Older Maternal Age Is Associated With Depression, Anxiety, and Stress Symptoms in Young Adult Female Offspring

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The evidence regarding older parental age and incidence of mood disorder symptoms in offspring is limited, and that which exists is mixed. We sought to clarify these relationships by using data from the Western Australian Pregnancy Cohort (Raine) Study. The Raine Study provided comprehensive data from 2,900 pregnancies, resulting in 2,868 live born children. A total of 1,220 participants completed the short form of the Depression Anxiety Stress Scale (DASS-21) at the 20-year cohort follow-up. We used negative binomial regression analyses with log link and with adjustment for known perinatal risk factors to examine the extent to which maternal and paternal age at childbirth predicted continuous DASS-21 index scores. In the final multivariate models, a maternal age of 30–34 years was associated with significant increases in stress DASS-21 scores in female offspring relative to female offspring of 25- to 29-year-old mothers. A maternal age of 35 years and over was associated with increased scores on all DASS-21 scales in female offspring. Our results indicate that older maternal age is associated with depression, anxiety, and stress symptoms in young adult females. Further research into the mechanisms underpinning this relationship is needed.

General Scientific Summary

This study suggests that older maternal age is associated with adverse symptoms of depression, anxiety, and distress in young adult females. Paternal age was not found to be associated with mental health outcomes for either males or females in this sample.

Keywords: Raine Study, DASS, cohort study, maternal age, paternal age

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Both younger and older parental age has been linked to mental health problems in offspring. There is a substantial literature relating young parenthood, particularly teenaged motherhood, to adverse mental health outcomes in young children (Do et al., 1998; Fergusson & Woodward, 1999; Harden et al., 2007; McGrath et al., 2014). Relative to offspring of older aged parents, offspring of teenaged mothers are at increased risk of mood disorders, internalizing problems (e.g., withdrawal, depression/anxiety, somatic symptoms), substance misuse, and juvenile crime (Fergusson & Woodward, 1999; Harden et al., 2007). In terms of psychiatric diagnoses, offspring of teenaged mothers have been found to have a 51% increased risk of having any psychiatric diagnosis, and
offspring of teenaged fathers a 28% increased risk (McGrath et al., 2014).

In the case of older parental age and offspring mental health problems, the research has focused overwhelmingly on psychiatric diagnoses. There is now strong evidence that the children of older fathers are at heightened risk of schizophrenia and autism spectrum disorders (Hultman, Sandin, Levine, Lichtenstein, & Reichenberg, 2011; Miller et al., 2011), while the evidence for increased risk for bipolar disorder diagnosis is mixed with some studies suggesting a relationship (Frans et al., 2008; Menezes et al., 2010) and others finding no association (Buizer-Voskamp et al., 2011; McGrath et al., 2014). It has also been suggested that the effect of paternal age may be sexually dimorphic (Byrne, Agerbo, Ewald, Eaton, & Mortensen, 2003; Miller et al., 2011). In the case of maternal age, advanced maternal age has been linked to increased risk for autism spectrum disorders (Sandin et al., 2012). Another study found that older maternal age increases risk for bipolar disorder diagnosis in offspring (Menezes et al., 2010), whereas other studies do not support this relationship (Frans et al., 2008; McGrath et al., 2014).

There is less information on the relationship between parental age and other mood disorders such as depression and anxiety. One large scale study using data from a Dutch population registry found adult offspring of both younger (<20) and older (≥40) fathers had significantly increased odds of a major depressive disorder diagnosis (Buizer-Voskamp et al., 2011). Similarly, relative to offspring of 25- to 29-year-old parents, the adult offspring of teenaged mothers and fathers, as well as older fathers, have been found to have increased incidence of mood disorders (McGrath et al., 2014). Conversely, Ferguson and Woodward (1999) found a significant linear association between increasing maternal age and decreasing rates of anxious and depressive disorders (as per Diagnostic and Statistical Manual of Mental Disorders, 4th ed. [DSM–IV] criteria) in 18-year-old offspring. Other studies using data from the Western Australian Pregnancy Cohort (Raine) Study have indicated that maternal age is a significant prenatal predictor of risk for child behavior outcomes from age 2 to 14 (Tearne et al., 2014) and that there is a significant linear association between maternal age and risk for problem internalizing and externalizing behaviors in children from ages 2–17, whereas older maternal age is associated with decreased risk for child behavior problems (Tearne et al., 2015).

