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Anxiety Disorders in Adolescents and Psychosocial Outcomes at Age 30

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Abstract

Background—Anxiety disorders are associated with adverse psychosocial functioning, and are predictive of a wide range of psychiatric disorders in adulthood.

Objective—The present study examined the associations between anxiety disorders during childhood and adolescence and psychosocial outcomes at age 30, and sought to address the extent to which psychopathology after age 19 mediated these relations.

Method—Eight hundred and sixteen participants from a large community sample were interviewed twice during adolescence, at age 24, and at age 30. They completed self-report measures of psychosocial functioning and semi-structured diagnostic interviews during adolescence and young adulthood.

Results—Childhood anxiety only predicted less years of completed education at age 30, whereas adolescent anxiety predicted income, unemployment, maladjustment, poor coping skills, more chronic stress and life events. Adult major depressive disorder (MDD) was the only disorder predicted by childhood anxiety, whereas adolescent anxiety predicted MDD, substance (SUD) and alcohol abuse/dependence (AUD) in adulthood. No adult psychopathology mediated the relationship between childhood anxiety disorders and psychosocial outcomes at age 30. Adult MDD, SUD and AUD partially or completely mediated the association between adolescent anxiety and most domains of psychosocial functioning at age 30.

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Contributors

Cecilia Essau wrote the manuscript. Peter Lewinsohn and John Seeley designed the study and wrote the protocol. Beatriz Olaya undertook the statistical analysis. All authors contributed to and have approved the final manuscript.

Conflict of Interest

The authors have no conflict of interest to report in relation to the research presented in this manuscript.

Limitations—The participants are ethically and geographically homogenous, and changes in the diagnostic criteria and the interview schedules across the assessment periods.

Conclusion—Adolescent anxiety, compared to childhood anxiety, is associated with more adverse psychosocial outcomes at age 30. Adolescent anxiety affects negative outcomes at age 30 directly and through MDD, SUD and AUD.

Keywords

Anxiety disorders; Psychosocial impairment; Course of anxiety disorders; Adolescent anxiety

1. Introduction

Anxiety disorders are among the most common disorders affecting adolescents (Costello et al., 2005). Recent epidemiological studies estimated that the prevalence of anxiety disorders in adolescents ranges between 10% and 31.9% (Merikangas et al., 2010). The high frequency of anxiety disorders in adolescents means that these disorders tend to have an early onset. Some anxiety disorders (e.g., separation anxiety disorder, specific phobias) tend to have an onset in childhood, while others (e.g., social anxiety) tend to have an onset in adolescence (Beesdo et al., 2009). In addition to being prevalent, anxiety disorders co-occur highly among themselves and with numerous other psychiatric disorders (Essau, 2003; Essau et al., 2000; Feehan et al., 1993; Lewinsohn et al., 1997; Wittchen et al., 1998). The most common comorbid pattern was that of anxiety and depressive disorders (Essau et al., 2000; Lewinsohn et al., 1997), with comorbidity rates ranging from 50 to 72%. Among those with both disorders, up to 75% reported the first onset of anxiety before that of depression (Essau et al., 2000). Adolescents with anxiety and comorbid disorders tend to have more severe symptoms of their disorders (Essau, 2005), higher mental health utilization (Essau, 2005; Lewinsohn et al., 1995), and higher rates of suicidal behavior (Rohde et al., 2001). Most anxiety disorders have an early onset, generally in childhood or early adolescence (Kessler et al., 1994; Mathew et al., 2011). Thus, the question of what happens to children and adolescents with an anxiety disorder after they become adults is of great concern.

According to several follow-up studies, anxiety disorders that begin early in life can become chronic (Feehan et al., 1993; Ferdinand and Verhulst, 1995; Keller et al., 1992; Letcher et al., 2012; Pine et al., 1998) and are associated with a high probability of recurrence (Bruce et al., 2005). The presence of anxiety disorders during adolescence also predicted a two- to threefold increased risk for anxiety in adulthood (Pine et al., 1998). Mathew and colleagues (2011) showed anxiety disorders in adolescence significantly predict the onset of MDD in adulthood. Adolescents who had more than three anxiety disorders had (a rate of MDD 3.5 times higher) and (a rate of illicit drug dependence 4 times higher) than peers without any anxiety disorders (Woodward and Fergusson, 2001).

