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The developmental course of loneliness in adolescence: Implications for mental health, educational attainment, and psychosocial functioning

Timothy Matthews[1], **Pamela Qualter**[2], **Bridget T. Bryan**[1], **Avshalom Caspi**[1,3], **Andrea Danese**[1,4], **Terrie E. Moffitt**[1,3], **Candice L. Odgers**[5], **Lily Strange**[1], **Louise Arseneault**[1]

[1]Social, Genetic and Developmental Psychiatry Centre, Institute of Psychiatry, Psychology and Neuroscience, King's College London, London, UK

[2]Manchester Institute of Education, University of Manchester, Manchester, UK

[3]Departments of Psychology and Neuroscience, Psychiatry and Behavioral Sciences, and Institute for Genome Sciences and Policy, Duke University, Durham, NC, USA

[4]National and Specialist CAMHS Trauma and Anxiety Clinic, South London and Maudsley NHS Foundation Trust, London, UK

[5]Sanford School of Public Policy, Duke University and Department of Psychology and Social Behavior, University of California, Irvine, CA, USA

Abstract

The present study examined patterns of stability and change in loneliness across adolescence. Data were drawn from the Environmental Risk (E-Risk) Longitudinal Twin Study, a UK population-representative cohort of 2,232 individuals born in 1994 and 1995. Loneliness was assessed when participants were aged 12 and 18. Loneliness showed modest stability across these ages (r = .25). Behavioral genetic modeling indicated that stability in loneliness was explained largely by genetic influences (66%), while change was explained by nonshared environmental effects (58%). Individuals who reported loneliness at both ages were broadly similar to individuals who only reported it at age 18, with both groups at elevated risk of mental health problems, physical health risk behaviors, and education and employment difficulties. Individuals who were lonely only at age 12 generally fared better; however, they were still more likely to finish school with lower qualifications. Positive family influences in childhood predicted reduced risk of loneliness at age 12, while negative peer experiences increased the risk. Together, the findings show that while early adolescent loneliness does not appear to exert a cumulative burden when it persists, it is nonetheless a risk for a range of concomitant impairments, some of which can endure.

Keywords

adolescence; development; loneliness; mental health

Corresponding author: Louise Arseneault, Louise.Arseneault@kcl.ac.uk.

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Conflicts of interest. None.

Introduction

Human biology is equipped with a variety of adaptations for responding to environmental threats, which can can be beneficial in the short-term but exert a cumulative burden on health if sustained over time. The stress response is a well-known example of such a mechanism (Danese & McEwan, 2012). Loneliness, similarly, has been described as a response to an environmental pressure that serves an adaptive purpose in the short-term, but harmful if prolonged (Cacioppo et al., 2006). According to this model based on evolutionary theory, being embedded in a social group promotes an individual's likelihood of surviving and rearing healthy offspring; hence, a longing for social connection serves an adaptive function. When individuals perceived themselves to be isolated from their social group, the psychological discomfort that arises from this, known as loneliness, motivates them to seek to renew their social connections. When this is achieved successfully, feelings of loneliness should be expected to subside. However, in some cases, loneliness can lead to maladaptive downstream social cognitions and interpersonal behaviors. This can sabotage potentially rewarding interactions and inhibit the formation of close bonds with others, thereby contributing further to feelings of loneliness (Qualter et al., 2015). Under these circumstances, loneliness can form a feedback loop that persists over time. This long-term form of loneliness acts as a chronic stressor that foreshadows negative health outcomes in later life, including an elevated risk for premature mortality (Holt-Lunstad et al., 2015).

Why some individuals become trapped in this "vicious cycle" of loneliness, while others are spared, is complex and likely to be influenced by multiple factors. Although the circumstances individuals find themselves in are an important determinant, loneliness has also been argued to be "trait-like" in nature (Mund et al., 2019) and has been shown to be partially under the influence of genetic factors (Goossens et al., 2015). Heritable characteristics may therefore play a role in the extent to which loneliness remains stable over time, rather than resolving in due course. A twin study of children reported that stability in loneliness across ages 7, 10, and 12 was driven by familial influences, both genetic and shared family environment (Bartels et al., 2008). However, the relative contributions of these influences varied by age, with the genetic component reducing and the shared environment component increasing around the onset of puberty. This reflects the fact that genetic and environmental effects on traits are not static; instead, "new" effects can emerge during certain developmental periods in response to biological or environmental changes (Silberg et al., 2007). This dynamic nature of the etiology of loneliness indicates that further research is required to examine how genetic and environmental effects continue to drive stability in loneliness beyond the childhood years.

Adolescence is a particularly salient period for loneliness: according to UK government statistics, nearly half of 10–12year-olds report feeling lonely at least some of the time, rising to near 60% in 16–24-year-olds, with 10%–15% of both groups reporting that they feel this way often (Office for National Statistics, 2018). Adolescence is a critical period for brain development in regions involved in social processing, corresponding with an increased sensitivity to social rejection (Blakemore & Mills, 2014). This occurs against the backdrop of significant life transitions, such as changing schools, and ultimately leaving school and

preparing for life as an independent adult, which can bring significant upheaval to social networks. Navigating these milestones could lead some individuals to drift in or out of loneliness across the adolescent years, and others to be troubled by loneliness persistently. Hence, the development of loneliness could be a dynamic phenomenon, and there may be distinct developmental profiles with different etiologies, in a similar manner to "early onset," "persistent," and "late-onset" forms of some mental health disorders (Thapar & Riglin, 2020).

Longitudinal studies have yielded broadly consistent findings on developmental patterns of loneliness in childhood and adolescence (e.g., Lay-Yee et al., 2021; Qualter et al., 2013; Schinka et al., 2013; Vanhalst et al., 2013). These studies indicate that both individuals with chronic high levels of loneliness, and those whose loneliness increases from a low baseline level, experience poorer mental health outcomes compared to non-lonely individuals. Beyond mental health, loneliness in young people is concurrently associated with a range of other difficulties, including risky health behaviors, poor sleep quality, low educational attainment, and unemployment (Matthews et al., 2019). Therefore, even if the longer-term physical health outcomes of chronic loneliness may not yet be detectable until later life, patterns of loneliness in adolescence could have implications for a variety of more proximal outcomes.

