The association between paternal psychopathology and adolescent depression and anxiety: A systematic review

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ABSTRACT

Introduction: Paternal psychopathology is associated with various adolescent outcomes. With emotional disorders presenting a significant public health concern in the adolescent age group, the aim of this systematic review was to synthesize evidence on the relationship between paternal mental health and adolescent anxiety or depression.

Methods: PubMed, Web of Science, Embase, Ovid MEDLINE, Global Health, and PsycINFO were searched for articles which primarily aimed to investigate the relationship between paternal mental health (exposure) and adolescent anxiety or depression (outcome). Articles were assessed for risk of bias, and findings are presented in a narrative synthesis. The protocol is registered on PROSPERO (CRD42018094076).

Results: Findings from the fourteen included studies indicated that paternal depression is associated with adolescent depression and anxiety. Findings relating to other paternal mental health disorders were inconclusive. Results largely suggested that adolescent depression and anxiety is equally associated with paternal and maternal mental health. The included studies were mostly cross-sectional, and the quality of included studies was mixed. Attempts to focus on the 11–17 year age range were hampered by the variability of age ranges included in studies.

Conclusions: Further longitudinal research is needed to clarify the association between paternal mental health disorders other than depression, and adolescent anxiety or depression. Mechanisms in this relationship should also be further explored, and could be informed by existing models on younger children.

1. Introduction

A recent review estimated that 15%–23% of children worldwide live with a parent with a mental health or substance use disorder (Leijdesdorff, van Doesum, Popma, Klaassen, & van Amelsvoort, 2017). Research on the impact that parental psychopathology has on children and adolescents tends to focus on mothers rather than fathers (Cabrera, Volling, & Barr, 2018). However, the role of fathers in their children’s lives cannot be reasonably overlooked, as they increasingly engage in childcare and head up single parent households (Livingston, 2013; Parker & Wang, 2013). Accordingly, the inclusion of fathers in research is growing, such that the impact of paternal depression on child and adolescent outcomes has been extensively studied and reviewed (Gentile & Fusco, 2017;
Kane & Garber, 2004; Sweeney & MacBeth, 2016), and potential mechanisms in the relationship between paternal and child psychopathology have been considered (Ramchandani & Psychogiou, 2009; Sweeney & MacBeth, 2016).

But while research is growing in this field, less consideration has been given to the impact of paternal mental health disorders other than depression. Given that disorders such as anxiety and substance abuse are known to be just as prevalent as depression in the general population, their potential impact warrants equal attention (McManus, Bebbington, Jenkins, & Brugha, 2016). A review which draws together evidence on paternal psychopathologies including but not limited to depression would provide insight to the comparative state of the literature regarding a range of disorders.

Moreover, previous reviews of the literature have tended to examine findings from adolescence alongside childhood and even infancy (Gentile & Fusco, 2017; Kane & Garber, 2004; Ramchandani & Psychogiou, 2009; Sweeney & MacBeth, 2016), despite indication that the effect of parental psychopathology changes throughout childhood and adolescence (Connell & Goodman, 2002). Previous reviews have also investigated both internalising and externalising outcomes in offspring, despite the predictors, course and outcomes of these disorders being heterogeneous and warranting independent and comprehensive study (Liu, 2004; Liu, Chen, & Lewis, 2011). Adolescents are particularly affected by anxiety and depressive disorders (Sadler et al., 2018), which during this developmental period are associated with suicide and a wide range of psychosocial and physical health issues, some of which extend into adulthood (Essau, Lewinsohn, Olaya, & Seeley, 2014; Keenan-Miller, Hammen, & Brennan, 2007; McLeod, Horwood, & Fergusson, 2016; Windfuhr et al., 2008; Woodward & Fergusson, 2001). Therefore these disorders were selected as the focus for this review.

Consequently, our primary aim was to synthesise the evidence on the relationship between paternal mental health (exposure) and adolescent anxiety and depression (outcome). Focusing on this association will provide insight on a comparatively neglected relationship between fathers and adolescents, and on the circumstances surrounding anxiety and depression, which represent a major public health concern in this age group. Our secondary aims were to summarise findings on any potential mediators and moderators of this relationship investigated by included studies, and the relative importance of maternal mental health where investigated by included studies.

2. Methods

2.1. Inclusion and exclusion criteria

The protocol for this systematic review was registered on PROSPERO and can be accessed at www.crd.york.ac.uk/PROSPERO/display_record.asp?ID=CRD42018094076. We followed PRISMA reporting guidelines, and a checklist can be found in Supplementary File 1.

In light of the changing role of fathers between the 20th and 21st centuries, we sought to capture studies published since the year 2000 to ensure that the studies were as reflective of contemporary father-adolescent relationships as possible (Livingston, 2013; Parker & Wang, 2013). In addition, to ensure that we did not confine results for child, adolescent and adult offspring, we focused on the 11–17 year age range. With ongoing debate around the ‘true’ age of adolescence (Sawyer, Azzopardi, Wickremarathne, & Patton, 2018), this period was felt to capture adolescent years with the most certainty: 11 is the lowest age captured by globally relevant adolescent health surveys (Sawyer et al., 2018), and 18 is adopted by the United Nations to mark adulthood (UN General Assembly, 1989).

Therefore, we included studies which (i) as part of the primary aim, investigated the direct relationship between paternal mental health as the exposure, and adolescent anxiety or depression as the outcome; (ii) included adolescents in the 11–17 year age range at the time of outcome measurement; (iii) were published in English; (iv) were published since the year 2000. We excluded studies which did not meet inclusion criteria, or (i) were systematic reviews, grey literature, or conference abstracts; (ii) only investigated paternal mental health as a confounder, mediator or moderator; (iii) did not use primary data, or if using secondary data, did not use a methodology which had been previously published; (iv) did not use a validated measurement instrument or diagnostic procedure to measure the relevant exposure and outcome; (v) included adolescents outside the 11–17 year age range but did not either stratify analyses to focus exclusively on the 11–17 year age range, or report a mean age inside the 11–17 year age range.