To our knowledge, there are no studies that examine the incidence of symptoms of depression and anxiety (as opposed to diagnosis with a major depressive or anxious disorder) as a function of parental age in young adults. Furthermore, parental age has not been examined in relation to stress in offspring as far as we are aware. Investigation of these issues is important because it is recognized that mental health issues may not always be limited to those who meet diagnostic criteria, the broader spectrum of mental health outcomes in offspring. In line with previous literature, it was hypothesized that offspring of teenaged mothers and fathers would be at increased risk of elevated DASS-21 scores. The existing findings relating to mood symptoms in offspring and older parents are mixed, and as such we sought to clarify what relationships, if any, existed between older parental age and depression, anxiety and stress in offspring.

Method

Study Population

The Raine Study is a population-based prospective pregnancy cohort study. The methodology for the study has been described in detail elsewhere (Newnham, Evans, Michael, Stanley, & Landau, 1993). Briefly, 2900 pregnant women were recruited to the study between 16 and 20 weeks’ gestation through the public antenatal clinic at King Edward Memorial Hospital (KEMH) in Perth, Western Australia, or surrounding private practices between May 1989 and November 1991. The criteria for enrolment into the study were English language proficiency sufficient to understand the implications of participating in the study, an expectation that they would deliver at KEMH, and an intention to remain in Western Australia to facilitate follow-up of their child(ren). Ninety percent of eligible women agreed to take part. Participants provided data on psychosocial and sociodemographic characteristics at enrolment and again at 34 weeks’ gestation. A total of 2,868 live infants and their families have since undergone assessment at ages 1, 2, 3, 5, 8, 10, 14, 17, 20, and 23 years. Written parental and adolescent/young adult consent (14, 17, 20, and 23) was provided at each follow-up. It has been previously reported that the initial Raine sample overrepresented socially disadvantaged families, and that selective attrition of the sample over time led to a closer representation of those in the sample to the Western Australian population (Robinson et al., 2010). The protocols for the study were approved by the Human Research Ethics Committees at KEMH and the Princess Margaret Hospital for Children in Perth, Western Australia. Ethics approval for the 20-year follow-up was obtained from the University of Western Australia Human Research Ethics Committee.

Loss to Follow-up

Data collection for the 20-year follow-up took place between March 2010 and April 2012. There were 2,125 young adults eligible for follow up at 20 years. Of the 1,565 (74%) who participated, 78% (n = 1,220) completed the DASS-21. Characteristics of those who completed the DASS-21 at follow-up compared with those from the original cohort who did not are presented in Table 1.

Mental Health Data

Anxiety, depression, and stress were assessed by using the short form of the Depression Anxiety Stress Scales (DASS-21; S. H. Lovibond & P. F. Lovibond, 1995b). The DASS-21 is a short form of the 42-item DASS (S. H. Lovibond & P. F. Lovibond, 1995b),
with both scales found to have good reliability and validity in clinical and nonclinical samples (Antony, Bieling, Cox, Enns, & Swinson, 1998; Crawford & Henry, 2003; Henry & Crawford, 2005). The DASS comprises three 7-item scales measuring depression, anxiety, and stress. The depression scale assesses dysphoria, hopelessness, devaluation of life, self-depreciation, lack of interest/involvement, anhedonia, and inertia; the anxiety scale measures autonomic arousal, skeletal musculature effects, situational anxiety, and subjective experience of anxious affect; and the stress scale assesses difficulty relaxing, nervous arousal, being easily upset/agitated, irritable/overreactive, and impatient (S. H. Lovibond & P. F. Lovibond, 1995b). Participants were asked to rate the severity of each symptom during the past week on a 4-point scale ranging from 0 (“did not apply to me at all”) to 3 (“applied to me very much, or most of the time”). Scores were doubled as per the scoring instructions.

