sweden and the US.

H1. Heritability of depression associated with single parenthood will vary across cultures ("gene-culture" interaction).

H2. Differences between single and partnered parents will be smaller in Sweden than in the US, both phenotypically and quasi-causally.

Study 4 aims to explore the association between single parenthood and other negative mental health outcomes.

H1. When examined in the whole sample (equivalent to the general population), single parents will exhibit significantly higher levels of anxiety, perceived stress, and coping, than partnered parents.

H2. The association between single parenthood and negative health outcomes remains significant when controlling for genetic and shared environmental confounds, as well as for theoretically informed confounds of this association such as demographic variables or socioeconomic status.

Methods

Sample

In comparing the United States and Sweden, we used the Washington State Twin Registry (formerly known as the University of Washington Twin Registry or UWTR), and the Swedish Twin Registry (STR).

The UWTR is a cross-sectional community-based sample of adult twins reared together (Afari et al., 2006; Strachan et al., 2013). Recruitment began in 2002 and included twins 18 years of age and older at the time of recruitment (age range: 18-94, mean: 37) (Strachan et al., 2013). Twins were ascertained upon registering with the Washington State Department of Licensing, and were mailed necessary documentation, including a survey. Once one twin completed the survey, the co-twin was recruited and sent the same package. It is estimated that across mailings, response rates for initially registered (i.e. index) twins range from 21%-38%, while response rates for their co-twins range between 56%-76% (Afari et al., 2006). Twins completed surveys that included items on sociodemographics, health, and lifestyle behaviors. Twins were classified

as identical (monozygotic; MZ) or fraternal (dizygotic; DZ) using standard questions about childhood similarity, which determine zygosity with greater than 90% accuracy when compared with DNA-based methods (Eisen, Neuman, Goldberg, Rice, & True, 1989; Spitz et al., 1996; Torgersen, 1979). The dataset was designed to be representative of the state of Washington in terms of socioeconomic factors, gender and ethnic distribution, but lacks the ethnic diversity necessary to be representative of the US population (i.e. 88% white participants). We used all same-sex twin pairs for which parenthood status data is available in the dataset (partnered parents N= 1185 female MZ, 600 female DZ, 554 male MZ, 239 male DZ; single parents N= 286 female MZ, 146 female DZ, 82 male MZ, 45 male DZ).

The STR is the largest twin registry in the world, containing information from 170,000 Swedish twins born since 1886. Several birth cohorts make up the registry totaling 29,030 MZ male twin pairs, 27,372 MZ female twin pairs, and 28,686 DZ twin pairs. Data were collected through an online questionnaire, or if the participant preferred, a telephone interview followed by a mailed questionnaire. The survey gathered information regarding diseases with a focus on exposure during young adulthood and midlife. We analyzed data from a cohort of the Swedish Twin Registry (STR), the Swedish Twin study of Adults: Genes and Environment (STAGE). This cohort includes twins born 1959 to 1985 (age range: 20-47, mean: 33). The information collected includes history pertaining to physical and emotional health and health behavior, work history, family composition, and major life events. Of the 42,582 eligible twins, 59.6% responded (N= 25,364). Data for the current project came from all available same-sex twin pairs for which parenthood data was available (partnered parents N= 2062 female MZ, 1741 female DZ, 1270 male MZ, 1125 male DZ ; single parents N= 277 female MZ, 274 female DZ, 124 male MZ, 107 male DZ).

Measures

Single parenthood status: To be considered a parent, a twin had to report living with one or more children at the time of the survey. Single parenthood status was then determined based on self-reported marital status. Parents reporting widowhood were not included in the current analysis. Married and cohabitating/partnered parents made up the "married" group, while the "single" group consisted of the divorced or separated parents, as well as those parents reporting being single and/or living alone.

Mental health measures in UWTR

Depression was measured by using three items from the Patient Health Questionnaire (PHQ-9), a nine-item measure used for depression diagnosis and severity (Spitzer, Kroenke, & Williams, 1999). The PHQ-9 corresponds to the DSM diagnosis of depression and assesses the severity of the nine symptom criteria in the past two weeks. Although a brief, three-item version is not as ideal as the full nine-item questionnaire, it has been shown that even a two-item version can be used as a valid and reliable assessment of clinical depression, both in diagnosis and severity (Löwe, Kroenke, & Gräfe, 2005). The three-item assessment (PHQ-3) measured anhedonia ("little interest or pleasure in doing things"), depressed mood ("feeling down, depressed, or hopeless"), and fatigue or decreased energy ("feeling tired or having little energy") over the past 4 weeks. Symptomatology was rated on a 4-point scale (0=*not at all*; 1=*several days*; 2=*more than half the days*; or 3=*nearly every day*). The reliability (Cronbach's α) of the three items is very high (.79-.81).

Anxiety was measured using the Anxiety scale of the Brief Symptom Inventory (BSI; Derogatis & Melisaratos, 1983), a short self-report instrument designed to measure nine primary symptom dimensions and three global indices. The instrument has high internal consistency (α=.96) and

the Anxiety scale has a high correlation with the Depression Anxiety Stress Scale – Anxiety subscale (Pearson's r=.61).

Perceived stress was measured using nine items from the Perceived Stress Scale (PSS; Cohen, Kamarck, & Mermelstein, 1983), which measures stress perception in the past four weeks. Items include: "In the past 4 weeks, how often have you been upset because of something that happened unexpectedly?"; "In the past 4 weeks, how often have you felt nervous and "stressed"?"; and "In the past 4 weeks, how often have you felt that things were going your way?" (reverse scored). Symptoms were rated on a 5-point scale (0=*never*; 1=*almost never*; 3=*sometimes*; 4=*fairly often*; or 5=*very often*) and were scaled so that higher scores reflect greater perceived stress.

Coping was measured using the Brief Resilient Coping Scale (BRCS; Sinclair & Wallston, 2004), a four-item, uni-dimensional outcome measure designed to capture how an individual copes with stress adaptively. The items assess creativity ("I look for creative ways to alter difficult situations"), tenacity ("I actively look for ways to replace the losses I encounter in life"), "resilience ("I believe I can grow in positive ways by dealing with difficult situations"), and optimism ("regardless of what happens to me, I believe I can control my reaction to it"). Items are scored on a 5-point scale from 1=*does not describe me at all* to 5=*describes me very well*.

Mental health measures in STR

Depression was assessed using an abbreviated version of the Center for Epidemiologic Studies Depression scale (CESD; Radloff, 1977), which is a 20-item measure of the experience of depressive symptomatology across nine domains over the past week. A modified, 11-item questionnaire was administered to STAGE participants, and assessed dysphoria ("felt depressed, sad"), disrupted sleep ("sleep was restless"), and fatigue ("could not get going", "everything I

did was an effort). Symptomatology was rated on a 3 point scale (0 = *Never or almost never*; 1 = *Seldom*; 2 = *Often*; 3 = *Always or almost always*). The reliability (Cronbach's α) of the short form of CESD is .86.

Covariates: All available theoretically informed covariates will be included in the analyses. Research suggests age and number of children as correlates of the relationship between single parenthood and mental health outcomes, so they were used as controls in all analyses. Moreover, socioeconomic status has been suggested as a prevalent mechanism through which single parenthood is linked to detrimental health outcomes. Hence, the highest level of education, as well as household income (widely used in research and indicative of total resources available to the family unit), were used as measures of socioeconomic status.

The highest level of education was coded on a scale of 1-3 in STR (1 = *Low level: Elementary school*; 2 = *Mid level: All higher-then-elementary but non-university types of education*; 3 = *High level: University*) and on a scale of 1-8 in UWTR (1 = *never attended school/only Kindergarten only*; 2 = *Grades 1-8*; 3 = *Grades 9-11*; 4 = *Grade 12/High School diploma/GED*; 5 = *some college*; 6 = *Associate's Degree/Vocational or Trade School Degree*; 7 = *Bachelor's Degree*; 8 = *graduate or professional degree)*. Household income was coded on a continuous scale in STR, and categorically on a scale of 1-11 in UWTR (1 = *less than $20,000*; 2 = *$20,000 to $29,999*; 3 = *$30,000 to $39,999*; 4 = *$40,000 to $49,999*; 5 = *$50,000 to $59,999*; 6 = *$60,000 to $69,999*; 7 = *$70,000 to $79,999*; 8 = *$80,000 to $89,999;* 9 = *$90,000 to $99,999;* 10 = *$100,000 to $149,999;* 11 = *$150,000 or more*).

Statistical analysis

We carried out the comparison between single and partnered parents in twin pairs by fitting a series of structural equation models using the robust weighted least squares (WLSMV)

estimation option in the Mplus 7.11 program, used for model fitting (Muthén & Muthén, 2013). WLSMV assumes data to be missing completely at random, which allows for the use of all the data available (Muthén & Muthén, 2010). RMSEA was used as measure of model fit, with the cutoffs 0.01, 0.05, and 0.08 indicating excellent, good, and adequate fit, respectively (MacCallum, Browne, & Sugawara, 1996). When comparing nested models (i.e. a model in which the parameters were constrained to be equal between men and women and a model in which they were allowed to be free) we used the Wald test (Wald, 1943). Analyses controlled for linear effects of age, income, education level, and number of children. Each model presented was run separately for mothers and fathers, and separately for the US and Sweden, except in the case of cross-cultural comparisons. Below we present our modeling approach, step by step.

Step 1: Descriptive statistics

One of our goals was to provide a full description of each parent sample. This is especially important for our single fathers sample, given the rarity of single father studies to date. We presented frequencies of parenthood statuses, as well as means and standard deviations of all variables, by gender, parenthood status, and nationality.

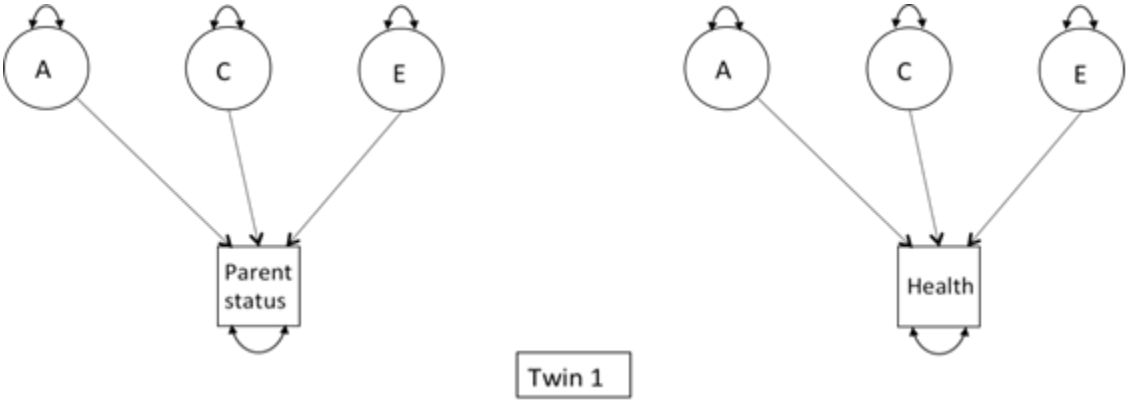
Step 2: Univariate biometric decomposition

We began by fitting univariate twin models (Figure 2), which partition the variance of an outcome into three components: additive genetic influences (A), shared environmental influences (C), and non-shared environmental influences (E).

Genetic factors (A) represent the proportion of the variance in the outcome that is associated with the additive effect of genes. Genetic variance correlates at 1.0 for monozygotic (MZ) twins, who share 100% of their genes, and .5 for dizygotic (DZ) twins, who share on average 50% of their genes. Shared environmental variance correlates at 1.0 for both MZ and DZ

twins, as they are assumed to fully share parts of their environment such as parental influences, socioeconomic status, and many other childhood experiences. Non-shared environmental factors (E) do not correlate between twins, as these represent experiences that are not shared, and which contribute to twins' unique, within-pair variance. These consist of any environmental factors that make twins different from one another, and include measurement error.