Adolescents with anxiety disorders have been reported to show significant impairment in multiple domains of psychosocial functioning (e.g., educational underachievement; Woodward and Fergusson, 2001), as well as general health, physical and cognitive functioning in adulthood (Essau et al. 2000; Feehan et al., 1993; Ferdinand and Verhulst,

1995; Keller et al., 1992; Lewinsohn et al., 1998; Pine et al., 1998; Reinherz et al., 1993). However, the mechanisms through which anxiety disorders impact psychosocial outcomes are unknown. Recent studies have documented similarity in psychosocial impairments experienced by adolescents with anxiety, MDD, and SUD (Angold et al. 1999; Essau, 2003, 2008; Karlsson et al., 2006; Nottelmann and Jensen, 1999). Studies have also identified significant overlap in the risk factors for anxiety and MDD such as being female and stressful life events (Essau et al., 2000; Lewinsohn et al., 1995). Additionally, Mathew et al. (2011) found that poor interpersonal functioning in adolescents conferred risk for both anxiety and depression; these factors included loneliness, emotional reliance, and impaired relations with family and peers.

Due to the high comorbidity between anxiety, MDD and SUD, it remains unknown whether psychosocial impairments are specific to anxiety or to these comorbid disorders. The association between adolescent anxiety and psychosocial impairments in adulthood could be related to the fact that many adolescents with anxiety disorders experience another psychiatric disorder as adults (Keller et al., 1992; Pine et al., 1998). It is possible that having other disorders or recurrent anxiety disorder accounts for psychosocial impairments in adulthood (Keller et al., 1992). Furthermore, psychosocial impairment observed in adulthood could have been present in adolescence. As reported in several studies, adolescents with anxiety disorders are significantly impaired in various life domains, especially in social and academic performances (Essau, 2003). In this case, adult psychosocial impairments observed in anxious adolescents may reflect continuities in psychosocial, some of which may have preceded, and may even have contributed to, adolescent anxiety.

On the basis of this general background, the present study reports the result of a 16-year longitudinal study on the association between an early onset of anxiety (i.e., childhood and adolescent anxiety) and psychosocial functioning in adulthood. Because most anxiety disorders tend to have an onset either in childhood or during adolescence, the present study will categorize the age of onset of anxiety into childhood and adolescence. The more specific aims are to address the following questions: (a) What is the association between childhood and adolescent anxiety and psychosocial outcomes at age 30? The psychosocial outcomes that were explored included highest education level completed, recent unemployment, annual household income, poor physical health, and family and friends support – as these are the most common outcomes being identified in similar longitudinal studies (Mathew et al., 2011; Woodward and Fergusson, 2001). (b) What are the associations between childhood and adolescent anxiety and psychopathology after age 19? The types of psychopathology examined were anxiety, MDD, SUD, AUD. (c) Did other forms of psychopathology in adulthood mediate the relationship between childhood or adolescent anxiety and psychosocial outcome at age 30? (d) What are the associations between psychopathology after age 19 and psychosocial outcomes at age 30?

The hypotheses to be tested in this study were as follows: First, based on previous studies (Mathew et al., 2011), early onset anxiety (i.e., childhood or adolescent anxiety) is hypothesised to be associated with psychosocial impairment in academic, employment, health, and social/family domains. Specifically, individuals with a childhood-onset anxiety,

compared to those with an adolescent-onset anxiety are hypothesised to have low education achievement, recent unemployment, low annual household income, poor physical health, and lack of family and friends support. Second, childhood and adolescent anxiety is associated with the presence of MDD, AUD, and SUD after age 19. Third, the presence of adult psychopathology is expected to mediate the relationship between childhood or adolescent anxiety and psychosocial outcomes at age 30. Finally, there will be a strong association between psychopathology after age 19 and psychosocial outcomes at age 30.

To our knowledge, this is the first study that has differentiated between anxiety disorders that begin early in life by their age of onset in childhood and in adolescence. This is surprising given differences among anxious children and adolescents in duration, severity, comorbidity patterns and correlates of anxiety disorders (Essau, 2005; Orgiles et al., 2012). Therefore, what is needed is a study that examines the association between childhood and adolescent anxiety and psychosocial functioning at adulthood. Another novel aspect of this research is to examine the extent to which other psychopathology in adulthood mediate the association between childhood and adolescent onset anxiety.