The depression and anxiety scales of the 42-item DASS show good convergent validity with the Beck Anxiety and Depression Inventories (Lovibond & Lovibond, 1995a). Several studies have suggested temporal stability of the DASS across time (Brown, Chorpita, Korotitsch, & Barlow, 1997; Cunningham, Brown, Brooks, & Page, 2013; Page, Hooke, & Morrison, 2007; Willemsen, Markey, Declercq, & Vanheule, 2011). A large-scale study has shown stability of symptoms as measured by the DASS over 3 to 8 years (Lovibond, 1998). Analyses specific to the DASS-21 have shown a quadripartite structure, which consisted of a general factor that the authors suggested reflected general psychological distress and orthogonal factors suggested to represent depression,
anxiety, and stress (Henry & Crawford, 2005). While there is evidence for a common factor representing shared variance underlying the DASS scales, there is also strong evidence for specific factors underlying the depression, anxiety, and stress subscales. Furthermore, there is extensive evidence in the literature that anxiety and depression are not independent constructs (Clark & Watson, 1991), and thus evidence for shared variance underlying the DASS subscales provides support for the construct validity of the DASS. As a result, the three subscales were included as the outcome measures in the present study.

**Predictor Variables**

Parental age and date of birth were recorded at initial recruitment. Both maternal and paternal age in years at birth of the study child were calculated and modeled as continuous and categorical variables. In the case of parental age as a categorical variable, age was stratified into 5-year age groups (<20, 20–24, 25–29, 30–34, 35–39, ≥40 years of age), but for mothers the older two age categories were collapsed to form one (≥35) because of small numbers of women aged 40 and over in the sample. This categorization is often used in classification of population fertility data and broader epidemiological investigations (Australian Bureau of Statistics, 2010; Buizer-Voskamp et al., 2011). A maternal and paternal age of 25–29 years was set as the reference group in all analyses because the peak fertility rate for Australian women was in this age group at the time of recruitment to the Raine Study (Australian Bureau of Statistics, 2010).

**Control Variables**

We adjusted for several prenatal variables previously established as key predictors of mental health outcomes in the Raine cohort (Tearne et al., 2014). These variables included maternal education (12 or more years of education compared with 11 or fewer), maternal smoking during the first 18 weeks of pregnancy (no smoking compared with any smoking), maternal experience of stressful life events in the first 18 weeks of pregnancy (two or fewer compared with three or more), total family income as at 18 weeks (AUD compared with ≥24,000 AUD, in accordance with the poverty line at the time of collection), and maternal diagnosis of gestational hypertension (no hypertension compared with any hypertension).

**Statistical Analyses**

We compared characteristics of participants who completed the DASS-21 at the 20-year follow-up with nonparticipants from the original cohort based on gender, race, maternal education, total family income at 18 weeks’ gestation, maternal smoking in the first 18 weeks of pregnancy, gestational hypertension, and maternal and paternal age at birth of study child. Their mothers were more likely to have finished high school and were less likely to have smoked and experienced stressful life events during pregnancy with the study child. Boys from the original cohort were less likely than girls to participate at age 20. There were no differences based on race or gestational hypertension between participants and nonparticipants from the original cohort. Given the significant differences between the initial Raine population and those that completed the DASS at the 20-year follow-up, inverse probability weighting was used to standardize the sample and adjust for bias that may result from nonrandom attrition. Weights were created using the previously mentioned pregnancy variables, from which a probability of participation and the inverse of this probability were created. Applying these weights created a sample with approximately similar distribution to that of the original Raine cohort.

The internal consistency of the DASS-21 scales was measured by Cronbach’s alpha (Total scale = .93; Depression scale = .89; Anxiety scale = .76; Stress scale = .86). Median and mean DASS-21 total and subscale scores are presented in Table 2. Median and mean scores in this sample were slightly higher than those reported in another nonclinical sample (Henry & Crawford, 2005). There were significant differences between male and female scores on all subscales of the DASS-21, with females scoring higher.