Figure 2. Univariate ACE models. Twin 1 shown for clarity.

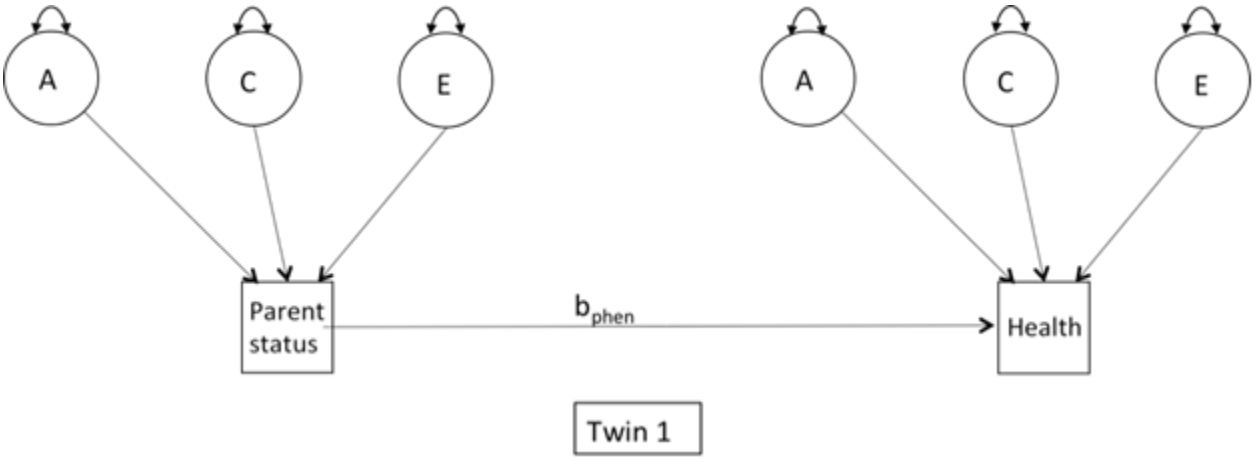


We used the results of our univariate models to compare the heritability of each health outcome for single versus partnered parents within cultures, and for depression also across cultures.

Step 3: Regression of health outcomes on parental status

We then regressed each health outcome on parenthood status to get an estimate of this association at the phenotypic level (Figure 3). This result is equivalent to a population-level association, calculated without using a twin design, and represents the effect of parenthood status on health outcomes without controlling for any possible genetic or shared environmental confounds.

Figure 3. Structural equation model representation of phenotypic model used in data analysis. This model is equivalent to a population-level regression.

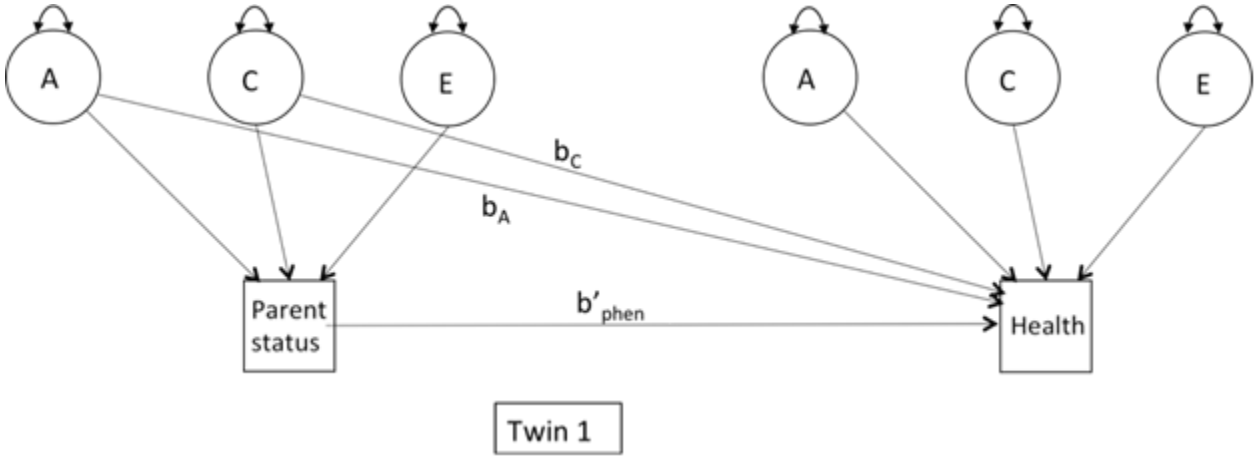


Step 4: Genetically informed regression of health outcomes on parental status

Since we cannot randomly assign participants to parenthood status, using pairs of MZ and DZ twins raised in the same family provides the strongest available method for approximating the causal effect of single parenthood on health outcomes. Assessing this relationship within twin pairs allows us to control for the effects of many measured and unmeasured confounds that vary between families, such as underlying genetic or environmental backgrounds that single parenthood and health outcomes may share. Below we describe the statistical model used to test questions like "is single parenthood quasi-causally associated with depression?"

To help answer such questions, we fit a genetically informed phenotypic regression model (Figure 4), in which the effect of parenthood status on health was estimated holding constant any genetic and shared environmental effects common to both (Turkheimer & Harden, 2013). In this model, we simultaneously regressed each health outcome on parenthood status (parameter *b'phen* - see Figure 4) *and* on the A and C variance components of parenthood status.

Figure 4. Structural equation model representation of quasi-causal model used in data analysis. This model controls for genetic and shared-environmental confounds.



If the *b'phen* parameter remained significantly different from zero after adjusting for the A and C regressions in the model, it was interpreted as a quasi-causal effect of parenthood status on the mental health outcome in question. Such a result would support the causal hypothesis, suggesting that parenthood status has a *quasi-causal* effect on that particular outcome, above and beyond genetic and shared environmental influences that might explain the association. This means that in a pair of identical twins discordant for parenthood status (i.e. one twin is a partnered parent and the other one is a single parent) we would observe significantly different health levels between the twins. Conversely, if the uncontrolled regression parameter *bphen* was significant, but *b'phen* was substantially reduced, even nonsignificant, the selection hypothesis would be supported. This means that the apparent effect of parenthood status on depression in the phenotypic model resulted at least in part from uncontrolled genetic and shared environmental factors. In other words, in a pair of identical twins in which one is a single parent and one is not, we observed the same health levels in both twins.

The twin design, of course, cannot control for *all* possible confounds of a causal relationship, but only for those which are shared by pairs of MZ twins who were raised together, such as genetic predispositions, race, or childhood socioeconomic status. We therefore assert that the twin design allows us to establish a *quasi-causal effect* of single parenthood on health outcomes (Turkheimer & Harden, 2013).

Step 5a: "Gene-culture" interaction

We were able to test for a version of gene by environment interaction that we will call "gene-culture" interaction. This is a simple version of the traditional gene-environment interaction twin analysis, where the moderator is defined by culture. Our analysis looked for a statistically significant effect of the cultural environment on our estimates, and on the variation in these estimates.

To test for a gene-culture interaction effect, we extended the model described above by comparing the MZ and DZ twins from the US sample and the MZ and DZ twins from the Swedish sample in a four group model. In this model we looked at the difference in the *bA* coefficient (see Figure 4), which represents the heritability of depression associated with single parenthood by conducting a chi-square test (i.e. Wald test). We interpreted significant results as evidence of a gene-culture interaction effect. In other words, that heritability of depression associated with parenthood status varies across cultures.

Step 5b: "Quasi-causal effect-culture" interaction

We also tested for an interaction effect of culture on the phenotypic and quasi-causal estimates obtained from the models in step 4, following the same reasoning as the one in step 5a. To test for this interaction effect, we looked at the difference in the *bphen* and *b'phen* coefficients (see Figures 3 and 4) before and after controlling for A and C. If chi-square test

results are not significant, this is interpreted as no significant difference in estimates between cultures. In other words, cultural exposure does not moderate the phenotypic or quasi-causal effect of single parenthood on depression. If, on the other hand, chi-square tests yield a significant result, this would suggest the presence of an interaction effect, suggesting that the effect of single parenthood on depression varies by cultural exposure.

Results

Results are organized by study. The first study presents descriptive comparisons of demographic and mental health variables by parenthood status in US and Swedish twin samples (Study 1). The second study aims to examine causal likelihood estimates of the association between single parenthood and depression by eliminate nonrandom selection factors (Study 2). The third study aims to conduct a cross-cultural examination of twin samples from the US and Sweden by comparing heritability estimates and multivariate model results in the two populations (Study 3). The final study replicates the methodology of Study 2 in exploring other mental health outcomes (i.e. anxiety, perceived stress, coping; Study 4). Due to low sample size, phenotypic and biometric model estimates could not be computed or interpreted for father samples. Instead, we present descriptive statistics and graphical representations of group differences between single and partnered fathers. All parameter estimates were constrained to be the same for twin pairs under the assumption that twin siblings are interchangeable dyads (J. A. Olsen & Kenny, 2006). We report the detailed results below.

Mothers

Study 1: Descriptive analysis of single parenthood and mental health outcomes

Descriptive statistics are presented in Tables 1a and 1b for the US and Swedish datasets, respectively. In the US, partnered mothers appeared to be on average about one year younger

than single mothers, and reported a significantly higher level of household income ($t(608) = 19.14$, $p < .05$) and education ($t(668) = 6.79$, $p < .05$). No significant difference in number of children between the two groups was observed ($t(580) = 1.03$, $p = .30$).

Table 1a - UWTR descriptive statistics
Frequency, means, and SDs by gender

	Fathers	Mothers
Partnered	793	1785
Single	127	432
Mean (SD)		
Age (range: 18-97, M = 41.58, SD = 17.45)		
Partnered	53.93 (15.55)	47.79 (14.61)
Single	48.53 (14.81)	48.70 (14.77)
Number of children (range 1-7, M=2.22, SD=1.11)		
Partnered	2.37 (1.17)	2.17 (1.03)
Single	1.79 (1.05)	2.10 (1.19)
Household income (range: 1-11, M=5.26, SD=2.7)		
Partnered	6.51 (2.06)	6.14 (2.29)
Single	4.35 (2.57)	3.57 (2.52)
Education (range 2-11, M=6.56, SD=1.91)		
Partnered	7.03 (1.91)	6.58 (1.90)
Single	5.86 (1.93)	5.90 (1.85)
Depression (range: 0-9, M = 1.63, SD = 1.87)		
Partnered	1.09 (1.53)	1.54 (1.75)
Single	1.70 (1.95)	2.20 (2.23)
Anxiety (range: 0-24, M = 2.3, SD = 3.22)		
Partnered	1.41 (2.19)	2.06 (2.79)
Single	2.29 (3.60)	3.14 (3.98)
Perceived stress (range: 0-39, M = 8.88, SD = 5.59)		
Partnered	8.26 (4.68)	8.57 (4.82)
Single	8.18 (4.49)	9.43 (6.87)
Coping (range: 4-20, M = 14.31, SD = 3.36)		
Partnered	14.59 (3.39)	14.17 (3.35)
Single	14.29 (3.74)	14.31 (3.39)

Partnered US mothers reported significantly lower mean levels of depression ($t(562) = -5.64$, $p < .05$) and anxiety ($t(521) = -5.27$, $p < .05$) than their single counterparts. No significant mean differences were reported in perceived stress ($t(193) = -1.52$, $p = .12$) and coping scores ($t(640) = 0.73$, $p = .46$).

Similarly, in Sweden partnered mothers were on average slightly younger (i.e. 1.5 years) than single mothers, and reported higher household income ($t(2907) = 34.15$, $p< .05$), education ($t(765) = 6.35$, $p < .05$), and number of children ($t(788) = 2.65$, $p < .05$) than their single counterparts. As in the US, Swedish partnered mothers reported lower levels of depression than their single counterparts ($t(698) = -13.21$, $p < .05$).