2. Methods

2.1. Participants

The present study used data from the Oregon Adolescent Depression Project (OADP) (Lewinsohn et al., 1993), a longitudinal study of a large cohort of high school students who were randomly selected from nine high schools in western Oregon as previously described (Rohde et al., 2007) (Figure 1). A total of 1,709 adolescents (ages 14–18; mean age 16.6, SD=1.2) completed the initial (T1) assessments. About a year later, all T1 participants were invited to participate in the second assessment. However, 1507 adolescents, with a mean age of 17.2 years participated at time 2 (T2; 88%). For the third assessment, all adolescents with a history of a depressive disorder by T2 (n=360) or a history of non-mood disorders (n=284), and a random sample of adolescents with no history of psychopathology by T2 (n=457) were invited to participate in a third (T3) evaluation. All non-white T2 participants were retained in the T3 sample to maximize ethnic diversity. A total of 1101 young adults participated at time 3 (T3), with a mean age of 24.2 years. At age 30, all T3 participants were asked to complete another interview assessment (mean age=30.45, SD=0.70). Of the 941 who participated in the T3 assessment, 816 (87%) completed the T4 assessment; due to missing data, the sample size ranged from 752 to 816 for individual outcome variables.

Of these 816 individuals, more than half of the sample were female (58.8%), with a mean age at T4 of 29.7 years (SD=0.70). The majority of the participants were White (85.9%), others were African American (1%), Hispanic (3%), American Indian (3%), Asian (3%), and “other” (2%). About half of them were married (56.2%), and 41% had a bachelor's degree or higher.

2.2. Measures

2.2.1. Diagnostic Measures—At T1 and T2, participants were interviewed with a version of the Schedule for Affective Disorders and Schizophrenia for School-Age Children

(KSADS; Orvaschel et al., 1982). Follow-up assessments at T2 and T3 were jointly administered with the Longitudinal Interval Follow-Up Evaluation (LIFE; Keller et al., 1987). The K-SADS/LIFE procedure provided information regarding the onset and course of disorders since the previous interview. The T4 interview consisted of a joint administration of the LIFE and the Structured Clinical Interview for DSM-IV (SCID; First et al., 1996) to probe for new or continuing episodes since T3. Diagnoses were based on DSM-III-R criteria for T1 and T2 and Diagnostic and Statistical Manual of Mental Disorders, 4th edition (DSM-IV) (American Psychiatric Association, 1994) criteria for T3 and T4. The aggregate anxiety disorder diagnostic category included generalized anxiety disorder; overanxious disorder; post-traumatic stress disorder; panic disorder; agoraphobia; social phobia; simple phobia; obsessive-compulsive disorder; and separation anxiety disorder. SUD included alcohol abuse or dependence (AUD) and sedative/hypnotic/anxiolytic, cannabis, stimulant, opioid, cocaine, and hallucinogen/PCP abuse or dependence, and polydrug dependence. Disruptive disorder included attention deficit and hyperactivity disorder, oppositional defiant disorder and conduct disorder. The interrater reliability for depressive disorders (MDD or dysthymia) in the four time assessments ranged from 0.82 to 1.00; for anxiety disorders it ranged from 0.76 to 0.87; and for SUD from 0.81 to 0.96.

The criterion to define the presence of childhood anxiety was that at least one anxiety episode or disorder had an onset before age 11. Participants with adolescent anxiety were those who had at least one episode or disorder with an onset at age 11 or after, and also included those with a childhood episode with a continuity into adolescence. Finally, those with at least one episode or disorder with an onset at or after age 19 were considered to have an adult anxiety disorder, and also included participants with a childhood or adolescent episode which continues into adulthood (i.e. after age 19). One participant could have one, two or three types of anxiety. Age of onset was not treated as a continuous variable because categorizing continuous variables might lead to loss of information and power. Furthermore, categorizing the age of onset for anxiety disorder makes the analysis and interpretation of results simple. First, the presence of anxiety disorders was not exclusive, as one participant could have one, two or three types of anxiety disorders. In this way, it is possible to control for the presence of other predictors, for example, to examine the effect of childhood anxiety above and beyond the effect of adolescent anxiety and vice versa. Second, the use of anxiety as a categorized variable enabled the determination of the distribution of the disorders by gender, age; this method also made it possible to examine the prevalence of comorbidity with other mental disorders (MDD, AUD, SUD and disruptive disorders).

Participants were also classified as having a MDD, AUD, SUD and disruptive disorders (i.e., included attention deficit and hyperactivity disorder, oppositional defiant disorder and conduct disorder) before age 19 if they had at least one episode or disorder with an onset before that year. Participants with at least one episode at or after age 19 were considered to have an adult disorder.