### Table 2

**Median, Mean (SD) Depression Anxiety Stress Scale (DASS)**

<table>
<thead>
<tr>
<th>Scale</th>
<th>Females Median, M (SD)</th>
<th>Males Median, M (SD)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression</td>
<td>6.00, 7.97 (8.43)</td>
<td>4.00, 6.22 (7.43)</td>
<td>.004**</td>
</tr>
<tr>
<td>Anxiety</td>
<td>4.00, 5.85 (6.48)</td>
<td>2.00, 4.32 (4.74)</td>
<td>&lt;.001***</td>
</tr>
<tr>
<td>Stress</td>
<td>8.00, 10.52 (8.64)</td>
<td>6.00, 7.46 (7.21)</td>
<td>&lt;.001***</td>
</tr>
</tbody>
</table>

* **Significant at p < .01. *** Significant at p < .001.
Maternal and paternal age were moderately correlated with each other ($r = .47$, $p > .01$).

We initially tested whether there was an interaction between maternal age and offspring gender, and paternal age and offspring gender, and offspring outcomes. There was a significant interaction between maternal age and offspring gender, and paternal age and offspring gender for total DASS scores and all symptom scale scores. As such, all analyses were stratified based on gender (see supplementary information available online).

In the final multivariate models, where we adjusted for age of other parent and known confounders, a maternal age of 30–34 years was associated with significantly increased stress (RR = 1.27, $p = .031$) subscale scores in female offspring relative to the reference group (Table 3). A maternal age of 35 years and over was associated with increases in all subscale scores in female offspring (depression: RR = 1.51, $p = .026$; anxiety: RR = 1.51, $p = .029$; stress: RR = 1.36, $p = .033$). There was some evidence of an association between a paternal age of 30–34 years and decreased risk for internalizing disorders across childhood (Fergusson & Woodward, 1999), and was associated with decreased risk for depressive and anxious disorders in 18-year-old offspring (Buizer-Voskamp et al., 2011; McGrath et al., 2014) and was associated with decreased risk for depressive and anxious disorders in 18-year-old offspring (Buizer-Voskamp et al., 2011; McGrath et al., 2014; Miller et al., 2011). A key difference is that our study examined self-reported symptoms of depression, anxiety, and stress rather than clinical diagnoses. It is plausible that the risk factors for psychological adjustment and distress differ from those risk factors identified for more severe psychiatric outcomes. The results of our study suggest that when moving beyond diagnosis to consider a broader spectrum of psychological distress and adjustment in offspring, paternal age is not an important factor of influence, at least in this sample when using the DASS-21 as an outcome variable. This is an important finding when placed in the context of the existing literature, because it suggests that father’s age may have a differential impact on different types of psychiatric distress/illness and may not be relevant for all outcomes. It is plausible that at the level of distress, rather than disorder, associations with parental age may stem from environmental factors, such as interactions with the parent, rather than biology. It may be the case that the significance of maternal and not paternal age as predictors of offspring outcomes may reflect an imbalance in key relationships in the caregiving of the child, such that maternal age exerts a greater influence because mothers may have played a greater caregiving role. In the few existing studies examining maternal age and mood disorders in offspring, older maternal age has been found to have no significant association with offspring outcome in two studies (Buizer-Voskamp et al., 2011; McGrath et al., 2014) and was associated with decreased risk for depressive and anxious disorders in 18-year-old offspring (Fergusson & Woodward, 1999), and decreased risk for internalizing disorders across childhood (Tearne et al., 2015). Our study suggests that maternal age is implicated in the subsequent experience of symptoms of depression, anxi-

### Table 3

**Adjusted Analyses Estimating the Effect of Maternal and Paternal Age on Total Depression Anxiety Stress Scale (DASS) Scores and Depression, Anxiety, and Stress Subscale Scores in Girls**