There are a variety of ways in which the developmental path of loneliness in adolescence could shape poor outcomes. For instance, chronic loneliness could exert a cumulative effect on mental health over time, in a similar manner to the aggregation of other risk factors such as economic deprivation and stressful life events (Evans & Cassells, 2014). Under this scenario, the higher prevalence of mental health problems among lonely individuals could be largely driven by chronically lonely cases. Alternatively, loneliness may simply be a concomitant stressor whose negative effects remain stable for the duration that it is experienced, in which case the mental health profile of "newonset" loneliness would be broadly similar to that of chronic loneliness. This would be in line with the concept of equifinality, whereby individuals arrive at the same outcome from different starting points (Cicchetti & Rogosch, 1996). Meanwhile, individuals who escape loneliness may in doing so escape the worst outcomes, or, alternatively, loneliness experienced in early adolescence that subsequently "remits" could still cast an enduring shadow, either on mental health or other areas of functioning such as sleep problems (Harris et al., 2013).

In addition, the developmental course of loneliness during adolescence could itself be influenced by prior experiences. To the extent that environmental influences play a role in stability and change in loneliness, identifying modifiable aspects of the environment that could change its course would be informative for interventions and preventative measures. Previous research indicates that peer problems such as bullying and social exclusion in childhood foreshadow greater loneliness in adolescence (Matthews et al., 2019, 2020; Yang et al., 2020). On the one hand, this could reflect a chronic trajectory of loneliness that is set in place at the time of the bullying; on the other hand, it could reflect a stable underlying vulnerability to loneliness that endures into later adolescence even if the bullying is no longer ongoing (Matthews et al., 2020). Beyond peer influences, the family home

environment and parenting behaviors may also play a role in shaping loneliness trajectories (Stickley et al., 2016).

The present study investigates patterns of stability and change in loneliness between early and late adolescence, using longitudinal and genetically sensitive approaches. First, we used a twin study design to quantify genetic versus environmental contributions to the persistence of loneliness over time. Second, we created groups reflecting different developmental profiles of loneliness: individuals who experienced loneliness in both early and late adolescence, those whose loneliness was limited to early adolescence, and those who were not lonely in early adolescence but who experienced loneliness later on. Third, we compared mental health and functional outcomes in late adolescence in these three groups, in comparison to individuals who were never lonely in adolescence. Fourth, we examined childhood predictors across these different developmental profiles of loneliness. These analyses provide an insight into how the experience of loneliness impacts on development, with implications for the timing and targeting of interventions.

Method

Participants

Participants were members of the Environmental Risk (E-Risk) Longitudinal Twin Study, which tracks the development of a birth cohort of 2,232 British children. The sample was drawn from a larger birth register of twins born in England and Wales in 1994–1995 (Trouton et al., 2002). Full details about the sample are reported elsewhere (Moffitt & E-Risk Study Team, 2002). Briefly, the E-Risk sample was constructed in 1999–2000, when 1,116 families (93% of those eligible) with same-sex 5-year-old twins participated in home-visit assessments. This sample comprised 56% monozygotic (MZ) and 44% dizygotic (DZ) twin pairs; sex was evenly distributed within zygosity (49% male). 90% of participants were of white ethnicity.

Families were recruited to represent the UK population with newborns in the 1990s, to ensure adequate numbers of children in disadvantaged homes and to avoid an excess of twins born to well-educated women using assisted reproduction. The study sample represents the full range of socioeconomic conditions in Great Britain, as reflected in the families' distribution on a neighborhood-level socioeconomic index (ACORN [A Classification of Residential Neighborhoods], developed by CACI Inc. for commercial use; Odgers, Caspi, Bates, et al., 2012; Odgers, Caspi, Russell, et al., 2012). Specifically, E-Risk families' ACORN distribution matches that of households nationwide: 25.6% of E-Risk families live in "wealthy achiever" neighborhoods compared to 25.3% nationwide; 5.3% versus 11.6% live in "urban prosperity" neighborhoods; 29.6% versus 26.9% live in "comfortably off" neighborhoods; 13.4% versus 13.9% live in "moderate means" neighborhoods, and 26.1% versus 20.7% live in "hard-pressed" neighborhoods. E-Risk under-represents "urban prosperity" neighborhoods because such houses are likely to be childless.

Follow-up home visits were conducted when the children were aged 7 (98% participation), 10 (96%), 12 (96%), and at 18 years (93%). There were 2,066 individuals who participated

in the E-Risk assessments at age 18, and the proportions of MZ (56%) and male same-sex (47%) twins were almost identical to those found in the original sample at age 5. The average age of the twins at the time of the assessment was 18.4 years ($SD = 0.36$); all interviews were conducted after their 18th birthday. There were no differences between those who did and did not take part at age 18 in terms of socioeconomic status (SES) assessed when the cohort was initially defined ($\chi 2_{(2,\ N = 2,232)} = 0.86$, $p = .65$), age-5 IQ scores ($t_{(2,208)} = 0.98$, $p = .33$), or age-5 emotional or behavioral problems ($t_{(2,230)} = 0.40$, $p = .69$ and $t_{(2,230)} = 0.41$, $p = .68$, respectively).

Home visits at ages 5, 7, 10, and 12 years included assessments with participants as well as their mother (or primary caretaker). The home visit at age 18 included interviews only with the participants. The Joint South London and Maudsley and the Institute of Psychiatry Research Ethics Committee approved each phase of the study. Parents gave informed consent and twins gave assent between 5–12 years and then informed consent at age 18.