2.2.2. Psychosocial Functioning—At each assessment, a battery of psychosocial measures was administered to the participants (e.g., Lewinsohn et al., 1994, 2003). Measures of adult functioning in the present study are based on data obtained at the T4 (age 30) assessment. (a) Academic and occupation functioning at age 30 included: highest grade

completed; number of weeks of unemployment during the past year (0 weeks; 1 to 13; 14 to 26; 27 to 39; 40 to 51; and 52 or more weeks); and annual household income (ranging from no income to \$50,000 or more). (b) Quality of the relationship with family members and friends ($\alpha=.90$; 20 items; Procidano and Heller, 1983); and social adjustment over the past 2 weeks, as assessed by mean item score on the 54-item Social Adjustment Scale (SAS) and its subscales (adjustment at work, social/leisure activities, family, marital role, parental role, and family unit) ($\alpha=.70$; Weissman and Bothwell, 1976; higher scores indicate poorer adjustment). (c) Health functioning included poor physical health ($\alpha=.50$; self-rated physical health codified as 0=good or very good, and 1= regular to bad), and number of suicide attempts. (d) Other measures included: life satisfaction ($\alpha=.89$; 15 items chosen from Andrews and Withey, 1976; Campbell et al., 1976; higher scores indicate lower satisfaction); coping skills ($\alpha=.77$; 17 items; Rohde et al., 1990); and stressful life events ($\alpha=.71$; 33 items; Holmes and Rahe, 1967).

2.2.3. Statistical Analysis—Analyses were restricted to participants with complete information at T4 ($n = 816$); Mediation analysis was comprised of a series of regression analyses (Baron & Kenny, 1986). To be considered a mediator, the exposure (childhood and adolescent anxiety) must be associated with the outcome (psychosocial outcomes at age 30); the mediator (adult anxiety, MDD, SUD, and alcohol abuse) must be predicted by the exposure; the outcome must be predicted by the mediator while adjusting for the exposure; and the effect estimate of the exposure must be reduced when adjusting for the mediator.

To test the criteria for mediation, generalized estimating equations (GEE; Liang and Zeger, 1986; Zeger and Liang, 1986) were used to explore the relationship between predictor variables, mediators and outcomes. GEE were used to control for ‘school’ clustering data. In GEE, the standard errors of the regression coefficients are estimated in a way that accounts for correlated observations within schools. Binomial distribution with logit link function were used when the dependent variable was binary (adult anxiety, MDD, SUD and AUD, poor health status). Multinomial distribution with cumlogit link were used when the psychosocial outcomes were ordinal (years of completed education, number of weeks unemployed, and income), normal distribution with identify link for quantitative outcomes (SAS questionnaire scores, coping skills score), and poisson distribution and log link for integer data (family and friends support, number of suicidal attempts, chronic stress level and number of stressful live events).

Since comorbidity of anxiety and depression is frequently high, the effect of Anxiety on psychosocial functioning could be explained by the presence of MDD. Therefore, MDD was treated as potential confounder in all the GEE models. In the first step, a GEE model was performed for each psychosocial outcome, with both predictors (childhood and adolescent anxiety) introduced simultaneously and controlling for the effect of gender, the same psychosocial outcome at T1-T2 (when available), and the presence of MDD, SUD, AUD, and Disruptive Disorders before age 19. This sought to control for gender differences in anxiety disorders, stability of constructs over time, and the effects of psychopathology in general during childhood and adolescence. In the second step, separate GEE models were performed to test whether childhood or adolescent anxiety predicted adult anxiety, MDD, SUD and AUD, controlling for gender and the presence of psychopathology before age 19.

In the third step, GEE models were performed to determine the effect of potential mediators (anxiety, MDD, SUD and alcohol during young adulthood) on psychosocial outcomes at age 30, controlling for the effect of gender, the same psychosocial outcome at T1-T2, the predictors (childhood and adolescent anxiety) and psychopathology after age 19. By doing so, the possible effect of comorbid MDD is controlled in all models.

Sobel's tests for indirect effects were computed (Sobel 1982). Because the mediators and some outcomes were binary, standardized coefficients were used in the tests (MacKinnon & Dwyer, 1993). Evidence of mediation was supported if z scores were greater than 1.96 (MacKinnon, 2008). Analyses were performed using PASW 19.0. Bonferroni's adjustment for multiple tests was performed. Missing data were excluded from the analysis and assumed to be missing at random (Rubin, 1976).

3. Results

At T4, 207 participants (25.4% of the sample) met criteria for one or more lifetime anxiety disorders: 18 (8.7%) had a lifetime prevalence of at least one generalized anxiety disorder, 12 (5.8%) met criteria for a lifetime obsessive compulsive disorder, 44 (21.3%) for separation anxiety disorder, 38 (18.4%) for specific phobia, 36 (17.4%) for social phobia, 20 (9.7%) for panic disorder with agoraphobia, and 39 (18.8%) for panic disorder without agoraphobia. Of the 207 participants with a lifetime diagnosis of an anxiety disorder, 66.7% (n=138) had only one disorder or episode, 22.2% (n=46) had two disorders or episodes, and 11.1% (n=23) had 3 or more episodes.