Table 1b - STR
Frequency, means, and SDs by gender

		Fathers	Mothers
	Partnered	2893	4249
	Single	299	633
Mean (SD)			
Age (range: 20-47, M = 33.15, SD = 7.63)			
	Partnered	37.97 (5.28)	37.17 (5.69)
	Single	38.55 (5.84)	38.72 (6.14)
Number of children (range: 1-7, M = 2.05, SD = 0.95)			
	Partnered	2.01 (0.85)	2.05 (0.86)
	Single	1.83 (0.87)	1.94 (0.96)
Household income (range: 0-145894.7, M = 4033.59, SD = 2852.96)			
	Partnered	5277.908	5226.543
	Single	3157.684	2719.685
Education (range: 1-3, M = 2.39, SD = 0.58)			
	Partnered	2.27 (0.58)	2.37 (0.58)
	Single	2.10 (0.58)	2.19 (0.62)
Depression (range: 0-33, M = 7.18, SD = 5.58)			
	Partnered	6.07 (4.64)	6.49 (5.40)
	Single	9.01 (5.66)	10.33 (6.70)

Univariate ACE models

Standardized biometric variance components for parenthood status and mental health outcomes are presented in Table 2.

All parenthood status and mental health phenotypes included some between-family variation, either genetic or shared environmental, in addition to significant nonshared environmental variance. There was evidence for moderate additive genetic influences across outcomes, whereas shared environmental contributions were much smaller, and not significantly different from zero in most comparisons. There was substantial nonshared environmental variance in all phenotypes, which simply means that identical twins were less than perfectly correlated for all outcomes.

Specifically, parenthood status showed similar influences in the US and Sweden: primarily from the nonshared environment (74% and 67% in the US and Sweden respectively), modest influence by genetic factors (26% and 23%), and none or very small influence from shared environmental factors (0% and 9%). A similar pattern of results was observed for depression across the two countries, as well as for other mental health outcomes in the US dataset. Depression was mainly influenced by nonshared environmental factors (69% and 62% in the US and Sweden, respectively), with a modest contribution from the shared environment (11% and 10%) and a moderate contribution from genetic factors (21% and 28%). Anxiety, perceived stress, and coping showed moderate contributions from A (22%, 40%, and 31%, respectively), none or minor from C (11%, 0%, 0%), and a large nonshared component (66%, 60%, 69%).

Table 2
Standardized ACE components for parenthood status and mental health outcomes

	Parenthood Status	Depression	Anxiety	Perceived Stress	Coping
US - mothers					
h^2	.258 (.080)	.207 (.082)	.224 (.083)	.397 (.051)	.314 (.023)
c^2	0	.106 (.071)	.114 (.074)	0	0
e^2	.742 (.080)	.687 (.024)	.663 (.023)	.603 (.051)	.686 (.023)
Sweden - mothers					
h^2	.232 (.192)	.281 (.065)			
c^2	.094 (.192)	.100 (.057)			
e^2	.674 (.083)	.619 (.019)			
US - fathers					
h^2	.351 (.511)	.036 (.126)	.207 (.122)	.466 (.062)	.265 (.034)
c^2	.029 (.443)	.212 (.110)	.028 (.101)	0	0
e^2	.621 (.147)	.752 (.036)	.765 (.040)	.534 (.062)	.735 (.034)
Sweden - fathers					
h^2	0	.376 (.023)			
c^2	.182 (.108)	0			
e^2	.818 (.108)	.624 (.023)			

Study 2: Phenotypic and biometric analysis of the association between single parenthood and depression

Tables 3a and 3b present the results of phenotypic and biometric models in which depression was regressed on parenthood status in the US and Sweden, respectively. These results are illustrated in Figure 5.

We estimated the total effect of motherhood status on depression and all other mental health outcomes in a series of phenotypic and quasi-causal models. For each series of models, we regressed the outcome onto a dichotomous variable representing motherhood status (i.e., partnered vs. single), and progressively added covariates: age in Model 2, socioeconomic

indicators (income and education) in Model 3, and finally number of children in Model 4. At each stage, regressions are conducted simultaneously on the outcome of interest and on covariates. Variance components that estimated as negative were set to 0.

Phenotypic and biometric models – US dataset

In all four phenotypic models depression levels differed significantly (i.e. $p<.05$) between single and partnered mothers such that partnered mothers reported significantly lower depression scores than their single counterparts. These effects held for each model in the series, both with no covariates (Model 1: b_{phen} = -0.36, SE = 0.06) and when covariates were progressively included (Model 2: b_{phen} = -0.363, SE = 0.06; Model 3: b_{phen} = -0.380, SE = 0.1; Model 4: -0.173, SE = 0.05).

Table 3a - UWTR mothers

Unstandardized Parameter Estimates for Phenotypic and Biometric Models for Depression

Estimate (se)	Model 1 No covariates	Model 2 Demographic cov[1]	Model 3 Add SES cov[2]	Model 4 All cov[3]
Phenotypic model				
b_{phen}	**-0.355 (0.057)**	**-0.363 (0.057)**	**-0.380 (0.097)**	**-0.173 (0.052)**
Biometric model				
b_A	-0.258 (0.363)	-0.292 (0.367)	-0.970 (1.203)	-0.164 (0.566)
b_C	0	0	0	0
b'_{phen}	**-0.281 (0.096)**	**-0.279 (0.096)**	-0.243 (0.146)	-0.150 (0.080)
Covariates				
Age		**-0.116 (0.021)**	**-0.223 (0.040)**	**-0.106 (0.024)**
Income			**-0.126 (0.036)**	**-0.090 (0.020)**
Education			**-0.096 (0.031)**	**-0.076 (0.019)**
Number of children				0.024 (0.029)
Goodness of Fit				
RMSEA (CFI/TLI)	0.033 (.897/.914)	0.034 (.863/.879)	0.012 (.994/.991)	0.011 (.997/.994)

Note. Bolded values are significant at <.05; bphen is the full phenotypic effect; b'phen is the genetically informed phenotypic effect; bA and bC are the indirect effects of parenthood status on the phenotype.

[1]Age

[2]Age, education, income

[3]Age, education, income, number of children

We then tested whether these results were consistent with a causal hypothesis by fitting a series of bivariate quasi-causal models. Motherhood status continued to have a significant effect on depression in Model 1 (b'_{phen} *= -0.281, SE = 0.1)*. This effect is apparent in Figure 5, MZ Mothers US panel in the top left.

Concordant pairs of partnered MZ twins (i.e. both members of the pair are partnered), in the darker, leftmost bar, reported significantly lower levels of depression than concordant pairs of single MZ twins, in the lighter, rightmost bar. The effect was closely approximated within pairs of MZ twins discordant for parenthood status, which suggests that genetic and shared environmental confounds could not be responsible for it, as these are controlled in a comparison within pairs of MZ twins. Thus, this analysis indicates the existence a quasi-causal effect of parenthood status on depression.

When controlling for age, the effect remained significant (Model 2: b'_{phen} *= -0.279, SE = 0.1)*. However, when SES covariates were introduced, the effect became non-significant (Model 3: b'_{phen} *= -0.243, SE = 0.15)*. Model 4 did not meaningfully modify estimates or model fit (Model 4: b'_{phen} *= -0.150, SE = 0.08)*. Across all models, the effect of age and SES variables on depression was significant and consistent, such that mothers who were older, earned more, and had higher levels of education reported lower levels of depression. Number of children did not have a significant impact on depression levels.

Phenotypic and biometric models – Swedish dataset

Phenotypic results in the Swedish sample exhibited a similar pattern to the US sample (see Table 3b). In all phenotypic models, depression levels differed significantly (i.e. $p<.05$) between single and partnered mothers such that partnered mothers reported significantly lower depression scores than their single counterparts.

Table 3b - STR mothers

Unstandardized Parameter Estimates for Phenotypic and Biometric Models for Depression

Estimate (se)	Model 1 No covariates	Model 2 Demographic cov[1]	Model 3 Add SES cov[2]	Model 4 All cov[3]
Phenotypic model				
b_{phen}	**-1.871 (0.127)**	**-1.965 (0.126)**	**-1.620 (0.126)**	**-1.615 (0.126)**
Biometric model				
b_A	-16.783 (174.619)	-17.694 (268.701)	-17.882 (279.157)	-16.919 (253.202)
b_C	0.740 (1.753)	-0.120 (1.982)	-1.112 (3.451)	-1.112 (3.429)
b'_{phen}	**-1.645 (0.309)**	**-1.626 (0.298)**	**-1.145 (0.252)**	**-1.140 (0.252)**
Covariates				
Age		**-1.043 (0.117)**	**-1.593 (0.151)**	**-1.587 (0.155)**
Income			**0.108 (0.051)**	**0.106 (0.051)**
Education			**-1.401 (0.145)**	**-1.398 (0.147)**
Number of children				0.121 (0.105)
No race data				
Goodness of Fit				
RMSEA (CFI/TLI)	0.011 (.994/.995)	0.039 (.889/.899)	0.059 (.987/.980)	0.052 (.987/.976)

Note. Bolded values are significant at <.05; bphen is the full phenotypic effect; b'phen is the genetically informed phenotypic effect; bA and bC are the indirect effects of parenthood status on the phenotype.

[1]Age

[2]Age, education, income

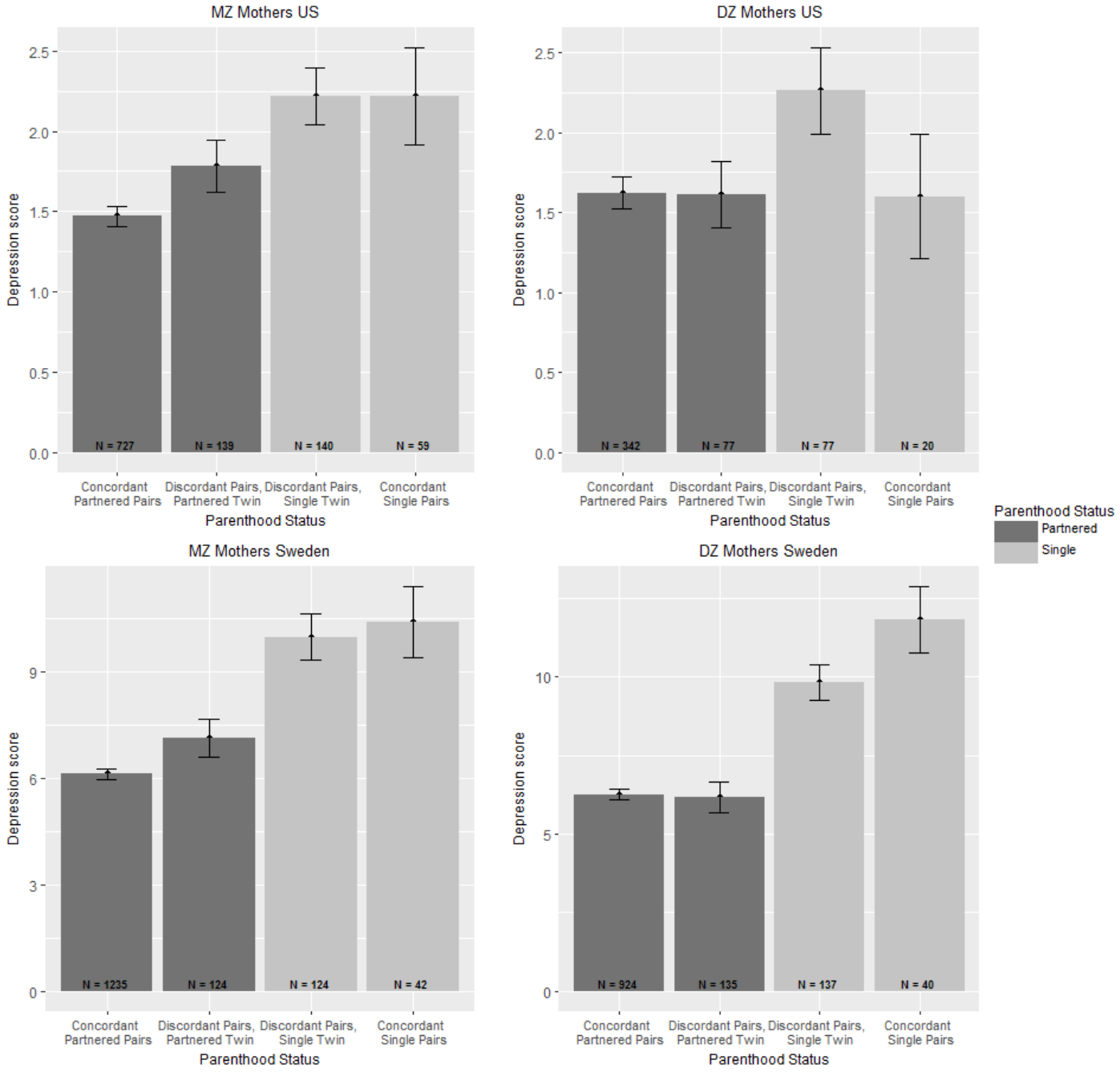
[3]Age, education, income, number of children

These effects held for each model in the series, both with no covariates (Model 1: b_{phen} = -1.871, SE = 0.127) and when covariates were progressively included (Model 2: b_{phen} = -1.965, SE = 0.126; Model 3: b_{phen} = -1.620, SE = 0.126; Model 4: b_{phen} = -1.615, SE = 0.126).