2.2. Outcome

The outcome of interest was anxiety or depression in adolescents. This included internalising problems and emotional symptoms.

2.3. Exposure

The exposure of interest was any paternal mental health problem, including drug and alcohol misuse.

2.4. Search strategy

The search was conducted between May and June 2019. PubMed, Web of Science Core Collection, and Ovid (Embase, Ovid MEDLINE, Global Health, and PsycINFO) were searched using key words on 21 May 2019. Key words were constructed around the following five concepts which needed to be captured by the included studies:
The full search strategy for Ovid MEDLINE can be found in Table 1.

Duplicates were removed in Endnote. Titles, abstracts and full texts were screened according to the study inclusion and exclusion criteria. Reference lists of included studies and related literature reviews were hand searched. Relevant titles found during the hand search were then screened according to eligibility criteria. The search and screening was conducted by AW, and independently cross-checked by DL. If the authors were uncertain of an article's eligibility, the article was discussed and screened together, and a consensus was reached. A PRISMA flow diagram is presented in Fig. 1.

2.5. Data extraction and analysis

A data extraction form was developed, piloted and completed by AW. Data was extracted on study design, participant characteristics, exposure and outcome measurement, covariates and relevant findings. Data extraction was independently cross-checked by DL. Disagreements over data extraction were discussed and a consensus reached. Authors were contacted to supply missing information.

Results are presented in a narrative synthesis. We did not find multiple studies which looked at the same exposures and outcomes, and which used similar study designs or statistical analyses to do so. Therefore in accordance with guidance from the Cochrane Collaboration which advises against combining the results of studies which make heterogenous comparisons, we did not synthesize the results in a meta-analysis (Cochrane Collaboration, 2011).

Risk of bias was assessed at the study level using the Newcastle-Ottawa Scale (NOS) for cohort studies (Wells et al., 2012) and a version of the scale adapted for cross-sectional studies (Herzog et al., 2013). Two items on sample size and statistical testing only appear in the cross-sectional version of the scale, but as an important aspect of study quality, we also applied these items to the cohort studies, as has been done previously (Epstein et al., 2018). AW and DL independently conducted risk of bias assessments. Disagreements were discussed and a consensus reached. Study quality and risk of bias were taken into consideration throughout the narrative synthesis to help ascertain the strength of the studies’ findings.

3. Results

3.1. Study characteristics

In total, 14 studies were identified for inclusion in this review, collectively providing data on 31 271 fathers and 41 205 adolescents. The characteristics and findings of included studies are outlined in Table 2. The majority of included studies utilised a cross-sectional design. Some made use of longitudinal cohorts, such as the National Longitudinal Survey of Children and Youth (Elgar, Mills, McGrath, Waschbusch, & Brownridge, 2007). The follow-up periods of interest for the longitudinal study designs were one to three years (Tyrell, Yates, Reynolds, Fabricius, & Braver, 2018) two years (Elgar et al., 2007), four and seven years (Lewis, Neary, Polek, Flouri, & Lewis, 2017). Reeb and Conger (2009) did not specify the length of follow-up period.

Lewis et al. (2017) analysed data from two cohorts, Growing Up in Ireland and the Millennium Cohort Study, giving them the largest total adolescent sample of the included studies (n = 6070 and n = 7768). The other studies had adolescent samples ranging from n = 81 (Kane & Garber, 2009) to n = 7809 (Amrock & Weitzman, 2014). Participating offspring ranged from ages 4–19 years (although we focus on results reported from analyses encompassing the adolescent years). A variety of instruments were used to
measure adolescent anxiety and depression, usually identifying disorders through the endorsement of emotions or behaviours by the adolescents or their parents. Only two studies conducted diagnostic interviews to identify adolescent anxiety and depression (Agerup, Lydersen, Wallander, & Sund, 2015; Ohannessian et al., 2005).

Studies most frequently investigated paternal affective and anxiety disorders in relation to adolescent anxiety and depression, with nine of the included studies reporting on associations with paternal depression, anxiety, or internalising problems. Four studies investigated the effect of paternal alcohol misuse on adolescents. Other investigated paternal mental health disorders were externalising problems, Post-Traumatic Stress Disorder (PTSD), nonspecific psychopathology, antisocial personality disorder, Attention Deficit Hyperactivity Disorder, and avoidant personality problems. The following sections summarise the findings for each of these exposures. Where possible, we focus on the results from final models and analyses reported by the included studies, typically after adjusting for covariates.

3.2. Overview of study quality

Risk of bias assessments for all included studies can be found in Table 3 (cohort studies) and Table 4 (cross-sectional studies). It was a requirement that all included studies state the association between paternal and adolescent mental health as part of their primary aim, therefore the objective of all studies was clear. However, some aspects of the included studies were poorly reported. In some cases, the study design was not explicitly defined, and the data extractors had to decide the most likely design (Agerup et al., 2015; Choi et al., 2013; Middeldorp et al., 2016; Rognmo, Torvik, Ask, Røysamb, & Tambs, 2012; Selimbasic, Sinanovic, Avdibegovic, Brkic, & Hamidovic, 2017). The final sample size of included fathers was not always stated (Boricewicz Marsanic, Aukst Margetic, Jukic, Matko, & Grgic, 2014; Ohannessian et al., 2005; Reeb & Conger, 2009; Selimbasic et al., 2017), such that the number of cases used in analyses was sometimes unclear. Additionally, the sample sizes were varied. Kane and Garber (2009) and Selimbasic et al. (2017) adopted the lowest adolescent sample sizes ($n = 81$ and $n = 120$ respectively), but did not report power calculations or otherwise justify their numbers.

Overall the studies were reasonably consistent in reporting important descriptive statistics such as adolescent gender and age. However, it was not always clear whether the participating parents were biological parents, stepparents, adoptive or foster parents.
Table 2
Characteristics and findings of included studies.