Among all the participants at T4, 9.8% (n=80) had at least an anxiety disorder during childhood (i.e., before 11 years), 13.5% (n=110) had at least an anxiety disorder during adolescence (before age 19; i.e., between 12 – 18 years) and 17.5 % (n=143) presented one or more episodes of anxiety from age 19 through 30. Table 1 shows the distribution of gender and psychopathology before and after age 19 among participants with childhood and adolescent anxiety. There were more girls than boys among participants with childhood and adolescent anxiety ($p < 0.001$). MDD before and after age 19 were also more common among participants with childhood anxiety ($p < 0.001$) and with adolescent anxiety ($p < 0.001$) compared to those participants with no anxiety disorders.

3.1. First step: Association between childhood and adolescent anxiety with psychosocial outcomes at age 30

Separate GEE models were performed to determine the association between psychosocial outcomes at age 30 (dependent variables) and childhood and adolescent anxiety. Both types of anxiety were introduced simultaneously in each model, with other covariables. After adjusting for Bonferroni correction, adolescent anxiety was significantly associated with poor total adjustment, poor adjustment at work, poor family relationships, problems with the family unit, less life satisfaction, poor coping skills, and more chronic stress (Table 2). Childhood anxiety was not associated with psychosocial outcomes at age 30.

3.2. Second step: Associations between childhood and adolescent anxiety and psychopathology after age 19

We focused on anxiety, MDD, SUD, and AUD after age 19 as potential mediators. Table 3 shows that anxiety during adulthood was strongly associated with adolescent anxiety but not with childhood anxiety. Those participants with adolescent anxiety had also greater risk of SUD and AUD during adulthood after adjusting for Bonferroni's correction. There were no significant interactions between gender and the above moderators.

3.3. Third step: Associations between psychopathology after age 19 and psychosocial outcomes at age 30

The third step of mediation was to examine whether adult anxiety, SUD and AUD predicted the same psychosocial outcomes at age 30 that were predicted by adolescent anxiety in the first step. GEEs models were tested for each psychosocial outcome at age 30, including the mediators, and controlling for the predictor (i.e. adolescent anxiety) and gender, childhood anxiety, MDD, SUD, AUD, disruptive disorder before age 19, the same psychosocial outcome at T2, if available, and other psychopathology after age 19 (MDD, SUD, AUD and anxiety) (Table 3).

Adult SUD was significantly associated with poor total adjustment, poor adjustment at the family unit, less life satisfaction, poor coping skills, and more chronic stress. The Sobel's test of the indirect effect indicated that there was a significant mediating effect for SUD in the association between adolescent anxiety and total SAS ($z=2.22$), poor adjustment at the family unit ($z=2.84$), coping skills ($z=-2.46$), and chronic stress ($z=2.77$), whereas there was not significant effect in the association between adolescent anxiety and life satisfaction ($z=1.16$).

Table 4 also shows that adult AUD was significantly associated with problems at the family unit and greater chronic stress levels. Tests for indirect effect determined that AUD only mediated the relationship between adolescent anxiety and chronic stress ($z=2.02$).

Anxiety during young adulthood was significantly associated with several psychosocial outcomes at age 30, after adjusting for gender and psychopathology before and after age 19: poor health status, poor total adjustment, poor adjustment at work, problems with the family relationships, problems at the family unit, less life satisfaction, poor coping skills, and more chronic stress. In all these cases, adult anxiety significantly mediated the relationship between adolescent anxiety and these outcomes (z scores ranging from -2.47 to 4.28).

4. Discussion

To our knowledge, the present study is the first to systematically (a) examine the associations between childhood and adolescent anxiety and psychosocial outcomes at age 30, and (b) address the extent to which psychopathology after age 19 mediated these relations. The results contribute to our understanding of the long-term psychosocial outcomes of childhood and adolescent anxiety in several ways. First, because of the large representative sample size, the problems associated with poor generalizability and referral bias could be minimized. Second, the study contained comprehensive measures of personal,

academic and social background of all the participants, which enabled the examination of the common and specific impact for anxiety and for other psychiatric disorders, as well as their psychosocial outcome. Third, since a wide range of anxiety and comorbid disorders were assessed at several assessment periods with a follow-up period of about 16 years, it is possible to carefully document both the onset and course of a wide range of disorders.