When controlling for between-family confounds, the effect of motherhood status on depression remained significant across all models, although they decreased in magnitude (Model 1: b'_{phen} = -1.645, SE = 0.309; Model 2: b'_{phen} = -1.626, SE = 0.298; Model 3: b'_{phen} = -1.145, SE = 0.252; Model 4: b'_{phen} = -1.140, SE = 0.252). This effect is illustrated in Figure 5, MZ Mothers Sweden panel in the bottom left. Concordant pairs of partnered MZ twins, in the darker, leftmost bar, report significantly lower levels of depression than concordant pairs of single MZ twins, in

the lighter, rightmost bar. This difference can also be seen within pairs of MZ twins discordant for parenthood status, represented by the middle bars.

Figure 5. Depression comparison by zygosity – US vs. Swedish mothers



As genetic and shared environmental confounds are controlled within pairs of MZ twins, this suggests a quasi-causal effect of motherhood status on depression. Moreover, this effect persists when important theoretically-informed confounds (i.e. age, SES, number of children) are controlled, which offers robust evidence for a quasi-causal effect of parenthood status on depression.

It is noted that age and education showed a significant negative effect on depression, which was consistent with results observed in the US dataset, whereas income appeared to have a significant *positive* effect on depression, such that higher income was correlated with higher depression levels. This effect persisted when income and education were introduced in the model by themselves, which indicates that it is not a result of multicollinearity.

Study 3: Cross-cultural comparison between Sweden and the US

Heritability estimates

Heritability of depression associated with parenthood status (parameter b_A in Table 5) was compared between the US and Sweden. For that purpose, depression scores were transformed into z-scores, and twin pairs from the two countries were included in a four-group model (i.e. MZ US mothers, DZ US mothers, MZ Swedish mothers, DZ Swedish mothers). Results showed that setting parameter b_A to be the same between countries did not significantly decrease the fit of the model. In other words, no significant difference in heritability of depression associated with parenthood status was observed between the two countries ($\chi^2 = .020$, $df = 1$, $p = 0.88$).

Model comparisons

Phenotypic and biometric model estimates of the impact of parenthood status on depression were examined in a four-group model in order to identify possible interactions with

culture. Phenotypically, depression scores of single parents were significantly higher than those of partnered parents in both countries. Moreover, there was a significant difference between estimates from the US and Sweden, suggesting an interaction with cultural background (χ^2= *29.76, df = 1, p <.001*).

Table 5 - Cross-cultural comparison
Standardized Parameter Estimates for Phenotypic and Biometric Models for Depression

Estimate (se)	UWTR	STR
Phenotypic model		
b_{phen}	**-0.093 (0.028)**	**-0.288 (0.023)**
Goodness of Fit		
RMSEA (CFI/TLI)	.040 (.987/.979)	
Model Comparison		
$b_{phenSweden}$=b_{phenUS}		
Wald test (df)	**29.76 (1)**	
P	<0.001	
Biometric model		
b_A	-0.100 (0.308)	-0.003 (0.623)
b_C	0	-1.998 (12.674)
b'_{phen}	-0.079 (.043)	**-0.220 (0.048)**
Covariates		
Age	**-0.099 (0.022)**	**-0.221 (0.022)**
Income	**-0.129 (0.028)**	**0.063 (0.028)**
Education	**-0.078 (0.019)**	**-0.144 (0.016)**
Number of children	0.017 (0.021)	0.022 (0.018)
Goodness of Fit		
RMSEA (CFI/TLI)	.040 (.987/.979)	
Model Comparison		
$b_{ASweden}$=b_{AUS}		
Wald test (df)	.020 (1)	
P	.88	
$b'_{phenSweden}$=b'_{phenUS}		
Wald test (df)	**4.81 (1)**	
P	0.03	

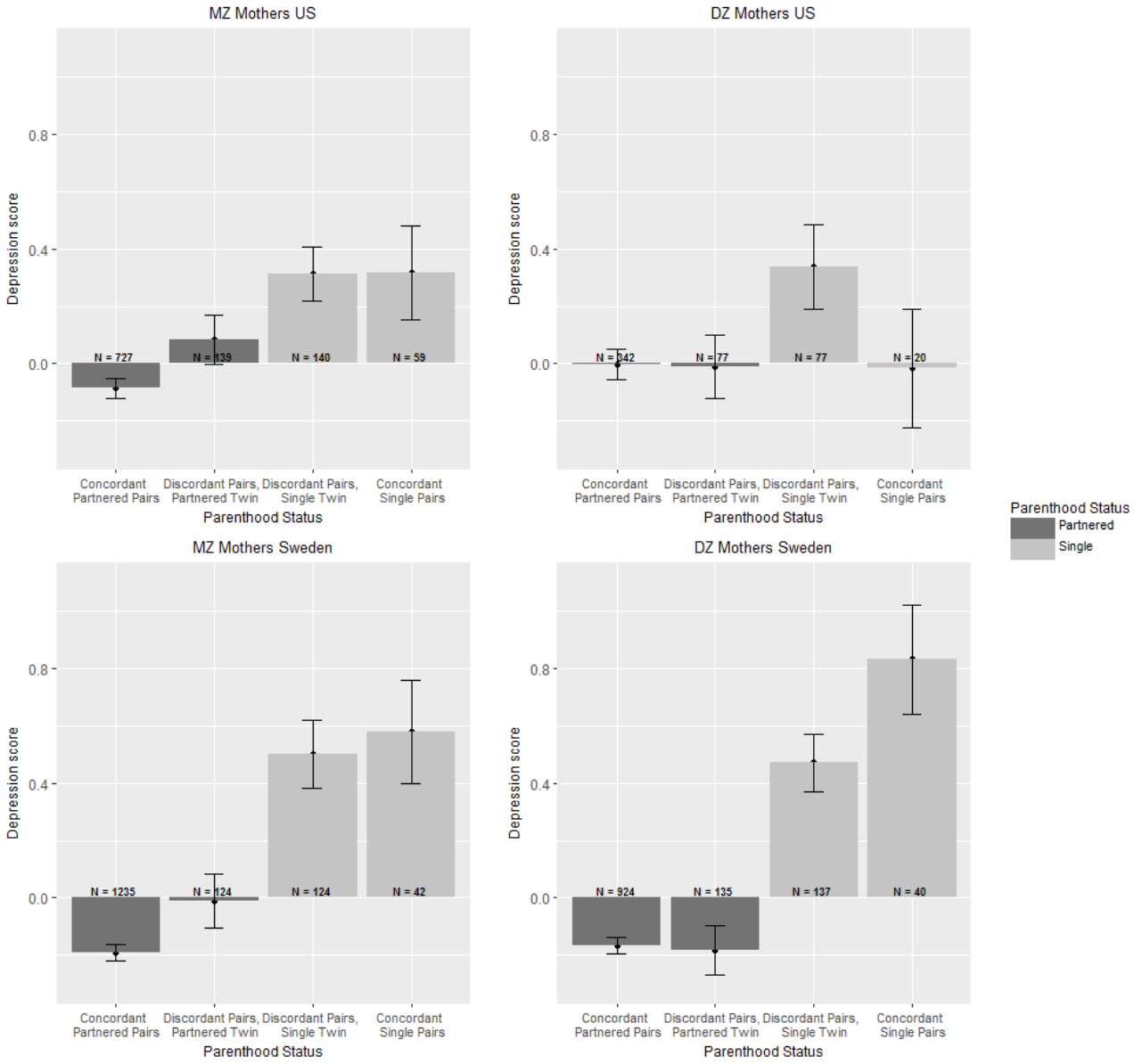
Note. Bolded values are significant at <.05;

bphen is the full phenotypic effect; b'phen is the genetically informed phenotypic effect;

bA and bC are the indirect effects of parenthood status on the phenotype.

When between-family confounds were controlled, the effect of parenthood status on depression became non-significant in the US, while remaining significant in Sweden. As was the case with the phenotypic effect, the two groups differed significantly ($\chi^2 = 4.81$, $df = 1$, $p = 0.03$). Results indicate that differences between single and partnered parents in Sweden are larger than those between their American counterparts. This difference is illustrated in Figure 7 using standardized depression scores (i.e. z-scores).

Figure 7. Standardized depression comparison by zygosity – US vs. Swedish mothers



Study 4: Phenotypic and biometric analysis of the association between single parenthood and other mental health outcomes

Due to lack of availability of appropriate mental health measures in the Swedish dataset, these analyses were only conducted using the US dataset. Complete results are described for US mothers in Table 4 and Figure 6.

Phenotypic and biometric models – Anxiety

The correlation between depression and anxiety in our sample is .58, and the results of the phenotypic and quasicausal models examining anxiety scores are highly similar to those of the depression models previously described. In the phenotypic models, anxiety levels differed significantly (i.e. $p<.05$) between single and partnered mothers such that partnered mothers reported significantly lower anxiety scores than their single counterparts. These effects held for each model (Model 1: b_{phen} = -0.643, SE = 0.102; Model 2: b_{phen} = -0.651, SE = 0.102; Model 3: b_{phen} = -0.379, SE = 0.097; Model 4: -0.378, SE = 0.097).

A series of bivariate quasi-causal models were then fitted to test whether these results are consistent with a causal hypothesis. Motherhood status continued to have a significant effect on anxiety in Model 1 (b'_{phen} *= -0.444, SE = 0.172)*. This effect is apparent in Figure 6, MZ Mothers US panel in the top left. Concordant partnered MZ twins, in the darker, leftmost bar, reported significantly lower levels of anxiety than concordant single MZ twins, in the lighter, rightmost bar. The effect was similar within pairs of MZ twins discordant for parenthood status (inner bars), and was approximated by effects observed in the DZ sample (Figure 6, DZ Mother US top right-hand panel), which suggests that genetic and shared environmental confounds are not accountable for this observed difference.