<table>
<thead>
<tr>
<th>Age</th>
<th>Depression scale</th>
<th>Anxiety scale</th>
<th>Stress scale</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal age</td>
<td>RR (95% CI)</td>
<td>RR (95% CI)</td>
<td>RR (95% CI)</td>
</tr>
<tr>
<td>&lt;20 (n = 42)</td>
<td>RR = 1.41 (.94, 2.11)</td>
<td>RR = 1.17 (.78, 1.74)</td>
<td>RR = 1.14 (.84, 1.55)</td>
</tr>
<tr>
<td>20–24 (n = 100)</td>
<td>RR = 1.08 (.79, 1.48)</td>
<td>RR = 1.17 (.85, 1.60)</td>
<td>RR = 1.01 (.80, 1.29)</td>
</tr>
<tr>
<td>30–34 (n = 200)</td>
<td>RR = 1.27 (96, 1.67)</td>
<td>RR = 1.19 [91, 1.57]</td>
<td>RR = 1.27 [1.02, 1.57]</td>
</tr>
<tr>
<td>≥35 (n = 111)</td>
<td>RR = 1.51 [1.05, 2.16]</td>
<td>RR = 1.51 [1.04, 2.18]</td>
<td>RR = 1.36 [1.03, 1.81]</td>
</tr>
<tr>
<td>Paternal age</td>
<td>RR (95% CI)</td>
<td>RR (95% CI)</td>
<td>RR (95% CI)</td>
</tr>
<tr>
<td>&lt;20 (n = 15)</td>
<td>RR = .96 (.55, 1.69)</td>
<td>RR = .72 [.41, 1.29]</td>
<td>RR = 1.17 (.75, 1.82)</td>
</tr>
<tr>
<td>20–24 (n = 81)</td>
<td>RR = 1.02 (.73, 1.42)</td>
<td>RR = 1.06 [.76, 1.49]</td>
<td>RR = 1.08 (.83, 1.39)</td>
</tr>
<tr>
<td>30–34 (n = 216)</td>
<td>RR = .82 (.63, 1.07)</td>
<td>RR = .93 [.71, 1.23]</td>
<td>RR = .80 [.65, .995]</td>
</tr>
<tr>
<td>35–39 (n = 108)</td>
<td>RR = .87 (.62, 1.22)</td>
<td>RR = .99 [.70, 1.40]</td>
<td>RR = .86 (.65, 1.12)</td>
</tr>
<tr>
<td>≥40 (n = 66)</td>
<td>RR = 1.01 (.67, 1.53)</td>
<td>RR = .92 [.60, 1.41]</td>
<td>RR = .95 (.68, 1.31)</td>
</tr>
</tbody>
</table>

**Note.** RR = rate ratio; $p < .05$; CI = confidence interval; n = number of offspring in each age bin; ref. = reference. Adjusted for age of other parent, maternal smoking in pregnancy, maternal education, total family income, maternal experience of stressful life events, and maternal gestational hypertension.
iety, and stress in female young adult offspring. This is somewhat different from the results presented in the aforementioned studies, although our findings are consistent with a study finding older maternal age may be associated with increased risk for bipolar affective disorder (Menezes et al., 2010). This finding is also broadly consistent with a number of studies suggesting advanced maternal age is associated with increased risk for autism spectrum disorders in offspring (Croen, Najjar, Fireman, & Grether, 2007; Durkin et al., 2008; Grether, Anderson, Croen, Smith, & Windham, 2009; King, Fountain, Dakhlallah, & Bearman, 2009; Parner et al., 2012; Sandin et al., 2012).