Previous studies have consistently shown that anxiety disorders that begin early in life tend to predict MDD at adulthood (Mathew et al., 2011; Pine et al., 1998; Reinherz et al., 1989; Woodward and Fergusson, 2001). However, to our knowledge, no studies have compared childhood and adolescent onset of anxiety disorders. Therefore, a novel aspect of the present study was the examination of psychopathology and psychosocial outcomes at age 30 by age of onset of anxiety disorders. Specifically, with regards to psychopathology, childhood anxiety was found to predict only MDD in adulthood, whereas adolescent anxiety predicted MDD, SUD and AUD in adulthood. When further adjusted for other forms of psychopathology before age 19 and childhood anxiety, these effects remained significant. The finding that both childhood and adolescent anxiety predict major depression was in line with previous studies (Bittner et al., 2004; Goodwin, 2002; Kessler et al., 1999; Woodward & Fergusson, 2001). Why adolescent anxiety was a predictor of a number of adult psychopathology (i.e., MDD, SUD and AUD) was not clear. It could be that the types and clinical characteristics of anxiety disorders which had an onset at childhood differed from those that had an onset at adolescence. As reported by Bittner et al. (2004), the clinical characteristics of anxiety which increased the risk of developing a depression were the presence of at least one anxiety disorder, severe impairment as a consequence of anxiety disorder and comorbid panic attack. Numerous other studies (Bittner et al., 2004) have reported that panic attack generally has its onset in adolescence and is a predictor of MDD and several other disorders such as SUD. In childhood on the other hand, the most common anxiety disorder is separation anxiety disorder (SAD) (Lewinsohn et al., 2008). However findings on the role of SAD in the development of other disorders in adulthood are mixed. In some studies, SAD was reported to increase the risk of other disorders (e.g., panic disorder, MDD) (Bittner et al., 2004; Bruckl et al., 2007; Lewinsohn et al., 2008), whereas other studies failed to replicate this association (Aschenbrand et al., 2003; Haywood et al., 2000; Silove et al., 1996). Furthermore, SAD tends to have a short duration of about 3 years (Lewinsohn et al., 2008). Most SAD symptoms tend to remit by adolescence and only 6% of the children with a SAD before the first interview still had SAD about one year later (Lewinsohn et al., 2008).

When psychosocial outcomes at age 30 were analysed separately by the age at which anxiety disorders begin, anxiety which began in childhood only predicted less years of completed education in adulthood, whereas anxiety which began in adolescence predicted income, unemployment, maladjustment, poor coping skills, chronic stress and higher number of life events at age 30. Why adolescent anxiety, compared to childhood anxiety, was associated with more negative psychosocial outcomes at age 30 was unclear. Speculatively, respondents who experienced their first anxiety disorders during adolescence, compared to during childhood, were more adversely affected by their anxiety because of the additional biological changes at puberty (Hyde et al., 2008). Furthermore, adolescence is a developmental period in which critical life roles and responsibilities (e.g., completing one's

education, obtaining a job, becoming financially independent, developing an adult social support system) are acquired (Rohde et al., 2007). As a consequent of their anxiety disorders, these adolescents may be impaired in various life domains resulting in not being able to acquire the roles that they need for their adult life.

MDD, SUD, and AUD in adulthood partially mediated the effect of adolescent anxiety in some psychosocial outcomes (i.e., unemployment, family support, social/leisure activities, and chronic stress) at age 30. This suggested that, regardless of the presence of psychopathology in adulthood (i.e., MDD, SUD, AUD), adolescent anxiety still had an important effect on these outcomes. MDD, AUD and SUD completely mediated the effect of adolescent anxiety on other forms of psychosocial outcomes (i.e., income, general maladjustment, maladjustment at family, coping skills, and life events) at age 30. This suggested that adolescent anxiety affects these areas through its effects on other forms of psychopathology during adulthood. The present finding further suggested that impairment associated with these psychosocial functioning could be ameliorated by reducing the development of MDD, SUD and AUD among individuals with adolescent anxiety disorders. However, additional strategies are needed to mitigate the persistent influence of adolescent anxiety disorders on these outcomes.