Table 4 - UWTR mothers

Unstandardized Parameter Estimates for Phenotypic and Biometric Models for Anxiety, Perceived Stress, and Coping

	Estimate (SE)	Model 1 No cov	Model 2 Demographic cov	Model 3 Add SES cov	Model 4 All cov
Anxiety	Phenotypic model				
	b_{phen}	**-0.643 (0.102)**	**-0.651 (0.102)**	**-0.379 (0.097)**	**-0.378 (0.097)**
	Biometric model				
	b_A	-0.692 (0.690)	-0.732 (0.694)	-0.970 (1.203)	-0.968 (1.219)
	b_C	0	0	0	0
	b'_{phen}	**-0.444 (0.172)**	**-0.430 (0.171)**	-0.243 (0.146)	-0.244 (0.145)
	Covariates				
	Age		**-0.091 (0.030)**	**-0.223 (0.040)**	**-0.206 (0.046)**
	Income			**-0.126 (0.036)**	**-0.125 (0.036)**
	Education			**-0.096 (0.031)**	**-0.099 (0.031)**
	Number of children				-0.034 (0.054)
	Goodness of Fit				
	RMSEA (CFI/TLI)	**.009 (.996/.997)**	**.040 (.908/.923)**	**.012 (.994/.991)**	**.007 (.998/.997)**
Perceived Stress	Phenotypic model				
	b_{phen}	**-0.652 (0.278)**	**-0.617 (0.278)**	-0.174 (0.253)	-0.166 (0.253)
	Biometric model				
	b_A	-1.877 (1.607)	**-3.348 (1.657)**	13.437 (103.199)	9.605 (104.386)
	b_C	0	0	0	0
	b'_{phen}	-0.095 (0.439)	0.499 (0.437)	-0.007 (0.321)	-0.064 (0.323)
	Covariates				
	Age		**-0.134 (0.068)**	**-0.453 (0.083)**	**-0.412 (0.105)**
	Income			**-0.251 (0.082)**	**-0.236 (0.082)**
	Education			**-0.321 (0.077)**	**-0.334 (0.079)**
	Number of children				-0.160 (0.146)
	Goodness of Fit				
	RMSEA (CFI/TLI)	**.016 (.968/.976)**	**.039 (.726/.772)**	**.009 (.996/.994)**	**.007 (.998/.997)**
Coping	Phenotypic model				
	b_{phen}	-0.060 (0.108)	-0.078 (0.108)	-0.174 (0.101)	-0.179 (0.101)
	Biometric model				
	b_A	0.391 (0.586)	0.665 (0.582)	-0.097 (0.948)	-0.125 (0.962)
	b_C	0	0	0	0
	b'_{phen}	-0.175 (0.170)	-0.278 (0.170)	-0.161 (0.145)	-0.161 (0.145)
	Covariates				
	Age		**0.059 (0.020)**	**0.102 (0.032)**	0.063 (0.038)
	Income			0.037 (0.035)	0.033 (0.034)
	Education			**0.249 (0.033)**	**0.259 (0.033)**
	Number of children				0.099 (0.052)
	Goodness of Fit				
	RMSEA (CFI/TLI)	**.024 (.938/.954)**	**.028 (.883/.902)**	**.018 (.984/.977)**	**.016 (.991/.985)**

Note. Bolded values are significant at <.05; bphen is the full phenotypic effect; b'phen is the genetically informed phenotypic effect; bA and bC are the indirect effects of parenthood status on the phenotype.

[1]Age

[2]Age, education, income

[3]Age, education, income, number of children

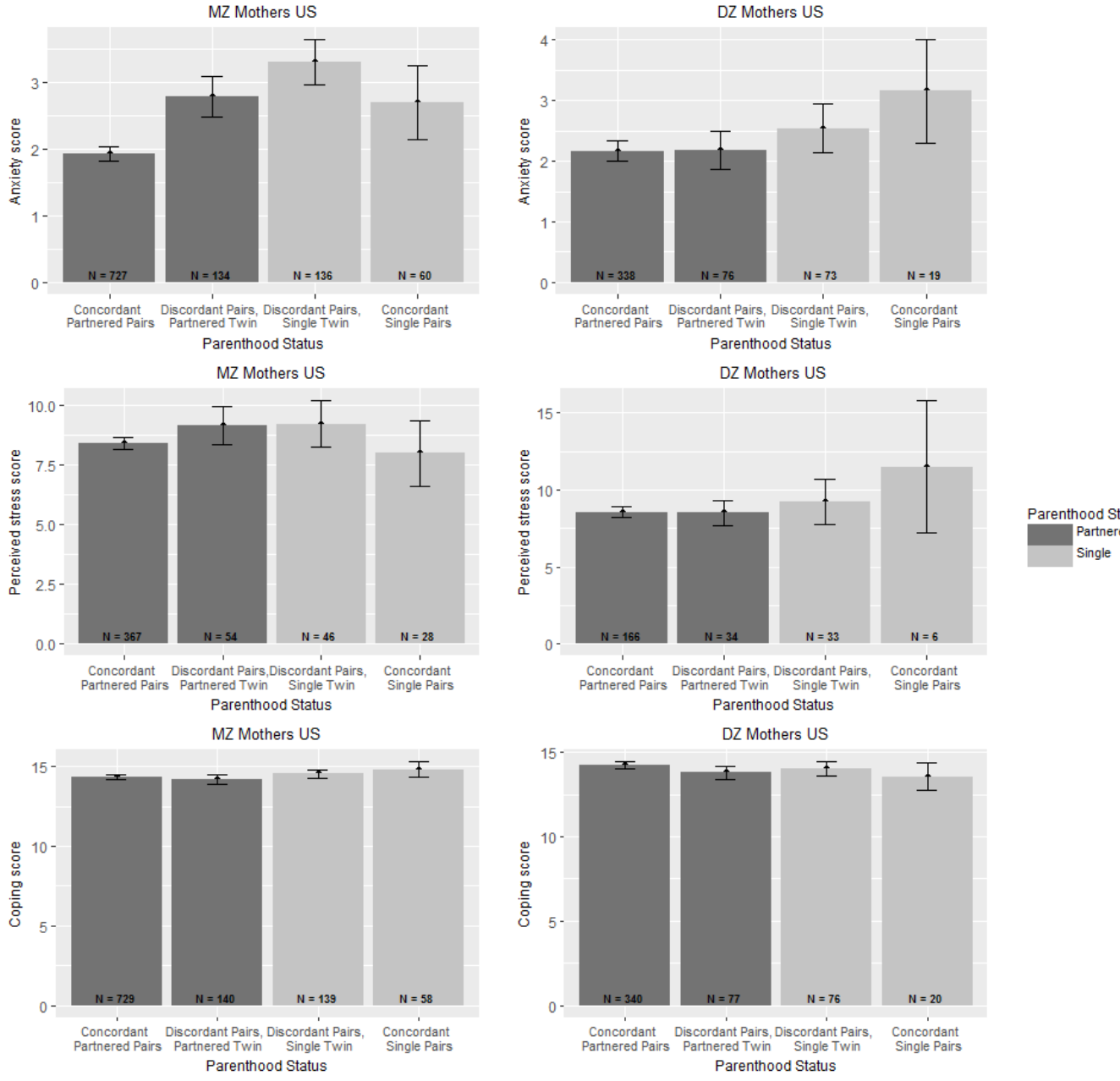
When controlling for age, the effect remained significant (Model 2: b'_{phen} = *-0.430, SE = 0.171).* However, when SES covariates were introduced, the effect became non-significant for both models which included these controls (Model 3: b'_{phen} = *-0.243, SE = 0.146*; Model 4: b'_{phen} *= -0.244, SE = 0.145).* Across all models, the effect of age and SES variables on anxiety was significant and consistent, such that mothers who were older, earned more, and had higher levels of education reported lower levels of anxiety. Number of children did not have a significant impact on anxiety levels.

Phenotypic and biometric models – Perceived Stress

In the phenotypic model, we observed a significant effect of parenthood status on perceived stress, in that partnered mothers reported significantly lower levels of perceived stress than their single counterparts (Model 1: b_{phen} = -0.652, SE = 0.278). This effect remained significant when controlling for age (Model 2: b_{phen} = -0.617, SE = 0.278), but became smaller and non-significant when SES controls were introduced (Model 3: b_{phen} = -0.174, SE = 0.253; Model 4: -0.166, SE = 0.253). All quasi-causal models yielded non-significant results (Model 1: b'_{phen} = -0.095, SE = 0.439; Model 2: b'_{phen} = 0.499, SE = 0.437; Model 3: b'_{phen} = -0.007, SE = 0.321; Model 4: b'_{phen} = -0.064, SE = 0.323), suggesting that the significant phenotypic effects found in Model 1 and Model 2 were confounded by genetic factors. Indeed, examining the inner bars on Figure 6, middle left panel, demonstrates that the perceived stress scores of discordant MZ twins are essentially identical regardless of their parenthood status, consistent with genetic selection. Model results indicate that age, income, and education have a significant impact on

perceived stress, such that older, more educated mothers with higher income report less perceived stress.

Figure 6. Mental health outcomes by zygosity – US mothers



Phenotypic and biometric models – Coping

We found no evidence of an effect of parenthood status on coping when comparing single and partnered twins. This was the case in both the phenotypic models (Model 1: b_{phen} = -0.060, SE = 0.108; Model 2: b_{phen} = -0.078, SE = 0.108; Model 3: b_{phen} = -0.174, SE = 0.101; Model 4: -0.179, SE = 0.101) and the biometric models (Model 1: b'_{phen} = -0.175, SE = 0.170; Model 2: b'_{phen} = -0.278, SE = 0.170; Model 3: b'_{phen} = -0.161, SE = 0.145; Model 4: b'_{phen} = -0.161, SE = 0.145). Model 2 results indicated a significant impact of age on coping, such that older mothers exhibited higher levels of coping. This effect remained significant when income and education were introduced in Model 3. Education had a significant impact on coping, such that higher levels of education correlated with better coping, whereas income did not have a significant effect. When introducing number of children in Model 4, only education remained significant.

Fathers

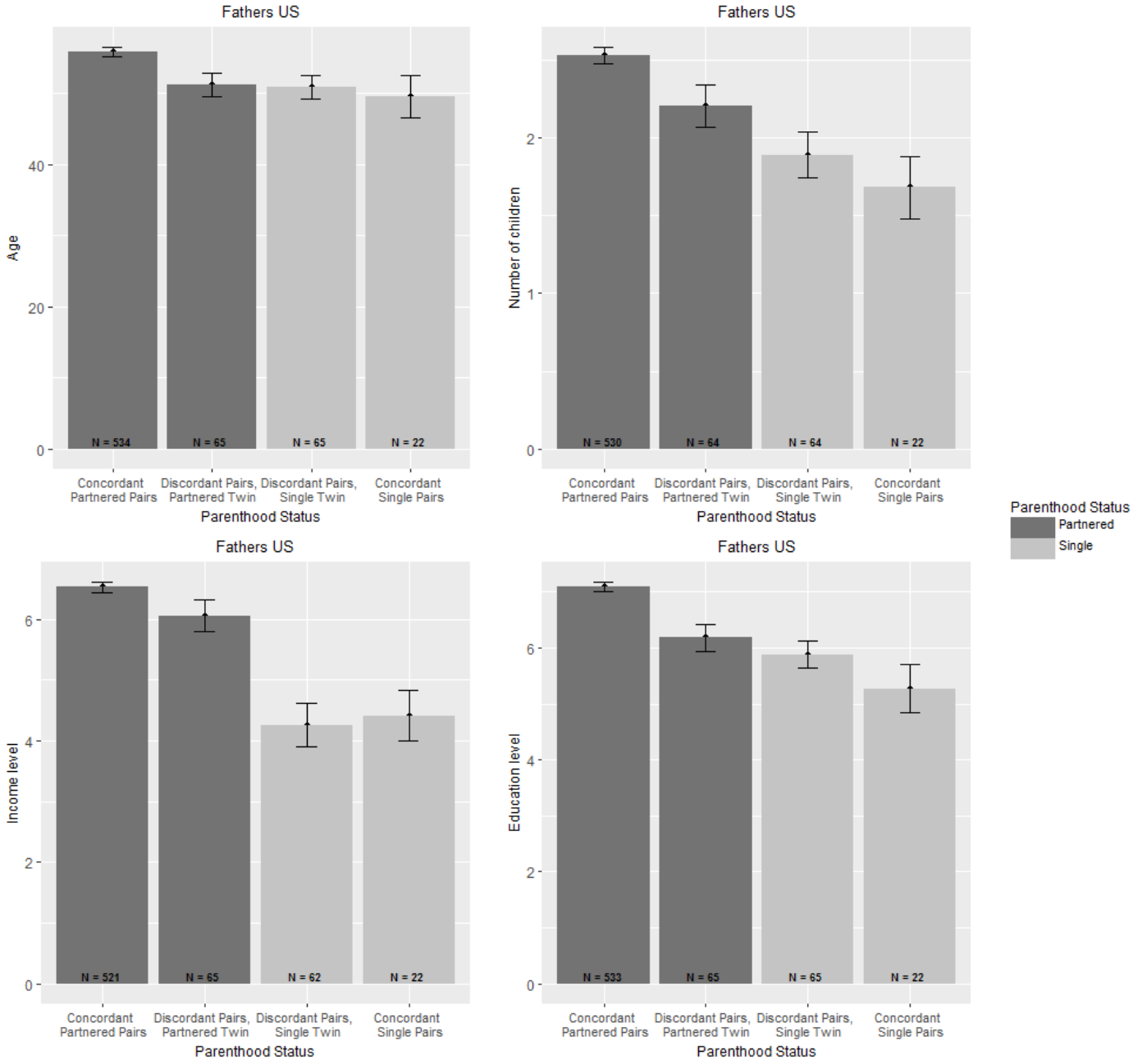
Partnered fathers in the US appeared to be older than their single counterparts, with an average age difference of 5.4 years. Partnered fathers also reported more children ($t(172) = 5.55$, $p < .05$) and higher levels of household income ($t(147) = 8.84$, $p < .05$) and education ($t(168) = 6.32$, $p < .05$) than single fathers (see Figure 8).