Measures

Loneliness—A measure of loneliness in early adolescence was derived using three items from the Children's Depression Inventory (Kovacs, 1992), completed when participants were aged 12. Each item was presented as a set of three statements, and participants were instructed to select the statement that described them best: (1) "I do not feel alone," "I feel alone many times" or "I feel alone all the time"; (2) "I have plenty of friends," "I have some friends but I wish I had more" or "I do not have any friends"; and (3) "Nobody really loves me," "I am not sure if anybody loves me," "I am sure that somebody loves me." Items were coded 0–2 and summed to produce a scale from 0 to 6 ($M = .48$, $SD = .86$, $\alpha = .48$). While internal consistency was low, this measure has been shown to perform similarly to more well-validated measures, in terms of its pattern of associations with known correlates of loneliness such as victimization (Matthews et al., 2020). Moreover, although drawn from an instrument designed to assess depression, the constituent particular items are very similar in content to items used in the Children's Loneliness Scale, which is considered the gold standard for assessing loneliness in children and young adolescents (Maes et al., 2017).

Loneliness in late adolescence was assessed at age 18 using four items from the UCLA Loneliness Scale, Version 3 (Russell, 1996): "How often do you feel that you lack companionship?", "How often do you feel left out?", "How often do you feel isolated from others?" and "How often do you feel alone?" A very similar short form of the UCLA scale has previously been developed for use in large-scale surveys, and correlates strongly with the full 20-item version (Hughes et al., 2004). The scale was administered as part of a computer-based self-complete questionnaire. The items were rated "hardly ever" (0), "some of the time" (1), or "often" (2). Items were summed to produce a total loneliness score from 0 to 8 ($M = 1.57$, $SD = 1.94$, $\alpha = .83$).

The correlation between loneliness measured at age 12 and at age 18 was $r = .25$. Individuals were classified as "lonely" at either age if their score on the loneliness scale fell within the top decile; individuals scoring below this threshold were categorized as "nonlonely." Overall, 17.6% of participants were classified as having been lonely at one or both time points. Using these dichotomous measures, four groups were defined based on individuals'

stability or change in loneliness between ages 12 and 18. Individuals who were non-lonely at both ages were categorized as "never lonely" (82.4%; N = 1,642); Individuals who were lonely at age 12 but not at age 18 were categorized as "age 12-limited" (8.5%; N = 170); individuals who were non-lonely at age 12 but lonely at age 18 were categorized as "age 18-limited" (6.8%; N = 135); and individuals who were lonely at both ages were categorized as "recurrent" (2.4%; N = 47).

Age-18 outcomes

Mental health.—Outcomes of loneliness group membership were selected based on the correlates of loneliness in late adolescence documented by previous research in this cohort (Matthews et al., 2019). Mental health outcomes included diagnoses, based on a structured interview, of major depressive disorder, generalized anxiety disorder, conduct disorder, and attention-deficit hyperactivity disorder (ADHD) as defined by the Diagnostic and Statistical Manual for Mental Disorders (DSM; American Psychiatric Association, 1994, 2013). Self-reports of past-year self-harm or suicide attempt were also included as mental health outcomes. Service use was based on self-reports of having seen a GP, psychiatrist, or counselor in the past year for mental health problems.

Lifestyle and health behaviors.—Participants who reported smoking at least one cigarette per day were coded as daily smokers. Sleep quality was assessed via the Pittsburgh Sleep Quality Inventory (Buysse et al., 1989), daily physical activity via the Stanford Brief Activity Survey (Taylor-Piliae et al., 2010), life satisfaction via the Satisfaction With Life Scale (Diener et al., 1985), and technology use via an adapted form of the Compulsive Internet Use Scale (Meerkerk et al., 2009).

Socioeconomic outcomes.—Participants were classified as not in employment, education, or training (NEET) if they reported neither studying nor working at age 18. Participants were considered to have low qualifications if they held none higher than a D grade on the General Certificate of Secondary Education (undertaken by school students in England, Wales, and Northern Ireland at the age of approximately 15–16 years). Participants' criminal offending history was obtained via linkage to the UK Ministry of Justice's Police National Computer.

Childhood predictors

Family influences.—Sibling warmth was assessed via participants' mothers' report at the age-10 home visit. The measure comprised of 6 items, such as "Do both your twins do nice things for each other?" and "Do your twins play and have fun with each other?" Response options were "No" (0), "Sometimes" (1), and "Yes" (2). Items were summed to form a scale from 0 to 12, with higher scores representating higher levels of sibling warmth (Jaffee et al., 2007).

Parental monitoring was assessed at age 12 using a question-aire based on the work of Stattin and Kerr (2000). Participants' mothers were asked 10 questions about how closely they monitored their child's behavior and movements. Items included, "Do you know where [participant] goes during his/her free time?" and "Do you usually know when

[participant] has tests or projects due at school?", with response choices "No, never" (0), "Yes, sometimes" (1), and "Yes, always" (2). Responses were summed to produce a scale from 0 to 20 (Wertz et al., 2016).

After the age 12 home visit, study interviewers completed a set of questions assessing their impressions of the home environment. Happiness in the home was assessed via three questions: "Is this a happy home?", "Do you think the parent is a good parent?", and "Does the parent enjoy parenting?" Items were coded "No" (0), "A little/somewhat" (1), and "Yes" (2), and were summed to produce a scale of happiness in the home.

Peer influences.—Bullying victimization was assessed using a combination of mothers' and participants' reports from multiple assessments across childhood. At the age 7, 10, and 12 assessments, mothers were asked whether their child "has ever been bullied by another child." At the age 12 assessment, participants were asked "Have you ever been bullied by another person?", and follow-up questions to a positive response ascertained at what age the victimization took place. Participants were categorized as having been severely bullied if they had experienced bullying victimization in both primary and secondary school (Fisher et al., 2015).