<table>
<thead>
<tr>
<th>Lead Author, Year, Location of study</th>
<th>Study design</th>
<th>Father sample size</th>
<th>Adolescent sample size, gender and age (range and/or mean and SD)</th>
<th>Relevant paternal exposure(s), measurement instrument(s) used</th>
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<tr>
<td>Agerup 2015 Norway</td>
<td>Cross-sectional</td>
<td>n = 186</td>
<td>Total n = 345; Females n = 250; Males n = 95; 13.8–16.6 years; M = 15.0 (0.6)</td>
<td>Internalising problems and externalising problems Adult Self-Report</td>
<td>Major depressive disorder/dysthymia versus depression not otherwise specified versus no depression Mood and Feelings Questionnaire; Kiddie Schedule for Affective Disorders and Schizophrenia-Present and Lifetime version; Children's Global Assessment Scale</td>
<td>Adolescent age and sex; parental perceived economic satisfaction, marital status of biological parents, long-term physical illness or disability and internalising/externalising problems</td>
<td>After fully adjusting for all other variables, there were no significant associations between adolescent depression and paternal internalising (OR = 1.02, p = 0.66) or externalising problems (OR = 1.01, p = 0.83).</td>
</tr>
<tr>
<td>Amrock 2014 United States of America</td>
<td>Cross-sectional</td>
<td>n = 7809</td>
<td>Total n = 7809; Females n = 3654; Males n = 4155; 4–17 years</td>
<td>Mental health status/nonspecific psychological distress Extended-form Strengths and Difficulties Questionnaire</td>
<td>Children's ethnicity, region, birth weight, and health insurance status; having delayed the child's medical care for any reason within the past year; family poverty status; number of children in the family; presence of chronic disease in family; parent's age, employment status, education status, body mass index, drinking, and smoking behaviours; child's relationship to respondent parent</td>
<td></td>
<td>After fully adjusting for all other variables, children aged 4 to 11 were more likely to have abnormal emotional symptoms if they had a psychologically distressed father (OR = 6.3, 95% CI: 2.2 to 17.7, p &lt; 0.001). No such relationship was found for adolescents aged 12 to 17 (p &gt; 0.05).</td>
</tr>
<tr>
<td>Boričević Maršanić 2014 Croatia</td>
<td>Cross-sectional</td>
<td>Not applicable – no fathers were directly recruited for this study 1</td>
<td>Total n = 244; Females 53.2% 1; 12–18 years; M = 15.0 (3.49)</td>
<td>Chronic PTSD Diagnostic assessment including a structured diagnostic procedure and analysis of military service data</td>
<td>Internalising syndrome (broken down into withdrawn/depressed, somatic complaints, anxious/depressed) Youth Self-Report</td>
<td>Participants matched for age, sex, educational level, family income, parental employment status, ethnicity and residential area</td>
<td>Compared to those whose fathers did not have PTSD, adolescents whose fathers had PTSD showed higher rates of problems on the internalising scale as a whole (OR 1.92, 95% CI: 1.11 to 3.33, p = 0.02). Broken down, they showed higher rates of somatic complaints (OR 2.74, 95% CI: 1.48 to 5.08, p = 0.001), anxious/depressed problems (OR 1.71, 95% CI: 1.00 to 2.89, p = 0.046), but not withdrawn/depressed problems (p &gt; 0.05).</td>
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<td>Choi 2013 Korea</td>
<td>Cross-sectional</td>
<td>Not applicable – no fathers were directly recruited for this study</td>
<td>Total n = 950; Males n = 390; Females n = 560; Males age M = 16.5 (1.0); Females age M = 16.5 (1.0)</td>
<td>Drinking problems Father-Short Michigan Alcoholism Screening Test</td>
<td>Depression Beck's Depression Inventory Anxiety Beck's Anxiety Inventory</td>
<td>Age, religion, participation in chb activities, smoking, living with parents</td>
<td>After adjusting for covariates, paternal drinking problems were associated with elevated odds of anxiety (OR 2.21, 95% CI: 1.05 to 4.63) but not depression (p &gt; 0.05) in male students. Conversely in female students, paternal drinking problems were associated with elevated odds of depression (OR 1.84, 99% CI: 1.24 to 2.74), but not anxiety (p &gt; 0.05). Paternal depressive symptoms were correlated with subsequent internalising problems in boys (r = 0.42, p &lt; 0.01) and in girls (r = 0.33, p &lt; 0.01). Paternal depressive symptoms were correlated with concurrent internalising problems in girls (r = 0.32, p &lt; 0.01) but not in boys (r = 0.13, p &gt; 0.05). Paternal depressive symptoms were highly correlated with internalising symptoms on the Child Behaviour Checklist (r = 0.33, p &lt; 0.01), but not on the Youth Self Report (r = 0.12, p &gt; 0.05). Regression results were not fully reported. After all adjustments, paternal depression and adolescent depressive symptoms were positively associated in both the Growing up in Ireland cohort (0.24, 95% CI: 0.03 to 0.45, p = 0.023) and the Millennium Cohort Study (0.18, 95% CI: 0.01 to 0.36, p = 0.041). Results were similar in multiply imputed and complete case samples.</td>
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<tr>
<td>Elgar 2007 Canada</td>
<td>Longitudinal cohort</td>
<td>n = 330</td>
<td>Total n = 6048 a; Males n = 3125; Females n = 2923; At Time 2 10-15 years; Males age M = 12.59 (1.69); Females age M = 12.58 (1.71)</td>
<td>Depressive symptoms 12-item version of Centre for Epidemiological Studies - Depression Scale</td>
<td>Internalising problems Items developed for the Montreal Longitudinal Survey and Ontario Child Health Study</td>
<td>None</td>
<td>Paternal depressive symptoms were correlated with subsequent internalising problems in boys (r = 0.42, p &lt; 0.01) and in girls (r = 0.33, p &lt; 0.01). Paternal depressive symptoms were correlated with concurrent internalising problems in girls (r = 0.32, p &lt; 0.01) but not in boys (r = 0.13, p &gt; 0.05). Paternal depressive symptoms were highly correlated with internalising symptoms on the Child Behaviour Checklist (r = 0.33, p &lt; 0.01), but not on the Youth Self Report (r = 0.12, p &gt; 0.05). Regression results were not fully reported. After all adjustments, paternal depression and adolescent depressive symptoms were positively associated in both the Growing up in Ireland cohort (0.24, 95% CI: 0.03 to 0.45, p = 0.023) and the Millennium Cohort Study (0.18, 95% CI: 0.01 to 0.36, p = 0.041). Results were similar in multiply imputed and complete case samples.</td>
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<td>Kane 2009 United States of America</td>
<td>Cross-sectional</td>
<td>n = 81</td>
<td>Total n = 81; Males n = 40; Females n = 41; M = 11.8 (0.63)</td>
<td>Depressive symptoms Beck's Depression Inventory</td>
<td>Internalising symptoms Child Behaviour Checklist; Youth Self Report</td>
<td>Mother's history of depression and mother's current depressive symptoms</td>
<td>Paternal depressive symptoms were highly correlated with internalising symptoms on the Child Behaviour Checklist (r = 0.33, p &lt; 0.01), but not on the Youth Self Report (r = 0.12, p &gt; 0.05). Regression results were not fully reported. After all adjustments, paternal depression and adolescent depressive symptoms were positively associated in both the Growing up in Ireland cohort (0.24, 95% CI: 0.03 to 0.45, p = 0.023) and the Millennium Cohort Study (0.18, 95% CI: 0.01 to 0.36, p = 0.041). Results were similar in multiply imputed and complete case samples.</td>