In line with previous studies, child and adolescent anxiety disorders were significantly more common among girls than boys (Essau et al., 2000; Hale et al., 2008; Letcher et al., 2012; Pine et al., 1998; Merikangas et al., 2010; Reinherz et al., 1989). Explanation for this gender difference is unclear and is beyond the scope of the present study. According to some authors, girls tend to be more susceptible to developing anxiety because of genetic predispositions (Silberg et al., 2001). It could also be related to the way in which boys and girls react to biological and social changes at puberty (Cyranowski et al., 2000). As argued by Cyranowski et al. (2000), the need for interpersonal affiliation at puberty is greater in girls than in boys. If these affiliation needs are not met, girls tend to be at an increased risk for anxiety (Hankin et al., 2007). While gender difference in anxiety disorders emerged in childhood and adolescence, studies have also reported a similar result in adulthood (Essau et al., 2004). Thus, answering the question as to the extent to which female gender may constitute a stable vulnerability factor in explaining the persistence of anxiety disorders is a critical gap in our knowledge base. Interestingly, despite this gender difference in the prevalence of anxiety disorders, gender did not moderate the association between anxiety and psychosocial functioning. Thus, the negative impact of anxiety on psychosocial functioning seems to be independent of gender. It could be that, regardless of gender, it is the avoidance behaviour which is the core characteristics of anxiety disorders that cause a wide range of psychosocial impairments (e.g., reduced academic performance and social relationship (Brown et al., 1987; Bifulco et al., 1998).

5. Limitations

The study has some limitations which should be considered. First, although the OADP sample is representative of youth in Oregon, the extent to which our findings are generalizable to other populations is unknown. Second, as the OADP is a 16-year longitudinal study, changes in the diagnostic criteria and assessment approaches used are

inevitable. Specifically, at T1 and T2, the K-SADS and DSM-III-R criteria were used, and at T3 and T4, the SCID and DSM-IV criteria were used. Third, there have been some concerns about the accuracy of retrospective symptom recall (Wells and Horwood, 2004), however, high interrater reliabilities for individual symptoms, found in the OADP data, seem to diminish this concern. Fourth, the age range was limited to age 30 years, such that information for some participants may not be completed. All the information related to the diagnoses, psychosocial outcomes were assessed via self-report. Fifth not all the participants who took part in T1 were invited to participate in T3. Furthermore, at T3, all non-White participants were retained in order to maximize ethnic and racial diversity. While this strategy reduced the number of participants who needed follow up, it helped to maximize the representativeness of the study population. The fact that all adolescents with a history of a depressive or non-mood disorders, and a random sample of adolescents without a history of psychopathology by T2 were invited to participate in a third evaluation may have an impact on our findings. Sixth, psychosocial variables were chosen because they have been shown to be important in the literature. However, it should be noted that some of the psychosocial variables included in the present study seemed to cover overlapping concepts. Finally, this is not a prospective study as the study was carried out when the participants were 14-18 years old at T1. As such the finding cannot inform a causal linkage between childhood or adolescents anxiety disorders and later psychosocial adjustment. Another potential limitation is the absence of control confounding by known risk factors for disorders (e.g., child abuse, family violence) and adult psychosocial outcome.

6. Conclusions

Adolescent anxiety is more important than childhood anxiety in predicting psychosocial outcomes and psychopathology at age 30 years. Adolescent anxiety affects deleterious outcomes in adults both directly and through others forms of psychopathology such as MDD, AUD, and SUD. Thus, prevention and intervention efforts that target MDD, AUD and SUD may be useful for targeting the negative outcomes of adolescent anxiety. A challenge for future research is to develop a better understanding of the process by which adolescent anxiety produces its negative psychosocial impact either directly or through various forms of psychopathology.

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Fig. 1.
Study design.

Table 1

Gender and psychopathology before and after age 19 by childhood and adolescent anxiety disorders groups.

	Childhood anxiety disorder (N=80)	Adolescent anxiety disorder (N=110)
	N (%)	N (%)
Female	62 (77.5)	90 (81.8)
Psychopathology before age 19		
Childhood anxiety	–	58 (52.7)
Adolescent anxiety	58 (72.5)	–
MDD	35 (56.3)	64 (58.2)
SUD	18 (22.5)	33 (30.0)
Alcohol	12 (15.0)	21 (19.1)
Disruptive disorder	10 (12.5)	13 (11.8)
Psychopathology after age 19		
Young adult anxiety	34 (42.5)	66 (60.0)
MDD	52 (65.0)	78 (70.9)
SUD	26 (32.5)	52 (47.3)
Alcohol	24 (30.0)	44 (40.0)

Table 2

Childhood and adolescent anxiety predicting psychosocial outcomes at age 30.