Social isolation was assessed at ages 5, 7, 10, and 12, using a selection of items from the Children's Behavior Checklist and the corresponding items from the Teacher's Report Form (Achenbach, 1991a, 1991b). The selected items captured signs of social withdrawal and peer rejection, such as "Would rather be alone than with others" and "Not liked by other children," which were coded "Not true" (0), "Somewhat or sometimes true" (1), or "Very true or often true" (2). Mother and teacher reports were combined, and children were categorized as "Low," "Moderate," or "High" in isolation based on their total score (Matthews et al., 2015). Children were considered to have experienced childhood isolation if they had been highly isolated at one or more ages, or moderately isolated at two or more ages.

Correlations between all study variables are presented in Supplementary Table S1.

Data analysis

Biometric analyses—First, to quantify the genetic and environmental influences on loneliness and its stability, we conducted behavioral genetic analyses using the classical twin study method (Rijsdijk & Sham, 2002). Briefly, the similarity between two twins for a given trait is defined as the sum total of additive genetic (A) and common environmental (C) effects. For MZ twins, the effect of A on within-pair similarity is approximately double that of DZ twins, due to their greater genetic similarity. The effect of C on within-pair similarity is assumed to be the same for both MZ and DZ pairs, as both grow up sharing the same home environment. Meanwhile, all differences between MZ twins are attributed to environmental factors unique to individuals (E). Based on this premise, the within-twin pair similarity of MZ versus DZ twins can be compared in order to estimate the relative contributions of A, C, and E to individual differences in phenotypes. Using structural equation modeling in the OpenMx package for R (Boker et al., 2011), a bivariate Cholesky decomposition was fit to the data, partitioning the variance in the age-12 and

age-18 loneliness scales – and the longitudinal association between them – into genetic and environmental explanatory factors.

The Cholesky approach (Figure 1) allows genetic and environmental effects on loneliness at age 12 (A1, C1, and E1) to also explain variance in loneliness at age 18, but not vice versa. Variance not explained by these age-12 effects is explained by "new" genetic and environmental effects specific to age 18 (A2, C2, and E2). The relative contribution of genetic and environmental effects to *stability* in loneliness at ages 12 and 18 is calculated by multiplying the paths connecting them via each of the factors and dividing by the phenotypic correlation (rPh); e.g. for the additive genetic factor: (a11 * a21) / rPh. *Change* in loneliness is reflected in the "new" effects at age 18. The contribution of additive genetic effects to change in loneliness is calculated as: a22 / (a22 + c22 + e22). These relative contributions of genetic and environmental factors reflect why some adolescents escape loneliness (or become lonely when they previously were not), and why some become chronically lonely.

Phenotypic analyses—Logistic regression was used to test whether patterns of loneliness across ages 12–18 were associated with mental health at age 18. First, a binary variable reflecting "ever lonely" versus "never lonely" was entered as the independent variable. In subsequent analyses, the three lonely groups were entered into the model with the contrast group "never lonely" omitted.

Next, to test whether the lonely groups differed from each other in their outcomes, three sets of pairwise comparisons were conducted for all outcome variables. First, the recurrent group was compared to the age 12-limited group, to determine whether loneliness experienced in early adolescence can have lasting implications even if it subsides. Second, the recurrent group was compared to the age 18-limited, to determine whether recurrent loneliness has a cumulative effect on outcomes, or if instead the magnitude of effect is constant across both groups. Third, the age 12-limited group was compared to the age 18-limited, to determine whether the timing of loneliness is relevant to any outcomes (for instance, if loneliness experienced in early adolescence has specific implications that loneliness limited to late adolescence does not).

To test whether individuals who experienced loneliness differed in their childhood histories from individuals who did not, the variable reflecting "ever lonely" versus "never lonely" was regressed on each of the childhood variables in logistic regression models. This was then repeated using the three other pairwise group variables as the dependent variable (recurrent versus age 12-limited, recurrent versus age 18-limited, and age 12-limited versus age 18-limited).

Statistical adjustments—All regression analyses were conducted in Stata version 16 (StataCorp, 2019). Sex and SES were adjusted for in each regression model. Participants in this study were pairs of same-sex twins, and therefore each family contained data for two individuals, resulting in nonindependent observations. To correct for this, the phenotypic analyses used the Huber−White or sandwich estimator (Williams, 2000), which adjusts the estimated standard errors to account for the dependence in the data. Further, due to the

large number of tests conducted, a significance level of .01 was chosen, and 99% confidence intervals are reported.

Open science—The premise and data analysis plan for this project were preregistered online at https://sites.google.com/site/moffittcaspiprojects/home/concept-paper_2020/matthews_2020b. Participant early parenthood had been preselected as an outcome variable; however, it was uncommon in this sample ($N = 41$) and when crosstabulated with the loneliness groups, some cells contained as few as one participant. For that reason, this variable was excluded from the analyses.

Results

Biometric analyses

The behavioral genetic analyses indicated both genetic and environmental contributions to loneliness. The full "ACE" model did not show significant difference in fit compared to the fully saturated model ($\Delta -2LL = 19.83$, $\Delta df = 15$, $p = .18$). The parameter estimates for the shared environment (C) factors were near zero, and could be dropped from the model without significant loss of fit ($\Delta -2LL = .10$, $\Delta df = 3$, $p = .99$). The results for the AE model are therefore presented (Figure 2). The model estimates indicated that genetic influences accounted for 25% of the variance in loneliness at age 12, with the remainder explained by nonshared environmental influences. At age 18, the total variance explained by genetic influences was 41%. 11% of variance was explained by genetic factors shared with age-12 loneliness, with the remaining 30% explained by "new" genetic influences unique to age 18. The environmental influences on loneliness at age 18 were largely specific to that age: only 1% of variance was explained by environmental effects shared with age-12 loneliness. Overall, genetic influences accounted for 66% of the stability in loneliness. Change in loneliness, by contrast, was largely explained by nonshared environment (58%).