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<tr>
<td>Lewis 2017 Republic of Ireland and United Kingdom</td>
<td>Two prospective cohorts - Growing up in Ireland and Millennium Cohort Study</td>
<td>n = 6070; n = 7768</td>
<td>Growing up in Ireland: Total n = 6070; Males n = 2909; Females n = 2951 b; M = 9 (0.13) at baseline; M = 13 (0.13) at follow-up. Millennium Cohort Study: Total n = 7768; Females n = 3945; Males n = 3823; 7 years at baseline; 14 years at follow-up</td>
<td>Growing up in Ireland Depressive symptoms Centre for Epidemiological Studies Depression Scale Millennium Cohort Study Psychological distress Kessler-6</td>
<td>Growing up in Ireland: Depressive symptoms Short Mood and Feelings Questionnaire</td>
<td>Maternal depressive symptoms, family income, paternal and maternal education, paternal and child age at time of exposure, child sex, ethnicity, whether the father was a biological parent, paternal and maternal alcohol use at time of exposure, child emotional symptoms at time of exposure, interparental conflict</td>
<td>Paternal depressive symptoms were highly correlated with internalising symptoms on the Child Behaviour Checklist (r = 0.33, p &lt; 0.01), but not on the Youth Self Report (r = 0.12, p &gt; 0.05). Regression results were not fully reported. After all adjustments, paternal depression and adolescent depressive symptoms were positively associated in both the Growing up in Ireland cohort (0.24, 95% CI: 0.03 to 0.45, p = 0.023) and the Millennium Cohort Study (0.18, 95% CI: 0.01 to 0.36, p = 0.041). Results were similar in multiply imputed and complete case samples.</td>
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<td>Middeldorp 2016 Netherlands</td>
<td>Cross-sectional</td>
<td>n = 530</td>
<td>Total n = 757; Females n = 296; Males n = 375; 6–18 years; Females age M = 11.7 (3.4); Males age M = 10.5 (3.2)</td>
<td>Depression, anxiety, avoidant personality, attention deficit/ hyperactivity and antisocial personality problems Adult Self Report</td>
<td>Depression and anxiety Child Behaviour Checklist</td>
<td>Parent age, offspring age, parental education</td>
<td>In the final multivariable model, only paternal anxiety significantly predicted child depression (β = 0.18, SE = 0.06, p = 0.01) and child anxiety (β = 0.25, SE = 0.07, p = 0.001). Paternal depression was associated with adolescent depression (β = 0.79, SE = 0.39, p &lt; 0.05, odds = 2.20, 95% CI: 1.04 to 4.68) but not anxiety. Paternal alcohol dependence was not associated with adolescent depression or anxiety.</td>
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<tr>
<td>Ohannessian 2005 United States of America</td>
<td>Cross-sectional</td>
<td>n = 426</td>
<td>Total n = 426; Females 52%; Males 48% 13–17 years; M = 15.10 (1.42)</td>
<td>Lifetime diagnoses of alcohol dependence, depression Semi-Structured Assessment for the Genetics of Alcoholism for Adolescents</td>
<td>Major depression and anxiety Semi-Structured Assessment for the Genetics of Alcoholism for Adolescents</td>
<td>Family type (control versus proband), race, whether a family had more than one participating adolescent (case independence versus dependence), adolescent gender, paternal antisocial personality disorder. Maternal mental health and interaction terms between adolescent gender and parental mental health were also included in modelling</td>
<td></td>
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<tr>
<td>Ranøyen 2015 Norway</td>
<td>Cross-sectional</td>
<td>n = 3239</td>
<td>Total n = 5732; Females n = 2883; Males n = 2849; 13–18 years; M = 15.8 (1.6)</td>
<td>Anxiety and depression symptoms Cohort Norway Mental Health Index Alcohol abuse Cut down Annoyed Guilty Eye-opener scale</td>
<td>Anxiety and depression symptoms Symptom Check List-5 Social anxiety symptoms Social Phobia and Anxiety Inventory for Children</td>
<td>Adolescent age. Maternal mental health was also included in path models</td>
<td>Paternal alcohol abuse was not associated with any of the mental health indicators measured in either sons or daughters. In Structural Equation Modelling, paternal anxiety/depression was associated with daughter anxiety/depression (β = -0.158, p &lt; 0.001) and social anxiety (β = -0.097, p &lt; 0.001), and with son anxiety/depression (β = -0.109, p = 0.001) but not social anxiety (β = -0.053, p = 0.056).</td>
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<tr>
<td>Reeb 2009 United States of America</td>
<td>Prospective longitudinal cohort</td>
<td>n = 428</td>
<td>Total n = 451; Females n = 236; Males n = 215; M = 13.2 at Year 1</td>
<td>Depressed mood Symptom Checklist-90-Revised Depression Subscale</td>
<td>Depressed mood Symptom Checklist-90-Revised Depression Subscale</td>
<td>Previous adolescent depressive symptoms, maternal depressive symptoms, family income, parental age, parental education, gender</td>
<td>Paternal depressive symptoms were associated with increases in subsequent adolescent depressed mood symptoms (β = 0.180, p &lt; 0.01).</td>
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<td>Rognmo 2012 Norway</td>
<td>Cross-sectional</td>
<td>n = 4012</td>
<td>Total n = 4012; Females n = 2015; Males n = 1997; 12–19 years; M = 16.0</td>
<td>Alcohol abuse Cut down Annoyed Guilty Eye-opener scale</td>
<td>Mental distress (comprising depression and anxiety symptoms) Short version of the Hopkins Symptom Checklist-25</td>
<td>Adolescent age, gender, the other parent’s alcohol abuse, parental income and education, having siblings, extraversion, psychoticism</td>
<td>Paternal alcohol abuse was marginally associated with greater mental distress in offspring (b = 0.13, 95% CI: 0.00 to 0.25, p = 0.05).</td>
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<tr>
<td>Selimbasic 2017 Bosnia and Herzegovina</td>
<td>Cross-sectional</td>
<td>Not reported</td>
<td>Total n = 120; Females n = 59; Males n = 61; M = 12.69 (1.46)</td>
<td>PTSD symptoms Harvard Trauma Questionnaire</td>
<td>Internalising Symptoms Child Behaviour Checklist for parents of children aged 6 to 18 Depression Depression self-rating scale</td>
<td>None</td>
<td>Paternal total PTSD was positively correlated with scores on total CBCL internalising (r = 0.355, p &lt; 0.01). Paternal total PTSD was also positively correlated with the CBCL anxiety/depression subscale (r = 0.389, p &lt; 0.01), but negatively correlated with the CBCL withdrawal/depression subscale (r = −0.267, p &lt; 0.01). No statistically significant findings emerged for adolescent depression level (p &gt; 0.05).</td>
</tr>
<tr>
<td>Tyrell 2018 United States of America</td>
<td>Longitudinal study</td>
<td>n = 392</td>
<td>Total n = 392; Females 52%; Wave 1 M = 12.89 (0.48); Wave 2 M = 13.89 (0.76); Wave 3 M = 15.53 (0.65)</td>
<td>Depressive symptoms Hopkins Symptom Checklist</td>
<td>Internalising symptoms Child Depression Inventory and Revised Children’s Manifest Anxiety Scale</td>
<td>Maternal depressive symptoms were also in the cross-lagged panel model</td>
<td>Paternal depression was not significantly associated with adolescent internalising symptoms at Waves 2 or 3 (p &gt; 0.05).</td>
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</table>