Partnered US fathers reported lower mean levels of depression than their single counterparts ($t(149) = -3.31$, $p < .05$). The same effect was observed for anxiety, in that partnered fathers reported significantly lower anxiety levels than single fathers ($t(139) = -2.64$, $p < .05$). No significant mean differences in perceived stress ($t(74) = -0.12$, $p = .89$) or coping scores ($t(159) = -0.84$, $p = .40$) were reported between single and partnered fathers.

Due to small sample size, phenotypic and quasicausal model results for father samples could not be interpreted. Instead, we present graphical illustration of means comparisons by

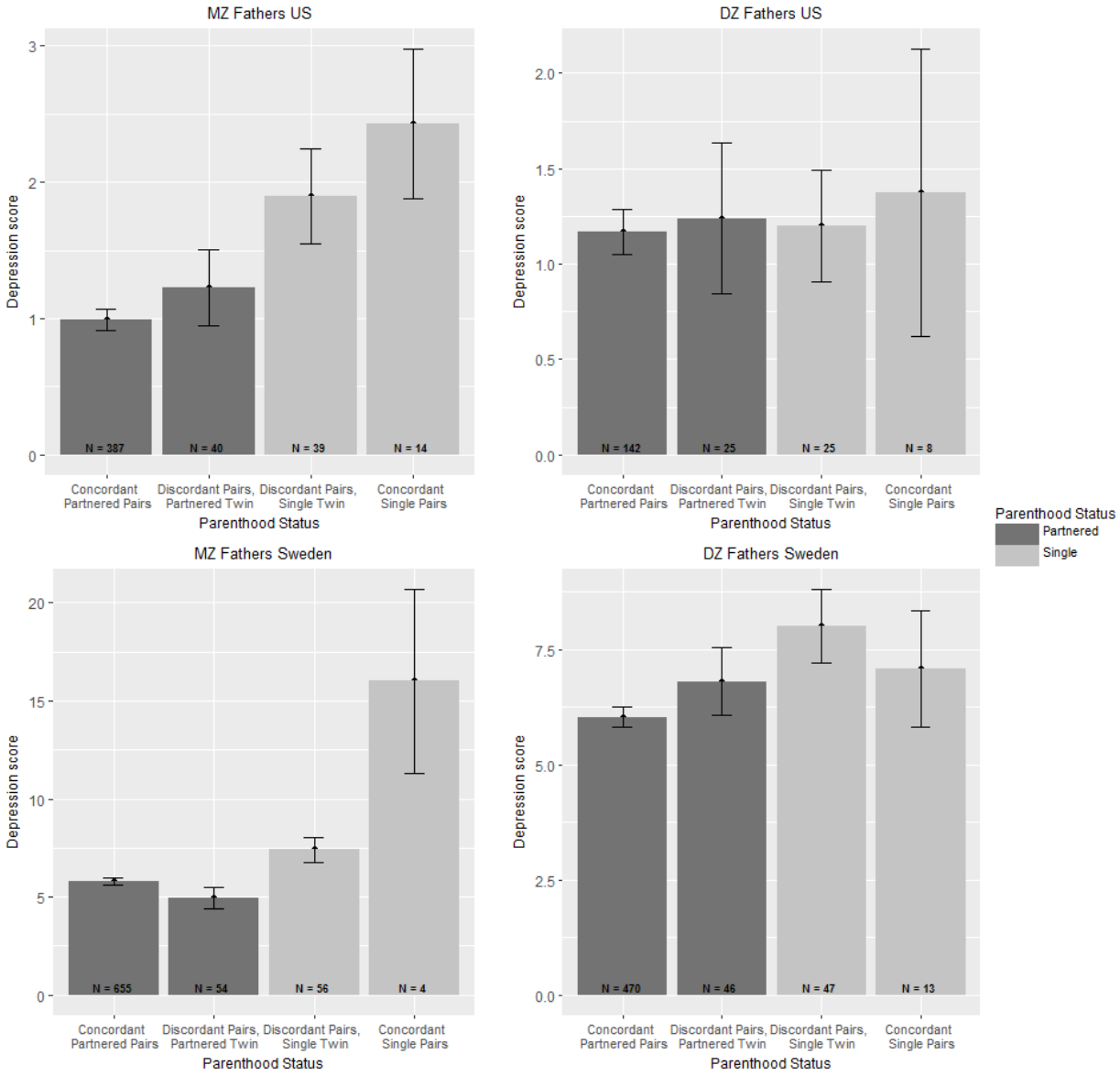
zygosity (i.e. MZ and DZ) between and within twin pairs. The effect of parenthood status on depression is illustrated in the top left panel of Figure 9.

Figure 8. Demographic variables – US fathers



Due to small sample size, phenotypic and quasicausal model results for father samples could not be interpreted. Instead, we present graphical illustration of means comparisons by zygosity (i.e. MZ and DZ) between and within twin pairs. The effect of parenthood status on depression is illustrated in the top left panel of Figure 9.

Figure 9. Depression comparison by zygosity – US and Swedish fathers



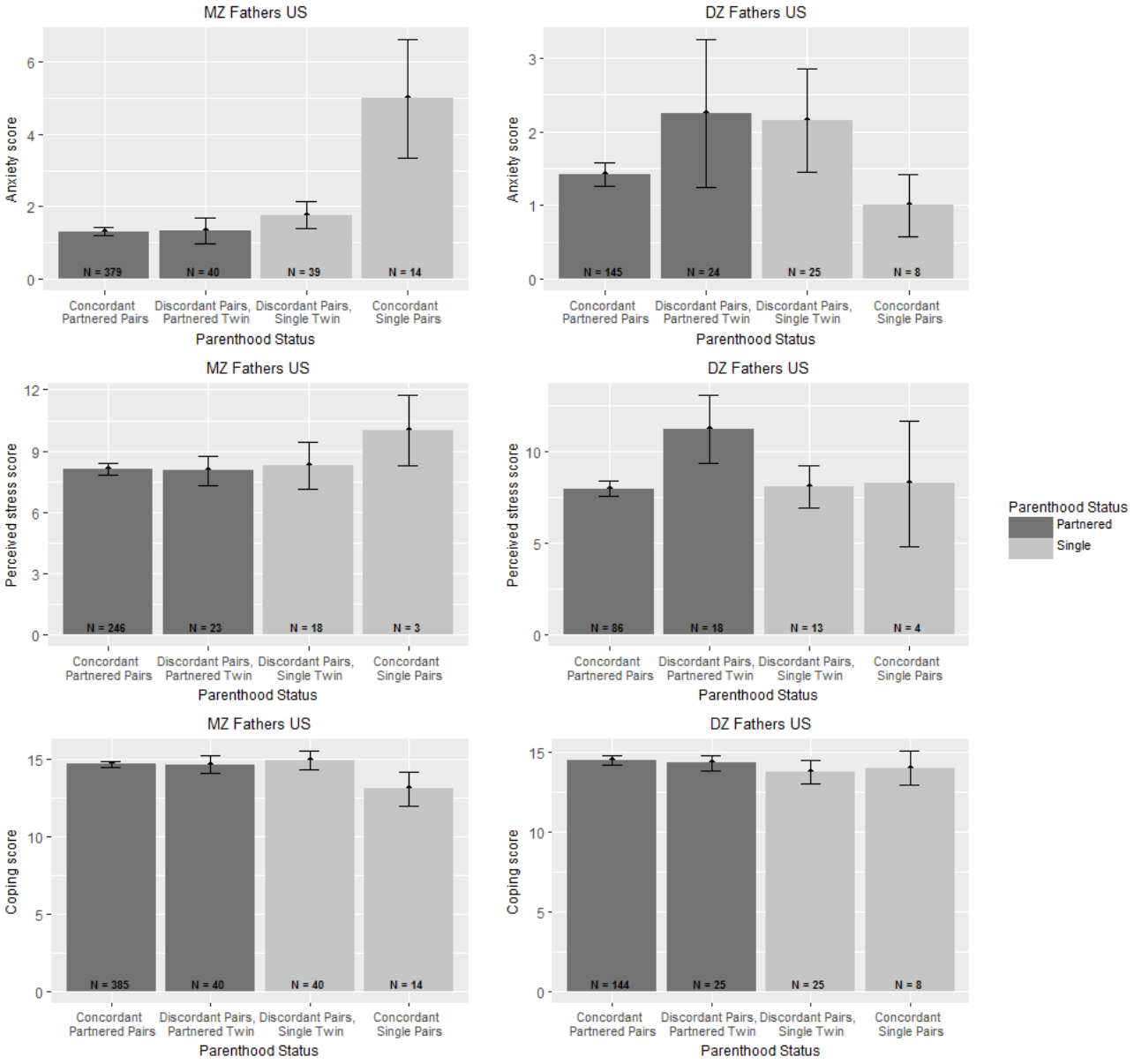
Concordant pairs of partnered MZ twins (darker, leftmost bar) reported lower levels of depression than concordant pairs of single MZ twins (lighter, rightmost bar). This effect persisted within pairs of MZ twins discordant for parenthood status (inner bars), although its magnitude was reduced. This suggests the existence of a quasi-causal effect of parenthood status on depression, because within-pair MZ comparisons control for genetic and shared environmental confounds.

The existence of a genetic confound can be observed by comparing MZ and DZ twins (top row panels of Figure 9). Within discordant pairs of twins, MZ partnered twins report lower mean depression scores than their single co-twins. Conversely, DZ twins in discordant pairs do not differ in their reported mean depression scores. This difference illustrates a genetic confound, because the within-pair difference between DZ twins includes the genetic difference between them.

As in the case of depression, other mental health outcomes' phenotypic and quasicausal model results for fathers could not be interpreted due to small sample sizes in some groups. Instead, we present graphical illustration of means comparisons between and within twin pairs.