Phenotypic analyses

Individuals who reported being lonely at either age fared worse at age 18 compared to those who had never been lonely (Table 1). Relative to never-lonely individuals, recurrently lonely individuals had elevated risk for all outcomes, with the exception of criminal offending. Individuals with age 18-limited loneliness did not differ in their qualifications compared to the never-lonely group, nor were they more likely to smoke daily, but they were at increased risk for all other outcomes (except, again, criminal offending). Those whose loneliness was limited to age 12 remained at increased risk for ADHD, self-harm, low life satisfaction, problematic technology use, and low qualifications, but not for other outcomes, including service use. Effect sizes for significant odds ratios varied in magnitude, with the strongest effects observed in the recurrent and age 18-limited groups (Cohen's d = .56–1.29 and .40–1.00, respectively), and small to moderate effect sizes in the age 12-limited group (Cohen's d = .19–.50). To ensure that the findings were not a product of an arbitrary cutoff score to define the groups, sensitivity analyses using alternative 25% and 5% cutoffs to differentiate lonely from non-lonely individuals showed very similar results (Supplementary Tables S2 and S3).

Pairwise group comparisons indicated differential outcomes of loneliness developmental profiles. Recurrently lonely individuals did not differ from those with age 18-limited loneliness, with the exception of having low qualifications (Cohen's d = .73), indicating that most outcomes of recurrent loneliness were driven largely by the fact that loneliness in late adolescence was assessed concurrently to the mental health outcomes (Table 2). Both the recurrent and age 18-limited groups had greater risk of depression, anxiety, self-harm, poor sleep quality, low life satisfaction, and problematic technology use at age 18, when compared to individuals whose loneliness was limited to age 12 (Cohen's d = .44–1.08). Individuals with age 12-limited loneliness had lower risk of most negative outcomes compared to the age 18-limited group; however, they were more likely to have low educational qualifications (Cohen's d = .38), indicating that loneliness experienced at the age of entry to secondary school could have specific implications for this particular outcome.

We conducted further comparisons between the groups examining childhood predictors. Greater sibling warmth, parental monitoring, and happiness in the home predicted a slightly reduced risk for ever having been lonely (Cohen's d =−.06 to −.12). However, bullying victimization and social isolation were associated with increased risk for ever being lonely (Table 3). The strongest effect size was that of bullying victimization (Cohen's d = .71). With the exception of sibling warmth, these factors also reduced the risk of being recurrently lonely compared to being lonely only at age 18. However, none of these factors differentiated recurrent loneliness from age 12-limited loneliness.

Discussion

Loneliness can often be a temporary feeling that individuals escape as their circumstances become more favorable. While this does not diminish the distress experienced at the time, of greater concern is the potential for loneliness to become a chronic problem that follows people through life and imposes long-term distress. The present study shows that although loneliness is associated with negative outcomes in mental health and functioning, experiencing it recurrently during adolescence does not necessarily translate into a cumulatively higher burden. However, loneliness experienced in early adolescence could have lasting implications for outcomes in later years, regardless of whether it recurs or persists over time. The findings also indicate that factors in the family home and peer relationships are relevant to children's vulnerability to becoming lonely. Moreover, the findings indicate that environmental factors play a greater role in explaining why individuals increase or decrease in loneliness, while genetic factors largely explain why individuals remain lonely (or non-lonely).

Loneliness was observed to be dynamic: only a minority of children fell into the "recurrent" category. Previous research supports the notion that chronic loneliness is less common than other developmental patterns, although estimates of its prevalence have ranged from below 5% (Schinka et al., 2013; Vanhalst et al., 2013) to above 20% (Qualter et al., 2013). This may partly be explained by the fact that studies have varied in terms of the measures used, the time lag between follow-ups, and the analytical approach. A goal of future research should be to establish well-replicated standards for categorizing chronic versus transient

forms of loneliness. In the present study, we present a method that we argue is an informed and plausible one, but this approach should be interrogated further in other samples and age groups.

Both chronic and transient forms of loneliness have been shown to be associated with poor health outcomes (Martín-María et al., 2020; Shiovitz-Ezra & Ayalon, 2010; Zhong et al., 2016). In the present study, individuals whose loneliness was confined to early adolescence still had poorer mental health at age 18 compared to those who were never lonely, albeit not to the extent that they made more use of mental health services. In comparison to the age 12-limited group, adolescents who were recurrently lonely appeared to be markedly worse off for certain mental health outcomes, such as depression and anxiety. However, this was largely due to loneliness being ongoing at the time mental health was assessed, as these recurrently lonely individuals did not differ significantly on any mental health outcome from those with age 18-limited loneliness. As such, the results do not suggest a cumulative association between recurrent loneliness and risk for clinically significant mental health outcomes; instead they reflect that this risk frequently co-occurs with loneliness, and in some cases can continue to be observed later in adolescence even if the loneliness is no longer ongoing. Therefore, while individuals who are able to escape loneliness are spared the worst outcomes, transient loneliness in early adolescence can nonetheless signal a lasting risk for mental health problems. However, it should also be noted that psychopathology and other difficulties could themselves be antecedents of loneliness, and these data do not allow for assumptions about the directionality of effects.

Although recurrent and age 18-limited loneliness were broadly similar in terms of outcomes, one exception to this was that the recurrent group was more likely to have lower qualifications. Moreover, individuals who experienced loneliness in early adolescence, even if they did not remain lonely in later adolescence, still had lower educational attainment by school-leaving age compared to those whose loneliness had emerged later. A possible interpretation is that loneliness could have a disruptive effect on education (Benner, 2011; Osterman, 2000), to the extent that those affected are unable to recover lost ground even if their loneliness reduces. This would have a number of implications. First, the association between adolescent loneliness and low qualifications could be set in place at the very beginning of high school. Second, it lends support to the hypothesis that loneliness could act as a force for downward social mobility (Matthews et al., 2019). Third, it attests to the importance of early intervention, not just to tackle loneliness but also to support affected students and ensure they do not fall behind.