Abbreviations: M = Mean, OR=Odds Ratio, CI=Confidence Interval, PTSD=Posttraumatic Stress Disorder, SE=Standard Error.

a This is the figure for all participating children. It is unknown precisely how many participating children had fathers who also participated, although the author estimates n = ~477.
b Missing data.
c Information supplied by the author, not available from publication.
d Not reported in the publication, but calculated using information available in the publication.
Table 3
Risk of bias assessment for cohort studies.

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<thead>
<tr>
<th>Lead author, year</th>
<th>Exposed cohort truly or somewhat representative of the target population¹</th>
<th>Non-exposed cohort drawn from same community as exposed cohort</th>
<th>Paternal mental health ascertained via diagnostic procedure or named measurement instrument</th>
<th>Adjustment made for baseline or prior adolescent depression or anxiety in relevant analysis</th>
<th>Sample size justified and satisfactory</th>
<th>Study controls for any covariates (other than prior adolescent depression or anxiety)</th>
<th>Adolescent depression or anxiety ascertained via diagnostic procedure or named measurement instrument</th>
<th>Follow-up long enough for outcomes to occur (≥1 year)</th>
<th>Attrition low (&lt;20%) or described and accounted for in analysis</th>
<th>Statistical test clearly described and appropriate b</th>
<th>Total star rating (maximum = 10)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elgar 2007</td>
<td>*)</td>
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<td>*)</td>
<td>8/10</td>
</tr>
<tr>
<td>Lewis 2017</td>
<td>*)</td>
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<td>9/10</td>
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<tr>
<td>Reeb 2009</td>
<td>–</td>
<td>*)</td>
<td>*)</td>
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<td>*)</td>
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<td>*)</td>
<td>*)</td>
<td>7/10</td>
</tr>
<tr>
<td>Tyrell 2018</td>
<td>–</td>
<td>*)</td>
<td>–</td>
<td>–</td>
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<td>*)</td>
<td>–</td>
<td>–</td>
<td>5/10</td>
</tr>
</tbody>
</table>

* = criteria for study quality met; - = criteria for study quality not met.

¹ Determined by whole-population, random or purposive sampling, or evidence of sample representativeness.

b Determined by reporting of an appropriate effect estimate (such as beta or odds ratio), confidence intervals or the standard error, and a p value.
Table 4
Risk of bias assessment for cross-sectional studies.