Future research should attend to uncovering potential mechanisms underlying the relationship between maternal age and depression, anxiety and stress symptoms in female offspring. It is possible that it is not so much age at pregnancy that underpins the relationship between maternal age and symptoms in female offspring, but age of the mother at follow-up assessment (which is an indirect effect of age at pregnancy). One possible hypothesis is difficulties in the mother–daughter relationship because of a large age difference between the two parties. The “older mothers” in our sample were 50–54 and 55 and over when their offspring were 20 years of age. It may be that a 30 or more year age difference between mother and daughter leads to a significant difference in the value systems held by each, as well as generational differences that may cause tension in the relationship, particularly during the transition period of young adulthood, leading to stress, worry, and sadness in the child. The increased incidence of depression, anxiety, and stress symptoms may reflect a stressful period in the lives of both mother and daughter. Another example of possible age-related differences in mother–daughter relationships is the impact of age-related health changes and problems in mothers. The median age at which women in Australia go through menopause is around 51 years of age (Do et al., 1998). Statistics from the Centers for Disease Control and Prevention suggest that once women enter their fifties, the leading causes of mortality are various cancers, heart disease, and chronic respiratory conditions (Centers for Disease Control and Prevention, 2010). It has been found that levels of emotional distress and behavioral problems escalate in adolescents and young adults with an immediate family member with a cancer diagnosis (Sahler et al., 1994), and another study suggested that adolescent female offspring are most negatively affected by a parent’s diagnosis with serious illness (Osborn, 2007). Thus, the higher risk of depression, anxiety, and stress in offspring of women in their fifties may be because of health-related stress and concern within the family. It may be that significant life changes are occurring in parallel in mothers and daughters, which may influence emotion dysregulation in offspring.

Another possible explanation for our results is that the relationship between advancing maternal age and offspring mental health outcomes observed in this study may be because of unmeasured confounding. Examining the relationship between maternal age and offspring mental health outcomes is complex, owing to the great number of variables associated with older motherhood that may also exert an influence on offspring outcomes. The statistical position taken in this study was that variables measured at the same time as the key outcome variables (i.e., prenatal variables) were considered as potential confounders, and our large sample size allowed for an exhaustive

Figure 1. Depression subscale. See the online article for the color version of this figure.
list of control variables to eliminate, as far as possible, confounding. However, there are myriad other factors that may influence the mental health of offspring. Recent studies in the area using quasi-experimental designs to control for environmental and genetic influences that vary within families using sibling-comparison analyses have yielded interesting findings. One study indicated that environmental factors associated with maternal age at childbirth which also vary within families are implicated in the incidence of delinquent behaviors in offspring (D’Onofrio et al., 2009), while another indicated that controlling for variables shared within families strengthened the association between advanced paternal age and various indices of psychopathology, consistent with a causal hypothesis (D’Onofrio et al., 2014). Although beyond this scope of this study, future research designs controlling for factors shared within families may leave researchers better placed to identify the specific factors, be they genetic, environmental, or both, that influence offspring behavior. This would allow us to better specify how maternal age may influence depression, anxiety and stress symptoms in young adult offspring, and why this relationship may be specific to female offspring.

There are a number of strengths associated with this study. Our prospectively collected data are drawn from a large cohort study, allowing us the opportunity for a comprehensive assessment of the impact of parental age on anxiety, stress, and depression symptoms in offspring in a nonclinical population. However, our findings must be interpreted in the context of a number of limitations. First, a limitation is our use of self-report data. Self-report measures have been validated as a valid means of assessing depression, anxiety, and stress (Antony et al., 1998). While we did not set out to measure clinical levels of distress, but rather more general symptoms of distress in our sample, we cannot rule out the possibility of over- or underreporting. A second limitation is the relatively small numbers of parents in the oldest (aged 40 and over) and youngest (19 and under) age groups at childbirth in our sample (2.3% and 9.7%, respectively). This may have impacted upon the strength of the influence of parental age upon offspring in these categories. Furthermore, the DASS-21 data measure symptoms over the past week. While a study using the longer version of the DASS scale has shown stability of each of the syndromes over substantial periods of time (3 to 8 years), future research could look to investigate the stability of symptoms over time in the Raine and similar cohorts. Another consideration was that it was not possible to differentiate between parental age at birth of first child versus birth of the study child. It has been suggested it may be parental age at birth of first child, not birth of the individual child, which predicts mental health outcomes in offspring (Petersen, 2011). An investigation of this type was beyond the scope of this study but is a worthy focus of future research. Finally, we controlled for a comprehensive range of other prenatal variables known to impact upon mental health in offspring, but this list is not exhaustive and does not take into account the myriad other influences on mental health across the life span. For example, family structure was not accounted for in this study. Many variables of interest, such as maternal mental health at follow-up, were not available to us. These variables may impact upon the relationships observed in the data, and further research is necessary to evaluate