Aside from this particular finding, the lack of statistically significant group differences between recurrent and age 18-limited loneliness indicates that, phenotypically, these two profiles of loneliness look very similar in adolescence – both are associated with increased risk of mental health problems, and both are foreshadowed by similar family and peer influences. The behavioral genetic findings show that at the etiological level, different influences are at play with regard to stability versus remission of loneliness. Most of the genetic influences on loneliness were specific to the age at which they were assessed; few genetic influences were shared between age-12 and age-18 loneliness. However, those genetic influences that were common to both ages explained much of the phenotypic

continuity of this phenomenon, while change in loneliness was explained more by the environment. This is consistent with patterns observed for a range of emotional and behavioral problems in childhood and adolescence (Hannigan et al., 2017).

Given that the stability of loneliness was modest, there is ample scope for interventions to induce change. Identifying specific environmental factors that contribute to change in loneliness would therefore be useful for improving outcomes. In the present study, we examined the role of family and peer influences in predicting the developmental course of loneliness. Similar to the pattern observed in the age-18 outcomes, recurrently lonely individuals did not differ substantially from the age 12-limited individuals in terms of their profile of childhood predictors. Instead, individuals who were lonely early in adolescence – regardless of whether their loneliness became recurrent – were more likely to have been bullied and socially isolated, and to have an unhappier home environment, compared to individuals whose loneliness emerged later in adolescence. Recurrently lonely individuals were further differentiated from the age 18-limited group by lower levels of parental monitoring and happiness in the home during childhood, suggesting a potential role of the family home environment in preventing the emergence and maintenance of loneliness at this age.

Limitations

A limitation of this study is that loneliness was assessed only at two time points, with a 6-year time lag. Consequently, more finegrained changes in loneliness across this interval could not be discerned. For the same reason, it was not possible to derive trajectories of loneliness using more sophisticated analytical techniques, such as growth mixture modeling or latent class growth analysis. Nonetheless, the four-group taxonomy used in this study is consistent with trajectories of child and adolescent loneliness that have been identified using such analytical approaches (van Dulmen & Goossens, 2013).

The threshold at which individuals are deemed to be lonely versus non-lonely is an arbitrary one, and there is as yet no consensus on how best to do this. For that reason, we treat loneliness as a continuum wherever possible. However, in order to construct the groups of loneliness profiles, it was necessary to make a decision about who should be deemed lonely at each age. The choice of a 10% cutoff is an informed one, as this is similar to the number of adolescents who report feeling lonely often (Office for National Statistics, 2018). Moreover, the sensitivity analyses indicated that findings remained fairly consistent regardless of whether the choice of cutoff is made more or less conservative. Nonetheless, the “extreme group” approach has some recognized statistical limitations (Preacher et al., 2005).

A related issue is that there is no consensus in the literature on how “recurrent,” “chronic,” or “persistent” loneliness should be defined. A chapter in a seminal text on loneliness (Young, 1982) suggests that persistence of 2 years or more merits the definition of “chronic.” This is similar to the criteria by which “persistent” depression is defined in the DSM (American Psychiatric Association, 1994, 2013). There are some limitations to identifying a group who reported being lonely at two consecutive developmental periods. First, it assumes that these individuals’ loneliness was stable for the entire 6-year duration

between assessments, when in fact it might have waxed and waned. Hence, this group is referred to as “recurrent” loneliness rather than “chronic.” Nonetheless, if loneliness is present both at age 12 and at age 18, this would suggest some stable underlying vulnerability, and the behavioral genetic findings attest to this. Second, however, individuals who were lonely for several consecutive years but who were no longer lonely by the age of 18 would not be counted, and therefore the prevalence of chronic loneliness may be underestimated here.

Implications

While the results do not support a cumulative association between recurrent loneliness and severity of mental health outcomes in young people, this does not detract from the importance of preventing loneliness from becoming a chronic problem. Clearly, an experience that is both distressing in its own right and goes hand in hand with pervasive poor functioning is something that should be prevented or tackled in a timely manner. In addition, the results indicate that lonely young adolescents remain at enduring risk for specific deficits in later adolescence regardless of whether they are still lonely by that time, suggesting that ongoing support is warranted even for those who have been able to escape loneliness. Moreover, loneliness is implicated in physical health problems and early mortality in later life (Holt-Lunstad et al., 2015), further attesting to the importance of interventions to break the cycle of loneliness during the early years.

Genetic influences on loneliness and its stability do not imply determinism. Environments can be modified in such a way that children with a genetic risk for loneliness nonetheless have a better chance of evading it. However, more research is required to pin-point which environmental interventions are most effective. The present results indicate that the childhood factors considered in this study do not have scope to prevent loneliness from emerging in the future, or to prevent childhood loneliness becoming chronic. They do, however, have some potential to prevent loneliness from emerging in early adolescence (and then, possibly, persisting over time). Other research has shown that child characteristics such as low trust and self-worth, and negative temperamental reactivity are associated with a chronic trajectory of loneliness in adolescence (Qualter et al., 2013); these too could be viable targets for intervention.

Our findings highlight the importance of developing interventions for loneliness among young people. Given that even loneliness that is limited to early adolescence predicts lower academic achievement, schools could play a central role in developing interventions for youth. Indeed, recent work suggests that schools can successfully provide universal intervention that helps young people manage and overcome loneliness, improving academic performance. Specifically, that work demonstrates the importance of (1) developing knowledge about formal and informal help available in and outside school in relation to loneliness (Lasgaard et al., in press) so that youth can access sources of support when and if they need them, and (2) social and emotional skills development in schools, supported by the first recent RCT of a loneliness intervention in schools (Hennessey et al., 2021) showing that lessons focused on making and sustaining friends, dealing with conflicts and resolving

problems, understanding emotions in oneself and others, and regulating and managing emotions reduced loneliness among youth.