<table>
<thead>
<tr>
<th>Lead author, year</th>
<th>Sample truly or somewhat representative of the target population</th>
<th>Comparability between respondents and non-respondents established and response rate satisfactory</th>
<th>Paternal mental health ascertained via diagnostic procedure or named measurement instrument</th>
<th>Sample size justified and satisfactory</th>
<th>Study controls for any covariates</th>
<th>Adolescent depression or anxiety ascertained via diagnostic procedure or named measurement instrument</th>
<th>Statistical test clearly described and appropriate</th>
<th>Total star rating (maximum = 7)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Agerup 2015</td>
<td>*</td>
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<td>*</td>
<td>*</td>
<td>5/7</td>
</tr>
<tr>
<td>Amrock 2014</td>
<td>-</td>
<td>-</td>
<td></td>
<td>*</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>4/7</td>
</tr>
<tr>
<td>Boričević Mašanić 2014</td>
<td>-</td>
<td>-</td>
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<td>*</td>
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<td>*</td>
<td>4/7</td>
</tr>
<tr>
<td>Choi 2013</td>
<td>-</td>
<td>-</td>
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<td>*</td>
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<td>-</td>
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<td>3/7</td>
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<tr>
<td>Kane 2009</td>
<td>-</td>
<td>-</td>
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<td>*</td>
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<td>-</td>
<td>-</td>
<td>2/7</td>
</tr>
<tr>
<td>Middeldorp 2016</td>
<td>-</td>
<td>-</td>
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<td>*</td>
<td>-</td>
<td>-</td>
<td>*</td>
<td>4/7</td>
</tr>
<tr>
<td>Ohannessian 2005</td>
<td>-</td>
<td>-</td>
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<td>*</td>
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<td>*</td>
<td>4/7</td>
</tr>
<tr>
<td>Ranøyen 2015</td>
<td>*</td>
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<td>*</td>
<td>-</td>
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<td>4/7</td>
</tr>
<tr>
<td>Rognmo 2012</td>
<td>*</td>
<td>-</td>
<td></td>
<td>*</td>
<td>-</td>
<td>-</td>
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<td>5/7</td>
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<tr>
<td>Selimbasic 2017</td>
<td>-</td>
<td>-</td>
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<td>*</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>2/7</td>
</tr>
</tbody>
</table>

* = criteria for study quality met; - = criteria for study quality not met.

a Determined by whole-population, random or purposive sampling, or evidence of sample representativeness.

b Determined by reporting of an appropriate effect estimate (such as beta or odds ratio), confidence intervals or the standard error, and a p value.
Where this was specified, there was heterogeneity between the studies: for instance, Ohannessian et al. (2005) only sampled biological fathers, Tyrell et al. (2018) further included stepfathers, and Elgar et al. (2007) also included stepparents, adoptive parents and foster parents. Additionally, many of the studies acknowledged some selection bias that caused the representativeness of their samples to be in doubt, and there was often limited capacity to generalise their findings to other populations. However, a major strength of the included studies is that almost all of them gave some consideration to covariates and confounders.

It was required that all studies use a validated instrument or diagnostic procedure to measure the relevant exposures and outcomes, so the psychometric properties of instruments in included studies are largely reasonable. However, there were some issues, such as Choi et al. (2013) using adolescents as informants to measure paternal drinking problems. There was also variation in whether studies assessed parents and adolescents for a diagnosis, or whether they measured symptoms, the clinical importance of which may be in question. Additionally, the procedures for data collection were sometimes poorly reported. In particular, blinding of assessors was only explicitly discussed in one study (Agerup et al., 2015), although this is likely because many studies used self-report measures rather than assessors.

3.3. Paternal depression and anxiety

Eight studies investigated the association between paternal depression and adolescent outcomes. Most studies found evidence for associations between paternal depression and either adolescent depression, anxiety, or internalising problems (Elgar et al., 2007; Kane & Garber, 2009; Lewis et al., 2017; Ohannessian et al., 2005; Ranoyen, Klockner, Wallander, & Jozefiak, 2015; Reeb & Conger, 2009). Three of these were longitudinal studies which received high study quality ratings. In particular, Lewis et al. (2017) employed two large prospective cohorts, providing evidence for a possible causal relationship between paternal and adolescent depression. Meanwhile, only two cross-sectional studies (neither of which had high study quality ratings) investigated paternal anxiety as an exposure, but both found it to be associated with adolescent depression and anxiety (Middeldorp et al., 2016; Ranoyen et al., 2015).

The majority of studies therefore found associations between paternal and adolescent depression and anxiety. However, some findings were contradictory. Tyrell et al. (2018) did not find paternal depressive symptoms to predict internalising problems in adolescent waves of their cross-lagged model of longitudinal data, although this was rated as the lowest quality longitudinal study. Middeldorp et al. (2016) also did not find any evidence for a cross-sectional association between paternal depression and adolescent anxiety or depression, although they acknowledge a response bias in their sample, with a possible underestimation of psychopathology prevalence rates in fathers. Ohannessian et al. (2005) found paternal depression to be cross-sectionally associated with adolescent depression but not anxiety, and Kane and Garber (2009) found paternal depressive symptoms to be cross-sectionally associated with mother-reported (Achenbach, 1991a), but not self-reported adolescent internalising symptoms (Achenbach, 1991b). Finally, Agerup et al. (2015) did not find paternal internalising problems to be cross-sectionally associated with adolescent depression, although their fairly modest sample size may have limited their power to detect an effect.

3.4. Paternal alcohol misuse

Choi et al. (2013) found a cross-sectional association between paternal alcohol misuse and adolescent depression and anxiety. However, it should be noted that Choi et al. (2013) did not consider the potentially confounding effects of socioeconomic status, and only measured paternal drinking using the adolescent as an informant, so the validity of their findings are in doubt. The remaining studies did not find alcohol abuse or dependence to be associated with adolescent depression or anxiety (Ohannessian et al., 2005; Ranoyen et al., 2015), or else only found a borderline significant association (Rognmo et al., 2012), and no longitudinal studies investigated this association.

3.5. Other paternal mental health disorders

Six studies investigated other paternal mental health disorders. Paternal externalising problems (Agerup et al., 2015), antisocial personality disorder and Attention Deficit Hyperactivity Disorder (Middeldorp et al., 2016) were not found to be cross-sectionally associated with adolescent depression or anxiety. However, findings regarding other paternal affective or anxiety disorders were very mixed. Paternal nonspecific psychopathology or distress predicted adolescent depression in a large, high quality longitudinal study (Lewis et al., 2017), but not adolescent emotional symptoms in a large cross-sectional study (Amrock & Weitzman, 2014). Paternal PTSD was cross-sectionally associated with adolescent internalising symptoms in Borićević Maršanić et al. (2014) but gave conflicting results in Selimbasic et al. (2017). Finally, paternalAvoidant personality problems were not cross-sectionally associated with adolescent depression and anxiety in Middeldorp et al. (2016).