	Childhood anxiety	Adolescent anxiety
	OR (95% CI)	OR (95% CI)
High grade complete ¹	0.74 (0.49–1.13)	0.86 (0.60–1.22)
Recent unemployment ¹	0.84 (0.39–1.82)	2.08 (1.19–3.62)
Annual household income ¹	1.38 (0.81–2.36)	0.55 (0.33–0.94)
Poor physical health ²	1.06 (0.53–2.09)	2.05 (1.14–3.70)
	B (SE)	B (SE)
Family support ³	0.05 (0.04)	–0.11 (0.04)
Friends support ³	0.01 (0.02)	–0.02 (0.04)
Total SAS ⁴	–0.07 (0.05)	0.17 (0.05)
Work SAS ⁴	–0.09 (0.06)	0.27 (0.06)
Social/leisure SAS ⁴	–0.18 (0.07)	0.24 (0.08)
Family SAS ⁴	–0.04 (0.04)	0.19 (0.03)
Marital Role ⁴	–0.12 (0.04)	0.11 (0.03)
Parenting Role ⁴	–0.05 (0.15)	0.09 (0.13)
Family unit ⁴	–0.15 (0.05)	0.22 (0.07)
Suicide Attempt ³	–0.28 (0.77)	0.03 (0.71)
Life satisfaction ³	–0.07 (0.02)	0.10 (0.02)
Coping skills ⁴	0.92 (0.81)	–1.95 (0.86)
Chronic Stress ³	–0.13 (0.07)	0.25 (0.05)
Stressful life events ³	–0.14 (0.05)	0.17 (0.08)

Odds ratios (OR) and 95% confidential intervals (CIs). Regression coefficients (B) and Standard Error (SE). In bold, significant level $p < 0.0025$ (based on Bonferroni's adjustment for multiple tests).

ORs are based on (1) multinomial distribution and cumlogit link and (2); and binomial distribution and logit link. Regression coefficients are based on (3) Poisson distribution and log link; and (4) normal distribution and identify link.

All models were adjusted for gender, the same psychosocial measure at T1 (when available), MDD, AUD, SUD, and disruptive disorder before age 19.

Table 3

Childhood and adolescent anxiety predicting Anxiety, major depressive disorder, alcohol and substance use disorders after age 19.

	Childhood anxiety	Adolescent anxiety
Disorders after age 19:	OR (95% CI)	OR (95% CI)
Anxiety	0.64 (0.34–1.18)	11.63 (4.53–29.86)
MDD	1.00 (0.68–1.48)	1.47 (1.06–2.05)
SUD	0.69 (0.47–1.00)	1.42 (1.24–1.63)
AUD	0.93 (0.74–1.16)	1.29 (1.15–1.43)

Note: MDD=major depressive disorder; AUD=alcohol abuse/dependence; SUD=substance abuse/dependence.

Odds ratios (OR) and 95% confidential intervals (CIs). In bold, significant level $p < 0.0025$ (based on Bonferroni's adjustment for multiple tests). ORs are based on binomial distribution and logit link.

Adjusted for gender, MDD, AUD, SUD and disruptive disorder before age 19.

Table 4

Associations between potential mediators (adult anxiety, SUD and AUD) and psychosocial outcomes at age 30.

	Adult SUD	Adult Alcohol	Adult Anxiety
Outcomes:	OR (CI 95%)	OR (CI 95%)	OR (CI 95%)
Poor health status ¹	1.23 (0.29-5.28)	0.91 (0.24-3.43)	2.05 (1.39-3.03)
	B (SE)	B (SE)	B (SE)
Total SAS ²	0.09 (0.04)	-0.03 (0.04)	0.20 (0.02)
Work SAS ²	0.08 (0.06)	0.02 (0.07)	0.16 (0.03)
Family SAS ²	0.09 (0.07)	-0.08 (0.06)	0.22 (0.06)
Family unit ²	0.29 (0.09)	-0.21 (0.10)	0.30 (0.04)
Life satisfaction ³	0.08 (0.01)	0.08 (0.02)	0.13 (0.03)
Coping skills ²	-1.76 (0.62)	0.03 (0.56)	-3.18 (0.75)
Chronic stress ³	0.27 (0.08)	-0.18 (0.08)	0.30 (0.04)

Odds ratios (OR) and 95% confidential intervals (CIs). Regression coefficients (B) and Standard Error (SE). In bold, significant level $p < 0.05$.

ORs are based on (1) binomial distribution and logit link. Regression coefficients are based on (2) normal distribution and identify link; and (3) Poisson distribution and log link.

Adjusted for gender, the same psychosocial measure at T1 (when available), MDD, AUD, SUD, disruptive disorder before age 19, childhood and adolescent anxiety, and psychopathology after age 19 (MDD, SUD, AUD, and Anxiety).