Conclusion

Encouragingly, most individuals who feel lonely in early adolescence do not continue to experience these feelings by the end of adolescence. Moreover, for those who do remain trapped in loneliness, this does not appear to correspond with a cumulative "load" on negative outcomes. However, there are important caveats to this. First, regardless of the developmental period in which it is experienced, and regardless of whether it is time-limited, loneliness signals risk for a range of concurrent difficulties, including mental health problems, lifestyle-related health risks, and difficulties in education and employment. Second, even those individuals who escape loneliness by late adolescence appear to have an enduring risk for certain outcomes, with lower educational attainment being particularly salient. Moreover, being spared loneliness early in adolescence is no guarantee that it cannot be experienced later, or that it is any less of a burden. Therefore, these findings attest to the importance of preventing loneliness – particularly the frequent, pervasive, and debilitating form of it that affects a minority of young people (Matthews et al., 2019; Office for National Statistics, 2018) – from emerging.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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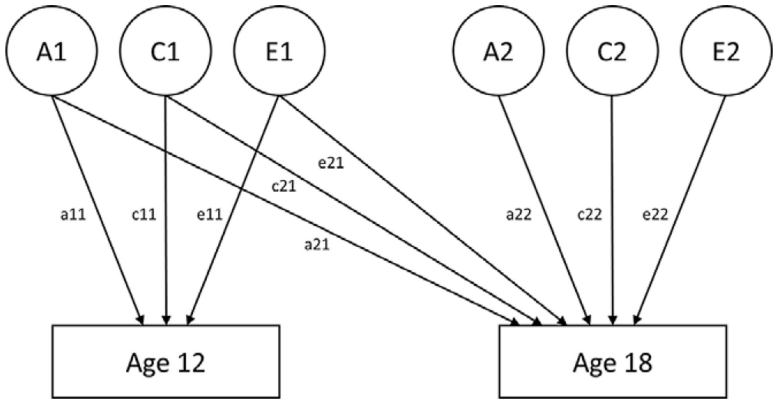

Figure 1.

Bivariate Cholesky decomposition. Due to the temporal ordering of the variables, genetic and environmental effects on age 12 loneliness are also allowed to explain variance in age 18 loneliness, but not vice versa. Paths a11, c11, and e11 reflect the genetic and environmental effects specific to loneliness at age 12. Paths a21, c21, and e21 reflect genetic effects on age 12 loneliness that are also shared by age 18 loneliness. Paths a22, c22, and e22 reflect genetic and environmental effects unique to age 18 loneliness.

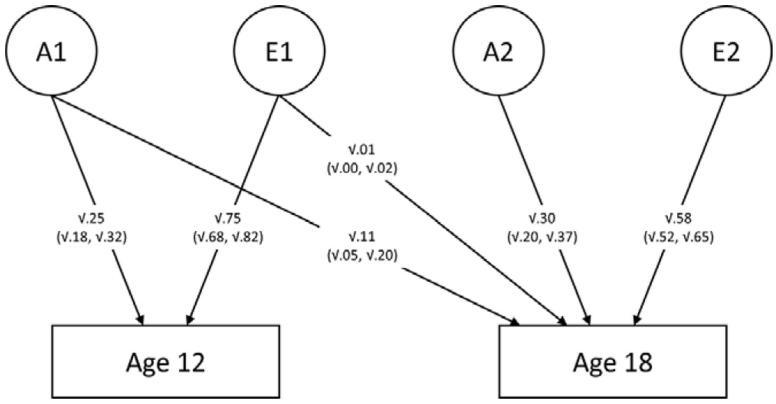

Figure 2.

Bivariate AE model of loneliness at age 12 and age 18. Path estimates reflect the proportion of variance explained by the A and E factors. The contribution of A to stability in loneliness is calculated as the product of paths a11 and a21, divided by the phenotypic correlation: (✓.25 * ✓.11) / .25 = 66%. Similarly, for E: (✓.75 * ✓.01) / .25 = 34%. The contribution of genetic factors to change in loneliness is calculated as path a22 divided by the sum of paths a22 and e22: ✓.30 / (✓.30 + ✓.58) = 42%.. Similarly, for E: ✓.58 / (✓.30 + ✓.58) = 58%

Table 1.

Associations between loneliness group membership and age-18 outcomes

		Developmental course of loneliness			
	***N* (%) or Mean (*SD*)**	**Ever versus never lonely**	**Age 12-limited versus never lonely**	**Age 18-limited versus never lonely**	**Recurrent versus never lonely**
Mental health					
		OR (99% CI)	OR (99% CI)	OR (99% CI)	OR (99% CI)
Depression	398 (20.2%)	**3.50 (2.45, 5.00)**	1.63 (.97, 2.74)	**6.18 (3.73, 10.23)**	**7.11 (3.12, 16.24)**
Anxiety	145 (7.4%)	**4.04 (2.49, 6.56)**	1.48 (.64, 3.39)	**5.95 (3.24, 10.95)**	**10.46 (4.51, 24.25)**
Conduct disorder	300 (15.2%)	**2.05 (1.37, 3.08)**	1.42 (.79, 2.55)	**2.98 (1.69, 5.24)**	**2.29 (.88, 5.96)**
ADHD	152 (7.7%)	**3.03 (1.90, 4.84)**	**2.48 (1.31, 4.70)**	**2.96 (1.53, 5.71)**	**5.62 (2.25, 14.06)**
Self-harm	273 (13.9%)	**3.71 (2.51, 5.50)**	**2.18 (1.27, 3.76)**	**4.79 (2.73, 8.41)**	**8.34 (3.76, 18.49)**
Service use	255 (13.0%)	**2.32 (1.54, 3.48)**	1.14 (.59, 2.18)	**3.95 (2.31, 6.73)**	**3.02 (1.25, 7.30)**
Lifestyle and health behaviors					
		B (99% CI)	B (99% CI)	B (99% CI)	B (99% CI)
Poor sleep quality	5.39 (3.16)	**1.31 (.78, 1.84)**	.45 (−.20, 1.10)	**1.87 (1.07, 2.67)**	**2.75 (1.43, 4.08)**
Physical activity	2.78 (1.06)	**−.19 (−.35, −.02)**	−.11 (−.35, .13)	−.20 (−.45, .04)	**−.40 (−.76, −.06)**
		OR (99% CI)	OR (99% CI)	OR (99% CI)	OR (99% CI)
Daily smoking	435 (22.1%)	**1.55 (1.06, 2.27)**	1.35 (.81, 2.22)	1.63 (.93, 2.87)	2.16 (.87, 5.40)
		B (99% CI)	B (99% CI)	B (99% CI)	B (99% CI)
Life satisfaction	3.87 (.73)	**−.56 (−.70, −.43)**	**−.27 (−.44, −.10)**	**−.82 (−1.00, −.65)**	**−.86 (−1.15, −.57)**
Problematic technology use	4.54 (3.91)	**2.05 (1.33, 2.77)**	**.99 (.15, 1.84)**	**2.64 (1.53, 3.75)**	**4.09 (2.02, 6.16)**
Socioeconomic outcomes					
		OR (99% CI)	OR (99% CI)	OR (99% CI)	OR (99% CI)
NEET	216 (11.0%)	**1.81 (1.15, 2.84)**	1.40 (.74, 2.62)	**2.07 (1.09, 3.92)**	**2.76 (1.01, 7.54)**
Low qualifications	417 (21.2%)	**1.78 (1.22, 2.59)**	**2.11 (1.29, 3.47)**	1.06 (.56, 1.98)	**3.52 (1.51, 8.22)**
Criminal record	202 (10.3%)	1.07 (.54, 1.78)	.99 (.49, 2.02)	1.30 (.63, 2.68)	.68 (.14, 3.32)