3.6. The role of maternal mental health

The role of maternal mental health was considered in the majority of studies. Reeb and Conger (2009) took account of the effects of maternal mental health as a covariate in longitudinal analyses, while others considered maternal mental health as another exposure associated with of adolescent mental health. Their findings largely suggested that adolescent anxiety or depression was associated with both maternal and paternal mental health, often reporting similar statistically significant or non-significant associations for each parent in both cross-sectional and longitudinal studies (Amrock & Weitzman, 2014; Lewis et al., 2017; Middeldorp et al., 2016; Ranoyen et al., 2015; Tyrell et al., 2018). However, there were some exceptions. For instance, maternal but not paternal...
depression was cross-sectionally associated with adolescent anxiety in Ohannessian et al. (2005). Two other cross-sectional studies suggested that adolescent wellbeing might be more associated with maternal than paternal mental health (Agerup et al., 2015; Rognmo et al., 2012).

3.7. Other findings

Several studies considered the role of adolescent gender, although the way they did so was varied, with some adjusting analyses for gender as a covariate and others stratifying analyses by gender. Among those which aimed to explore the effect of adolescent gender, one longitudinal study found a greater impact of paternal mental health in girls than in boys (Reeb & Conger, 2009). Others found that the effect of adolescent gender varied according to whether the outcome under study was depression or anxiety (Choi et al., 2013) and according to whether associations were concurrent or longitudinal (Elgar et al., 2007). Still more found no significant evidence for an effect of adolescent gender (Middeldorp et al., 2016; Ohannessian et al., 2005; Ranoyen et al., 2015; Rognmo et al., 2012; Selimbasic et al., 2017).

A range of other mediators and moderators in the relationship between paternal and adolescent mental health were also explored. However, these findings are tentative and should be interpreted with caution. For instance, there was longitudinal evidence from one study that parental rejection and low parental monitoring may be mediators on the pathway between parental depression more generally and adolescent internalising disorders (Elgar et al., 2007). Another longitudinal study suggested that father-daughter closeness may modify the effect of paternal depression on female adolescent depression (Reeb & Conger, 2009). Findings from other studies were more inconclusive. Rognmo et al. (2012) proposed a range of mediators and moderators between paternal alcohol abuse and adolescent mental health, but found no significant interactions, and did not present adequate mediation analyses to properly determine their role in the association. It is similarly unclear how the modifying effects of ethnicity, family structure and adolescent gender impacted Tyrell et al. (2018) findings during the study waves limited to adolescence.

4. Discussion

The primary aim of this review was to synthesize the evidence on the relationship between paternal mental health and adolescent anxiety and depression. Overall, there is evidence to suggest that some mental health disorders in fathers are associated with anxiety and depression in adolescents. Consistent with previous research, paternal depression was the most strongly evidenced variable associated with adolescent anxiety and depression, while evidence for an association with other paternal disorders was more limited.

4.1. Research implications

Paternal depression has received a large amount of attention in recent years, and as was the case at the time of Ramchandani and Psychogiou’s (2009) review, depression remains the most comprehensively studied psychiatric disorder in this field. But the effect of other paternal affective and anxiety disorders has been comparatively neglected. Other studies which investigated the effects of paternal anxiety including children outside the 11–17 year age range have produced mixed results (Al-Turkait & Ohaeri, 2008; Clark, Cornelius, Wood, & Vanyukov, 2004; Esbjorn et al., 2013). Depression and anxiety are highly comorbid (Kessler et al., 2015), and as such studies about depression may also speak to the association between paternal and adolescent anxiety to some extent. However, the research landscape would now benefit from a robust, large scale longitudinal study on the effect of paternal anxiety on adolescent outcomes. It would also benefit from further investigation of the effects of paternal comorbidities and of other paternal anxiety disorders, such as Obsessive Compulsive Disorder and PTSD.

Most studies did not find that alcohol misuse and other behavioural disorders in fathers were associated with anxiety or depression in adolescents. Findings in other age groups are more mixed (Agerup, Lydersen, Wallander, & Sund, 2014; Ayer, Kohl, Malsberger, & Burgette, 2016; Fals-Stewart, Kelley, Fincham, Golden, & Logsdon, 2004; Kelley & Fals-Stewart, 2004; Moss, Baron, Hardie, & Vanyukov, 2001; Moss, Lynch, Hardie, & Baron, 2002), but in this regard our findings are consistent with the suggestion that specific mental health disorders in parents are more likely to be associated that same mental health disorder in children (Clark et al., 2004). Although continued clarification of whether externalising disorders in fathers are associated with internalising disorders in adolescents might be beneficial, future research could focus on paternal anxiety and depression as risk factors for anxiety and depression in adolescents.

Connell and Goodman (2002) and Ramchandani and Psychogiou (2009) suggested that mothers might have a greater impact on emotional and internalising outcomes. Findings in other age groups regarding the primacy of maternal versus paternal mental health are mixed (Agerup et al., 2014; Al-Turkait & Ohaeri, 2008; Clark et al., 2004; Jacobs, Talati, Wickramaratne, & Warner, 2015). However, in our review the association between paternal mental health and adolescent anxiety and depression was not negligible, and most included studies did not find maternal mental health to be more influential than paternal mental health. Therefore the importance of fathers in adolescent emotional wellbeing must not be underestimated, and future research should continue to consider the effect of fathers to an equal extent as mothers.