Figure 2. Anxiety subscale. See the online article for the color version of this figure.
their impact. Despite these limitations, our data provide new insights into the impact of parental age on general symptoms of anxiety, depression, and stress in young adult offspring.

**Conclusions**

We found that a maternal age of 30–34 years was associated with significant increases in total DASS-21 scores in female offspring, and a maternal age of 35 years and over was associated with significant increases in total and subscale DASS-21 scores. Paternal age was not found to be associated with offspring depression, anxiety, and stress. It appears that when examining a broad spectrum of psychological adjustment, the relationships between parental age and offspring symptomatology differ from those in the literature on parental age and severe psychiatric outcomes. We

![Stress subscale](image)

*Figure 3. Stress subscale. See the online article for the color version of this figure.*

Table 4

<table>
<thead>
<tr>
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<th>Depression scale</th>
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<th>Stress scale</th>
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<tbody>
<tr>
<td>Maternal age</td>
<td>RR (99% CI)</td>
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<td>RR (99% CI)</td>
</tr>
<tr>
<td>&lt;20 (n = 22)</td>
<td>.71 (.44, 1.15)</td>
<td>.68 (.44, 1.05)</td>
<td>.63 (.41, .98)</td>
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<tr>
<td>20–24 (n = 109)</td>
<td>.77 (.55, 1.07)</td>
<td>.87 (.65, 1.16)</td>
<td>.80 (.61, 1.06)</td>
</tr>
<tr>
<td>30–34 (n = 158)</td>
<td>1.01 (.74, 1.37)</td>
<td>1.07 (.82, 1.41)</td>
<td>1.03 (.79, 1.33)</td>
</tr>
<tr>
<td>≥35 (n = 98)</td>
<td>.97 (.65, 1.46)</td>
<td>.92 (.65, 1.32)</td>
<td>.90 (.63, 1.28)</td>
</tr>
</tbody>
</table>

Paternal age

<table>
<thead>
<tr>
<th>Age</th>
<th>Depression scale</th>
<th>Anxiety scale</th>
<th>Stress scale</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;20 (n = 9)</td>
<td>.53 (.25, 1.17)</td>
<td>.97 (.51, 1.85)</td>
<td>.77 (.41, 1.45)</td>
</tr>
<tr>
<td>20–24 (n = 60)</td>
<td>.98 (.67, 1.43)</td>
<td>.94 (.68, 1.29)</td>
<td>.98 (.72, 1.34)</td>
</tr>
<tr>
<td>30–34 (n = 178)</td>
<td>1.11 (.83, 1.49)</td>
<td>.97 (.75, 1.25)</td>
<td>1.01 (.79, 1.29)</td>
</tr>
<tr>
<td>35–39 (n = 94)</td>
<td>.98 (.65, 1.46)</td>
<td>.86 (.60, 1.23)</td>
<td>.95 (.67, 1.34)</td>
</tr>
<tr>
<td>≥40 (n = 62)</td>
<td>1.06 (.69, 1.64)</td>
<td>1.15 (.80, 1.67)</td>
<td>1.23 (.85, 1.77)</td>
</tr>
</tbody>
</table>

*Note:* RR = Rate ratio = p < .05; CI = confidence interval; n = number of offspring in each age bin; ref. = reference. Adjusted for age of other parent, maternal smoking in pregnancy, maternal education, total family income, maternal experience of stressful life events, and maternal gestational hypertension.
suggest that maternal age when the young adult is assessed may be as important as considering age at pregnancy.

References