Note. B = unstandardized regression coefficient; CI = confidence interval; *N*= number; OR = odds ratio; *SD*= standard deviation. Analyses are restricted to cases with complete data for all variables (*N*= 1,968). Numbers in bold indicate significant associations at $p < .01$.

Table 2.

Pairwise comparisons between loneliness groups for age-18 outcomes

	Developmental course of loneliness		
	Recurrent v age 12-limited (*N* = 212)	**Recurrent v age 18-limited (*N* = 179)**	**Age 12-limited v age 18-limited (*N* = 297)**
Mental health			
	OR (99% CI)	OR (99% CI)	OR (99% CI)
Depression	**4.13 (1.67, 10.22)**	1.12 (.46, 2.72)	**.27 (.14, .51)**
Anxiety	**7.05 (2.33, 21.34)**	1.68 (.64, 4.40)	**.24 (.09, .62)**
Conduct disorder	1.51 (.51, 4.47)	.76 (.26, 2.19)	.49 (.23, 1.04)
ADHD	2.10 (.71, 6.27)	2.11 (.71, 6.24)	.84 (.36, 1.98)
Self-harm	**3.65 (1.48, 9.00)**	1.75 (.69, 4.40)	**.45 (.22, .91)**
Service use	2.55 (.90, 7.25)	.76 (.29, 2.03)	**.29 (.13, .63)**
Lifestyle and health behaviors			
	B (99% CI)	B (99% CI)	B (99% CI)
Poor sleep quality	**2.23 (.82, 3.63)**	.79 (−.67, 2.26)	**−1.43 (−2.44, −.41)**
Physical activity	−.32 (.73, .09)	−.19 (−.62, .23)	.09 (−.25, .43)
	OR (99% CI)	OR (99% CI)	OR (99% CI)
Daily smoking	1.41 (.55, 3.57)	1.28 (.46, 3.61)	.85 (.41, 1.75)
	B (99% CI)	B (99% CI)	B (99% CI)
Life satisfaction	**−.58 (−.92, −.25)**	−.02 (−.36, .32)	**.55 (.31, .78)**
Problematic technology use	**3.11 (.94, 5.28)**	1.45 (−.83, 3.74)	**−1.62 (−2.99, −.25)**
Socioeconomic outcomes			
	OR (99% CI)	OR (99% CI)	OR (99% CI)
NEET	2.05 (.60, 6.95)	1.36 (.43, 4.32)	.67 (.28, 1.60)
Low qualifications	1.64 (.63, 4.23)	**3.76 (1.23, 11.45)**	**1.98 (.94, 4.20)**
Criminal record	.66 (.11, 3.91)	.53 (.10, 2.85)	.76 (.30, 1.98)

Note. B = unstandardized regression coefficient; CI = confidence interval; OR = odds ratio; Analyses are restricted to cases with complete data for all variables (*N* = 1,968). Column headings report the pairwise *N* for each set of comparisons. Numbers in bold indicate significant associations at $p < .01$.

Table 3.

Childhood predictors of loneliness group membership in adolescence

	Developmental course of loneliness			
	Ever v never lonely (*N* = 1,910)	**Recurrent v age 12-limited (*N* = 204)**	**Recurrent v age 18-limited (*N* = 176)**	**Age 12-limited v age 18-limited (*N* = 290)**
	OR (99% CI)	OR (99% CI)	OR (99% CI)	OR (99% CI)
Family influences				
Sibling warmth	**.90 (.83, .98)**	1.04 (.84, 1.28)	.99 (.78, 1.26)	.96 (.82, 1.12)
Parental monitoring	**.89 (.85, .94)**	.90 (.78, 1.04)	**.86 (.75, .99)**	.95 (.85, 1.05)
Happy home	**.81 (.73, .91)**	.92 (.73, 1.15)	**.73 (.56, .97)**	**.82 (.67, 1.00)**
Peer influences				
Bullying	**3.60 (2.21, 5.86)**	1.61 (.66, 3.88)	**3.72 (1.31, 10.59)**	**2.35 (1.02, 5.40)**
Social isolation	**1.42 (1.25, 1.60)**	1.17 (.94, 1.45)	**1.49 (1.14, 1.95)**	**1.28 (1.03, 1.59)**

Note. CI = confidence interval; OR = odds ratio. Analyses are restricted to cases with complete data for all variables (*N* = 1,910). Column headings report the pairwise *N* for each set of comparisons. Numbers in bold indicate significant associations at $p < .01$.