There is speculation in the literature that sons might be more exposed to the effects of paternal mental health disorders compared to daughters. However, the small number of our included studies which investigated this did not find strong evidence for adolescent gender moderating the association between paternal and adolescent mental health. This finding might be interpreted in various ways. For instance, the increased vulnerability experienced by boys who are thought to spend more time with fathers (Raley & Bianchi, 2006) might be offset by the increased vulnerability experienced by girls who are thought to be more empathetic to the emotions of
others (Christov-Moore et al., 2014).

Other factors such as parenting practices and the father-adolescent relationship may play a role. Parenting practices are included in Ramchandani and Psychogiou’s (2009) proposed model on the pathway between paternal and child psychopathology. While this model might also apply to adolescents, it has been noted that the nature of relationships between fathers and their offspring changes as the child ages (Flouri & Buchanan, 2003). Therefore, a similar but distinct model might be developed for adolescents, with greater emphasis on factors which might be especially pertinent in the adolescent age group, such as father-adolescent conflict (Sheeber, Davis, Leve, Hops, & Tildesley, 2007) and peer support (Vaughan, Foshee, & Ennett, 2010). Additionally, the potentially mediating or moderating effects of family dynamics and structure may be worthy of further investigation in this particular context (Bramlett & Blumberg, 2007), such as the role of non-resident fathers (Flouri, 2006) and other family-level factors thought to mediate the association between father and adolescent mental health, such as marital conflict (Sweeney & MacBeth, 2016). Future studies should continue to explore mechanisms in the association between paternal and adolescent mental health, towards developing a pathway model which is informed by what is already known for younger children, but which also considers other factors relevant to the adolescent development period.

Finally, it is important to note that the focus of this review was the association between paternal mental health as an exposure and adolescent mental health as an outcome, and we only included studies which treated these variables as such. However, many of the studies included in this review were cross-sectional, such that we are unable to infer a direction of effect with any certainty. Indeed, there is growing evidence to suggest that the relationships between child and parent mental health may be reciprocal (Ahmadzadeh et al., 2019). Exploring bidirectional effects was beyond the scope of this review, but should be a topic for further investigation.

4.2. Clinical implications

Given the small number of high quality, longitudinal studies identified in this review, caution is warranted in adopting these findings into clinical practice. Nonetheless, the potential association between paternal and adolescent mental health could have tentative implications for how fathers and adolescents are supported. First, it remains important that primary care settings be equipped to identify mental health disorders in fathers, and that support is equally accessible for both mothers and fathers, particularly in light of known gender disparities in help-seeking behaviour (Oliver, Pearson, Coe, & Gunnell, 2005). Second, the mental well-being of the father (and indeed the family more generally) should continue to be considered when assessing and treating adolescents for mental health disorders. Kane and Garber (2004) have previously suggested that family-based interventions might benefit from including and supporting fathers as part of efforts made to treat adolescent mental health disorders. Whether or not this is successful may depend on other factors mediating or moderating the relationship between paternal and adolescent mental health – future efforts to explore these mechanisms will therefore contribute to the discussion around how best to intervene when paternal and adolescent mental health disorders co-occur.

4.3. Limitations

Our search terms and strategy were in keeping with the primary aim of our review, and we followed PRISMA reporting guidelines (Moher, Liberati, Tetzlaff, & Altman, 2009). However, our review has several limitations. First, due to time constraints, key researchers in the field were not contacted to suggest relevant titles.

Second, attempts to focus on the adolescent age range of 11–17 years were hampered by the variability of age ranges adopted in studies, with many excluded because they recruited children of various ages but did not report separate results for adolescents and younger children. This speaks to the challenge of identifying the precise definition of adolescence (Sawyer et al., 2018). Adolescence does represent a developmental period encompassing major biological and social transitions, and efforts to distinguish it from childhood in research and analyses must be made. Future studies should avoid conflating childhood and adolescence, and some attempt to stratify by age should be made in statistical analyses, such that conclusions on the differential associations in childhood and adolescence can be drawn.

Third, as discussed in our results section, the quality of included studies was mixed. Study quality was rated using existing versions of the NOS, and some items were less suited to this review – for instance, studies were only included if they used validated measurement instruments or diagnostic procedures for assessing mental health, so all studies necessarily met quality criteria for methods of exposure and outcome ascertainment. Nonetheless, inconsistent reporting of important methodological features such as study design, power calculations, data collection procedures, and the possible role of bias were particularly concerning. Authors in this field would benefit from following reporting guidelines for observational studies (von Elm et al., 2007).

Finally, we only included studies which investigated the direct association between paternal and adolescent mental health as part of the primary aim. While this ensured that potentially spurious secondary findings were not included, relevant findings may have been missed. Additionally, we could not draw meaningful conclusions regarding mechanisms in the relationship between paternal and adolescent mental health, because our primary interest was in the direct association.

5. Conclusions

The findings of this review reiterate the importance of paternal depression in adolescent mental health. However, the findings also highlight the need for continued consideration of the relationship between other paternal mental health disorders, particularly anxiety disorders, and adolescent anxiety and depression. Additional priorities for research include further exploration of
mechanisms, and study designs which can imply a causal direction and which do not conflate child and adolescent age groups. Importantly, despite previous implications that fathers primarily influence adolescent externalising and behavioural problems, evidence that they were any less implicated in their adolescent's emotional wellbeing than mothers was sparse in this review. The importance of the father in adolescent emotional wellbeing should not be underestimated.

Contributions

AW conducted the search, extracted the data, conducted risk of bias assessments, and wrote all versions of the manuscript. DL cross-checked the search and data extraction, conducted risk of bias assessments, and contributed to all versions of the manuscript. MA contributed to synthesis. NTF participated in design of the work and commented on drafts of the manuscript. All authors have approved the manuscript.

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NTF is part funded by the United Kingdom's Ministry of Defence, sits on the Independent Group Advising on the Release of Data at NHS Digital, and is also a trustee of two military related charities. The views expressed are those of the authors and not necessarily those of the NHS, the NIHR, the Department of Health and Social Care or the UK Ministry of Defence. DL and MA declare no other conflicts of interest.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.adolescence.2020.01.007.

References