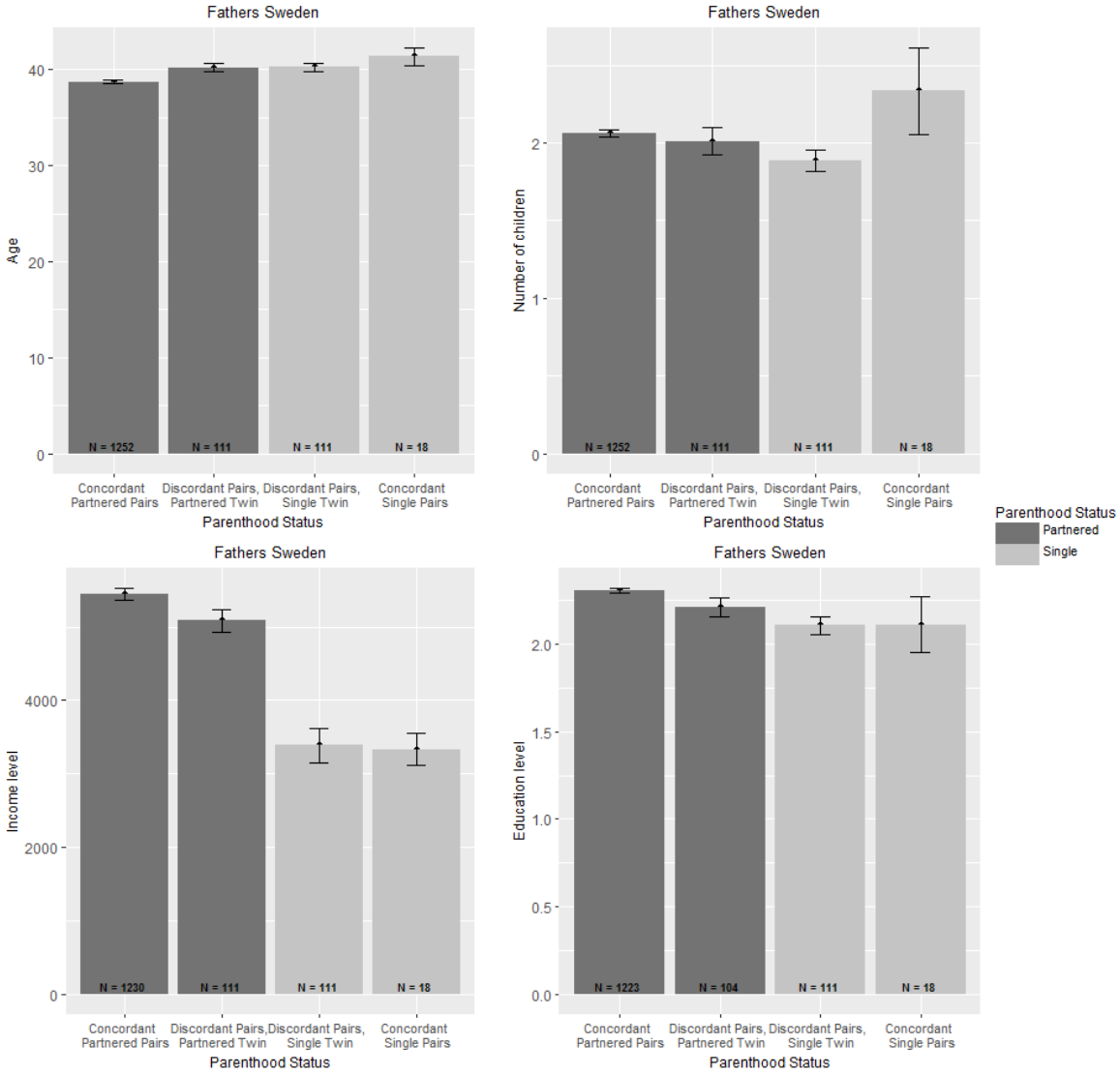
Figure 10 illustrates the effect of parenthood status on anxiety, perceived stress, and coping from top to bottom left-hand panels. It is noted that single fathers in concordant pairs report significantly higher levels of anxiety and lower levels of coping than their discordant counterparts and all groups of partnered fathers. Nevertheless, the inner bars in each panel indicate similar levels of anxiety and essentially identical levels of perceived stress and coping between discordant MZ twins, which suggests the absence of a quasi-causal effect. In other words, Figure 10 indicates that genetic and shared environmental factors confound the association between parenthood status and these mental health indicators.

Figure 10. Mental health outcomes by zygosity – US fathers



In Sweden, partnered fathers' average age was similar to that of single fathers (less than one year difference). Partnered fathers also reported a higher income ($t(427) = 17.43$, $p < .05$), education ($t(360) = 4.76$, $p < .05$), and number of children ($t(359) = 3.25$, $p < .05$) than their single counterparts. Interestingly, the average number of children of single fathers from concordant pairs was highest, as shown in Figure 11.

Figure 11. Demographic variables – Swedish fathers



The results present in the Swedish dataset closely approximate those found in the US dataset. Swedish partnered fathers reported lower levels of depression than their single counterparts ($t(300) = -8.17$, $p < .05$). Moreover, the presence of a quasi-causal effect is illustrated in the bottom left panel of Figure 9. Concordant pairs of partnered MZ twins reported significantly lower levels of depression than concordant pairs of single MZ twins. A similar – although smaller – difference could be observed within pairs of MZ twins discordant for

parenthood status, which indicates the presence of a quasi-causal effect. As expected, the effect of parenthood status on depression was attenuated in DZ twins who share on average only 50% of their genes. Nevertheless, the effect remained robust between and within pairs of DZ twins, such that single fathers from concordant and discordant pairs exhibited higher mean depression scores than partnered fathers.

Mean outcome differences between mothers and fathers

As described above, single mothers and fathers exhibited significantly higher mean levels of depression than their partnered counterparts in both the US and Sweden. As predicted, t-test results indicated that the difference between single and partnered fathers was smaller in magnitude than the difference between single and partnered mothers in both samples. The same pattern of results was observed for anxiety in the US dataset, where the difference between fathers was smaller in magnitude than the difference between mothers. No significant differences in perceived stress or coping were noted between single and partnered twins.

Discussion

The results of the current study shed light on the associations between parenthood status and several mental health outcomes: (i.e. depression, anxiety, perceived stress, and coping). Using a genetically informed research design, we tested whether the association between motherhood status and mental health is accounted for by non-random selection, causation, or both, and whether there are cross-cultural differences between the US and Sweden. Additionally, we descriptively examined whether the effect of single parenthood is different for mothers and fathers. No prior empirical research has examined the impact of single parenthood on mental health outcomes using a genetically informed design. This study also offers unique insight into the impact of culture on the association between single parenthood and depression by examining

samples from two different cultures. Lastly, this is one of few studies to provide information regarding single fatherhood.

Based on prior findings, we hypothesized that single parents would exhibit significantly higher levels of depression, anxiety, and perceived stress and lower levels of coping than their partnered counterparts across genders and cultures. We expected these differences to remain significant when controlling for genetic and shared environmental confounds, as well as for theoretically-informed covariates (i.e. age, education, income, number of children living in the home). Additionally, we expected to observe a "gene-culture" interaction effect on heritability and regression estimates and variation in estimates, such that single parents in Sweden would differ less from their partnered counterparts than single parents in the US. Lastly, we predicted that single and partnered fathers will differ less than mothers in mean levels of mental health outcomes.

Our results provided mixed support for our hypotheses. As expected, single mothers and fathers reported higher mean levels of depression and anxiety than their partnered counterparts. The detrimental effect of single parenthood did not, however, extend to perceived stress and coping. Moreover, contrary to our hypotheses, when introducing socioeconomic confounds the quasi-causal association between single motherhood and depression remained significant only in Sweden. These findings are discussed below.

Mothers

Study 1: Descriptive analysis of single parenthood and mental health outcomes

The purpose of this study was to provide full descriptions of our twin samples in the US and Sweden given the uniqueness of the study population and the scarcity of descriptive

information on perceived stress and coping. We hypothesized that single parents will report worse mental health outcomes than their partnered counterparts.

Our descriptive results painted a nuanced picture of the association between single motherhood and mental health outcomes. Findings for depression confirmed our hypotheses and replicated prior work. Specifically, both in the US and Sweden, single mothers reported significantly higher depression scores than their partnered counterparts. Depression and anxiety are highly comorbid in the general population (Sartorius, Üstün, Lecrubier, & Wittchen, 1996) and were correlated .58 in our sample. Unsurprisingly, results for anxiety closely approximated what was observed for depression, in that single mothers reported higher anxiety levels than their partnered counterparts. Conversely, results for perceived stress and coping did not support our hypotheses. Instead, no significant differences were observed between single and partnered mothers on these measures.

It is important to note demographic differences between single and partnered mothers in our samples. Consistent with prior research (Burstrom et al., 2010; Crosier et al., 2007; D'Ercole, 1988), we found that single mothers reported less education and household income than partnered mothers. In both countries, single mothers were slightly older than partnered mothers (1-1.5 years). Partnered mothers in Sweden reported more children on average than their single counterparts, whereas single and partnered US mothers did not differ in number of children.

Study 2: Phenotypic and biometric analysis of the association between single parenthood and depression

The purpose of the present study was to advance knowledge on the association between single parenthood and depression by using a genetically informed design. All current research on single parenthood is limited by nonexperimental designs, leaving numerous third variables (e.g.

genetic and environmental selection factors) as possible alternative hypotheses to the social causation hypothesis. The quasi-experimental properties of twin studies can help elucidate causality where experimental control is not possible (Turkheimer & Harden, 2013). We used a genetically informed design to parse selection from potential causation in the association between single parenthood and depression, and predicted that single parenthood would be associated with higher levels of depression. The twin models were, unfortunately, underpowered to detect effects of single fatherhood on mental health outcomes, so we only presented statistical results of single motherhood analyses.

At the full-sample level, single mothers reported being more depressed both in the US and in Sweden, controlling for age, number of children, and socioeconomic variables (i.e. education and income). This is consistent with prior research which indicates that while these factors, especially socioeconomic differences, have an important impact on the health and well-being of single mothers, they do not fully explain health disparities between groups. However, when accounting for genetic and shared-environmental confounds, a nuanced picture emerged across cultures.

As expected, the quasi-causal effect of single motherhood on depression in the US remained significant in models controlling for age. Nevertheless, when adding socioeconomic variables into the quasi-causal model, the effect became nonsignificant. In other words, SES mediated the quasi-causal relationship between single motherhood and depression in the US, such that single parenthood lead to low SES which then led to depression. A different picture emerged in Sweden, where the quasi-causal effect of single motherhood on depression remained significant after controlling for all confounds. This suggests that in Sweden single motherhood has a direct quasi-causal effect on depression.

Study 3: Cross-cultural comparison between Sweden and the US

Study 3 hypotheses were based on prior research on the impact of SES on depression (Lorant et al., 2003) and on differences in public policy between the US and Sweden, leading to greater social equality and support for single mothers in Sweden. Given these factors, we hypothesized that differences between single and partnered mothers will be smaller in Sweden than in the US, both phenotypically and quasi-causally. Contrary to our hypotheses, phenotypic differences between single and partnered mothers were significantly larger in magnitude in Sweden compared to the US. Moreover, when confounds were controlled, the effect became nonsignificant in the US while remaining significant in Sweden, suggesting that single motherhood has a direct impact on depression in Sweden, whereas the same is not true in the US.

Single motherhood and depression: conclusions

Our findings suggest that single mothers in Sweden are not only worse off than partnered mothers in Sweden, but may be worse off than their US counterparts. Although single and partnered mothers in both countries reported differences in education and household income, these discrepancies appeared to play a bigger role in the US than Sweden in explaining differences in depression between groups. In other words, higher depression scores of single mothers in the US compared to partnered mothers can be largely accounted for by differences in SES. Comparatively, in Sweden single mothers are significantly more depressed than partnered mothers even after accounting for the effect of differences in SES. Additionally, of note, whereas the effect of income on depression was significant in both our US and Swedish samples, the direction of the effect was opposite in the two datasets. In the US, as expected, higher income levels were associated with lower levels of depression. Conversely, in Sweden higher income levels were associated with *higher* levels of depression.

It is possible that participation in the labor market explains both of these surprising results. Level of employment is higher among Swedish (Burstrom et al., 2010) than US single mothers, most of whom do not work if they receive welfare assistance (Blau & Tekin, 2007; Edin & Lein, 2007). Moreover, single mothers often differ from partnered mothers in the classes of occupation they hold (Burstrom et al., 2010). When they do work, single mothers in the US hold low-paying jobs in higher proportion than other US job holders, as well as single mothers in other industrialized countries (Casey & Maldonado, 2012). Evidence for the mental health benefits of employment is mixed, with some studies suggesting that single mothers benefit from current employment (Zabkiewicz, 2010) and other finding no effect or a detrimental one (Baker & North, 1999).

It is conceivable that higher employment rates among single mothers in Sweden paradoxically lead to higher levels of dissatisfaction, and consequently depression, related specifically to their single motherhood status. The more Swedish single mothers work and earn, the harder it may be for them to balance their roles as head of household/primary earner/caregiver, so that higher incomes would be associated with higher depression levels. Conversely, in the US, policy measures do not allow single mothers the same flexibility, as working is more likely to yield a lower wage *and* remove their eligibility for welfare. As stated above, assistance in the US is based on socioeconomic need, making this aspect of a single mother's life especially salient, and exposing her to more stressors related to socioeconomic well-being. However, it is possible that this forced choice (work vs. welfare) enables single mothers to be better able to fulfill other roles that are important for their well-being. Unfortunately, detailed employment data was not available in our dataset. Future studies are

needed to explore employment participation and mental health outcomes of single and partnered mothers in a genetically informed design.

Importantly, our results should not be misinterpreted as suggesting that in the US single mothers are not more depressed than their partnered counterparts. Our results did indicate a quasi-causal effect of single motherhood on depression, which indicates that in a pair of MZ twins where one is a single mother and one is a partnered mother, the single twin is still more depressed than the partnered twin. One explanation for this effect, supported by our results, is that single motherhood is directly related to a significant decrease in resources, especially in the US, which then leads to depressive symptoms. The connection between single motherhood, lower SES, and more negative mental health outcomes is supported by prior research discussing the role of income in health (Brennenstuhl et al., 2012; Burstrom et al., 2010). Nevertheless, our data shows that SES does not fully explain the connection between single motherhood and depression, as this association remains significant in Sweden, a country where single parents are offered significant support by the state.

Other explanations for the association between single motherhood and depressive symptoms include the loss of intangible resources. It is possible that mothers who become single after being previously partnered lose not only financial security, but social support systems including romantic partner, extended family, and possibly family friends (Cairney et al., 2003). Additionally, separated and divorced mothers also experience a possible loss of their role as wife and caregiver, which has been associated with declines in mental health outcomes (Erdwins, Buffardi, Casper, & O'Brien, 2001). Future studies should compare single/never-married to separated and divorced mothers in order to tease apart possible effects of separation and loss of partnership.

Lastly, it is possible that single parenthood has, across cultures and societies, negative associations or stigma, which can lead to feelings of alienation and increased stress (Miller & Kaiser, 2001). Single mothers may consequently experience not only depressive symptoms, but also physical health consequences such as increased fatigue or risk for cardiovascular disease. Prior research has found such associations (Baker & North, 1999; Broussard et al., 2012; Floderus et al., 2008; Fritzell et al., 2007; Sarfati & Scott, 2001; Young et al., 2005), but genetically informed analyses are needed to eliminate the possibility of genetic confounds of these associations.

Study 4: Phenotypic and biometric analysis of the association between single parenthood and other mental health outcomes

The majority of research on single parenthood and mental health outcomes focuses on depression (Collings et al., 2013; Manuel, Martinson, Bledsoe-Mansori, & Bellamy, 2012; Sperlich et al., 2011b; Wang, 2004) or general indicators of health such as "self-rated health" (Colton et al., 2015; Fritzell et al., 2012) or "common mental disorders" (Cooper et al., 2008). The present study aimed to add to the existing base of research by examining three specific mental health outcomes: anxiety, perceived stress, and coping.

As expected, results for anxiety closely approximated those for depression. Specifically, when examined in the whole sample (equivalent to the general population) single mothers reported significantly higher levels of anxiety than their partnered counterparts. This effect remained significant in models controlling for age, but became nonsignificant when SES controls were introduced in the model. In other words, socioeconomic status mediated the quasi-causal relationship between single motherhood and anxiety, as was the case for depression.

When examined phenotypically, single mothers also appeared to report higher perceived stress scores than partnered mothers. However, when controlling for SES the effect became smaller in magnitude and nonsignificant, suggesting that SES factors play an important role in perceived stress levels, and that in our sample single motherhood did not contribute to those symptoms above and beyond SES differences. The importance of SES is confirmed by prior research suggesting that social disadvantage is associated with elevated exposure to stressful life events (Avison et al., 2007; Van de Velde et al., 2014) and that income fully accounted for differences in total daily hassles between single and partnered mothers (Compas & Williams, 1990).

Lastly, single and partnered mothers did not differ on coping scores. Prior research examining multiple facets of coping found that single and partnered mothers did not differ on most coping subscales (Compas & Williams, 1990), possibly because while stressors and setbacks differ for the two groups, both single and partnered mothers utilize coping strategies equally in order to manage daily strain.

Age and SES had significant effects for both anxiety and perceived stress, such that higher age and SES was consistently associated with lower anxiety and perceived stress. Conversely, education was the only SES indicator that appeared to have a consistent significant impact on coping, in that higher levels of education were significantly associated with better coping. Interestingly, as found in depression models, the number of children living with a mother was not a significant predictor of mental health.

Fathers

Given the rarity of single father studies to date, one of our study aims was to add to the current research in the field. The twin models were, unfortunately, underpowered to detect

effects of single fatherhood on mental health outcomes, so we presented statistical results of single motherhood analyses and descriptive results of single fatherhood analyses. Single fathers were younger than partnered fathers in the US, but older in Sweden. In both countries, partnered fathers reported significantly more children on average than their single counterparts, as well as more income and education.

Both in the US and Sweden, single fathers reported significantly higher depression scores than their partnered counterparts. These differences were observed both between pairs of twins concordant for parenthood status (i.e. both are single or partnered parents), as well as within pairs of twins discordant for parenthood status (i.e. one is partnered and the other is single). Similar results were observed for anxiety, in that single fathers reported higher anxiety levels than their partnered counterparts. Contrary to our hypotheses, no significant differences in perceived stress or coping were observed between single and partnered fathers.

When comparing mothers and fathers, our results suggest that mean depression and anxiety scores were higher for single mothers than for single fathers. The same was true for partnered mothers compared to fathers, and is in line with prior research showing that in general women tend to report more anxiety and depression than men (McLean, Asnaani, Litz, & Hofmann, 2011; Nolen-Hoeksema, 2001). Nevertheless, these baseline variations are not enough to explain the magnitude of differences between single and partnered twins, which was larger for mothers than fathers in our study. Our findings suggest that single parenthood is more detrimental for mothers than for fathers with regards to depression and anxiety. This may be because single fatherhood does not have the same social, financial, and personal implications as single motherhood or because single fathers receive more help when they are the custodial

parent. Larger samples are needed in order to facilitate genetically informed analyses and elucidate the impact of single fatherhood on mental health outcomes.

Overall Implications

The present studies extend prior research in important ways. This is the first genetically informed study to investigate the effect of single motherhood on any psychological outcome. This is the strongest available method to explore cross-sectional data, and we showed that depression is not only a byproduct of upbringing, or especially of genetic propensity, but is directly influenced by single motherhood, directly or through mediators. Furthermore, this investigation shows that single motherhood significantly impacts depression in Sweden even when we control for variables that were shown in the literature to possibly account for this effect. The effect of single motherhood on depression remained significant in our Swedish sample even when controlling for socioeconomic indicators such as income and education. Perhaps even more importantly, this is the first genetically-informed cross-cultural comparison of the effects of single motherhood on depression. Our results indicate that differences between single and partnered mothers are larger in Sweden than in the US despite sociocultural factors hypothesized to create the opposite effect. Lastly, this study adds to a small pool of research investigating the impact of single motherhood on outcomes such as anxiety, perceived stress, and coping, and an even smaller number of studies examining single fatherhood.

Our results are in line with prior investigations on US samples which reported decreases in effects of single motherhood on depression after controlling for income (Compas & Williams, 1990). Moreover, similar results were found in research comparing single and partnered mothers in Sweden to their counterparts in Britain which, like the US, adheres to a market-oriented family policy model (Burstrom et al., 2010). Burstrom et al. (2010) found, unexpectedly, that

Swedish and British single mothers showed highly similar negative health outcomes associated with motherhood status, and in some cases (i.e. limiting longstanding illness) Swedish mothers were worse off. It is noted that in our samples we did find a quasi-causal effect of single motherhood on depression in both countries. This effect was simply mediated by SES in US mothers, while in Swedish mothers it was directly observed.

Our results also add to research indicating that single parenthood is more detrimental for mothers than for fathers. While no significant differences between single and partnered parents of either gender were found for perceived stress or coping, differences in anxiety and depression were significant between groups. Unfortunately, although in line with prior work, these results cannot be considered reliable due to the scarcity of single fathers in our samples.

The impact of our findings goes beyond scientific novelty. The quasi-causal link between single motherhood and depression has important policy and healthcare implications. So far, policies aimed at supporting single mothers focus primarily on income distribution, with some countries adding provisions for child care. Our study shows that offering benefits such as money and child care is very important, but may not be enough to adequately support single mothers, who have specific mental health needs. The psychological health of a single mother does not only affect her own life, but also the life and outcomes of her children, so it is doubly important to use this kind of research in adjusting the type and level of support that single mothers receive from the state. Clinically, treatment interventions already consist of screening and customizing treatment based on people's known risk factors, such as blood pressure in the prevention and treatment of cardiovascular diseases (Kannel, 1996) or being an athlete in the treatment of eating disorders (Bratland-Sanda & Sundgot-Borgen, 2013). Our findings are a potential first step

towards customizing interventions based on parenthood status and the availability of not only socioeconomic support, but of a stable close relationship.

Limitations

Despite the strengths of our chosen method of analysis, the fact that our data is cross-sectional limits our ability to draw true causal conclusions. While the genetically informed methodology we are using is the best way to draw quasi-causal conclusions based on the type of data we have available, future studies should seek to replicate our findings using a longitudinal design. We also did not have enough data to discuss race differences, which would have been especially interesting given possible disparities in covariates. Thus, our findings generalize only to populations similar to our sample, which is primarily Caucasian and representative of Western culture. More exploration is needed before our conclusions can be generalized to other races or cultures.

It is important to note that in choosing to utilize secondary data from a large population survey we made an inherent tradeoff between depth of measurement and the ability to measure large, interesting samples. Thus, we have no information about the spouses of our participants, who are important confounds both genetically and environmentally. Additionally, we were not able to examine measures of relationship quality, parenting, or information about the children of our measured parents. There is also no available information on how study responders differed from non-responders, so it is possible that the results are biased by self-selection.

Lastly, it is difficult to draw direct comparisons between our study populations in Sweden and the US, given differences not only in the way depressive symptoms were measured in the two datasets, but also in the very nature of the definition of marriage and single parenthood in the

two countries. It would be useful for future studies to attempt to understand whether and how notions of marriage and parenthood differ in the two cultures.

Conclusions

Our study is the first one to examine single motherhood and its psychological outcomes using a genetically informed design. This methodology is important for its capacity to eliminate entire groups of confounding variables, such as possible genetic influences or any kind of upbringing or parenting variables (e.g. religious influences, racial or cultural effects). Our findings provide thus the strongest evidence to date illustrating the impact of single motherhood on depression and other mental health indicators. Replication in other twin samples is, of course, needed to advance knowledge of the putative selection and causal mechanisms underlying single motherhood and mental health indicators. Additionally, our study is the first one to conduct a genetically-informed cross cultural comparison of single motherhood and mental health outcomes in two countries with vastly different social policies. Lastly, we add to a small body of knowledge describing the relation between single fatherhood and mental health.

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