



Research paper

Childhood social adversity and risk of depressive symptoms in adolescence in a US national sample



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ABSTRACT

Objective: Childhood social adversity has been associated with an increased risk of depression and other psychiatric disorders in adolescence and early adulthood. However, the role of timing and accumulation of adversities has not yet been established in longitudinal studies. We examined the association between childhood adversities and adolescent depressive symptoms, and the impact of timing and accumulation of adversity.

Method: Longitudinal data were obtained from the Child Development Supplement to the Panel Study of Income Dynamics (n=2223), a nationally representative survey of US families that incorporates data from parents and their children. Negative binomial regression analysis was used to estimate effects of childhood social adversity on adolescent depressive symptoms, presented as Incidence Rate Ratios with 95% confidence intervals.

Results: Children exposed to social adversity reported higher levels of adolescent depressive symptoms captured by two depression scales. Single-parent household and residential instability were particularly associated with depressive symptoms. A positive relationship was found between cumulative adversity and the risk of adolescent depression. The timing of exposure appeared to have little effect on the risk of adolescent depressive symptoms.

Limitations: The structure of the data implies that alternative causal pathways cannot be fully discounted. The self- or parent-reported data is subject to recall bias.

Conclusion: Our findings support the long-term negative impact of childhood adversity on adolescent depressive symptoms, regardless of when in childhood the adversity occurs. Policies and interventions to reduce adolescent depressive symptoms need to consider the social background of the family as an important risk or protective factor.

1. Introduction

Depression is a common and potentially debilitating disorder occurring through the life-course (Fleisher and Katz, 2001; Patel et al., 2007; Saluja et al., 2004). The first onset of depression often occurs in childhood or adolescence, although treatment typically does not occur until later in life (Birmaher et al., 1996; Costello et al., 2006; Kessler et al., 2007; Patel et al., 2007). Furthermore, depression in childhood and adolescence is a risk factor for adult depression (Dunn and Goodyer, 2006; Melvin et al., 2013; Patel et al., 2007; Pine et al., 1999). According to the National Institute of Mental Health, depression is the most common mental health disorder among adolescents in

the United States, affecting around 11% of the US population aged 12–17 (National Institute of Mental Health, 2014). An even higher proportion of adolescents in the US, nearly 20%, are reported to have symptoms within the broader phenotypic spectrum of depression i.e. ranging from clinically overt depression to non-specific depressive symptoms (Saluja et al., 2004).

Most likely, depression is caused by a combination of genetic, environmental, as well as social and psychological factors (Birmaher et al., 1996; Heim and Binder, 2012; Merikangas et al., 2009; Patel et al., 2007; Schaffer and Kipp, 2014). Studies have consistently shown that depression and depressive symptoms occur more frequently among persons in socioeconomically disadvantaged groups (Gilman

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et al., 2002; Reiss, 2013; Torikka et al., 2014; Tracy et al., 2008), and that the socioeconomic gradient in depression is evident already in childhood (Gilman et al., 2002).

Accumulating evidence suggests that childhood social adversity, commonly captured by parental income, education and occupation, has a substantial influence on the occurrence of depression and depressive symptoms in childhood and adolescence (Amone-P'Olak et al., 2009; Butler, 2014; Goodyer, 2002; Hyland et al., 2016; Mossakowski, 2015).

Various pathways through which childhood social adversity influences child and youth depression have been discussed, including both psychosocial and biological mechanisms (Conger and Donnellan, 2007; Hudson, 2005; Swartz et al., 2016). Family socioeconomic factors may be particularly important for mental health in children and adolescents because of the decisive influence the family has upon children during the period they develop and unfold their personality (Amone-P'Olak et al., 2009). For example, childhood social adversity may affect children's mental health through its negative effect on parents, in terms of increase stress levels, parental marital conflict, and lower parenting quality (Conger and Donnellan, 2007). Biological explanations suggest that social adversity contributes to stress-induced brain dysfunction that in turn may lead to mental health problems (Schaffer and Kipp, 2014; Swartz et al., 2016).

Yet, few previous studies have examined a broad range of childhood adversities and their timing in relation to the risk of adolescent depression (Andersen and Teicher, 2008; Dunn et al., 2013; Heim and Binder, 2012; Kaplow and Widom, 2007). Studies examining the role of timing of adversity exposure have typically focused on childhood maltreatment, showing that, the earlier the exposure, the worse the outcome (Dunn et al., 2013; Heim and Binder, 2012; Kaplow and Widom, 2007). Studies investigating the effect of other adversities, and cumulative adversity are rare (Björkenstam et al., 2016). Furthermore, longitudinal studies with formally representative samples examining these associations are scarce (Mossakowski, 2015; Reiss, 2013). This is unfortunate since longitudinal studies offer a framework for studying long-term health effects of exposures during gestation, childhood, adolescence and later adult life, emphasizing the importance of time and timing in understanding the links between exposure and outcome (Lynch and Smith, 2005).

We address these limitations using a nationally representative panel survey of the US population. We examine five different childhood social adversities (single-parent household, poverty, household receiving public assistance, long-term parental unemployment, and residential instability) and their association with adolescent depressive symptoms. The adversities chosen were based on prior research that has shown them to have significant adverse health or social implications (Duncan and Brooks-Gunn, 1997; Gilman et al., 2003; Najman et al., 2010; Wadsworth and Butterworth, 2006; Weitoft et al., 2003). Our research questions were:

1. Is there an association between a range of childhood social adversities and adolescent depressive symptoms?
2. Does the association between adversities and adolescent depressive symptoms differ by age at exposure to childhood social adversity?
3. Does cumulative exposure to childhood social adversity increase the risk for adolescent depressive symptoms?

2. Methods

2.1. Study population

We used data from the three waves of the Child Development Supplement (CDS-I through CDS-III) survey in years 1997, 2002 and 2007, respectively, which have been part of the Panel Study of Income Dynamics (PSID) in the US. The PSID is a longitudinal study that began in 1968 with a nationally representative sample of about 5000 families in the US, with an oversample of African American and low-

income families (McGonagle et al., 2012). The household heads (defined by PSID as the person, at least 16 years old, with the most financial responsibility in the household) were re-interviewed annually from 1969 to 1997 and every other year thereafter.

In 1997, the PSID began collecting additional data on families that had children under the age of 13, known as the CDS survey. All PSID families with a child aged 0–12 in calendar year 1997 were invited to participate and up to two children were chosen at random from each eligible family. Data were collected for 3563 children residing in 2380 households that constitute our study population (CDS-I). Study participants and their families were re-interviewed in 2002–2003 (CDS-II) and 2007–2008 (CDS-III), until they reached the age of 18 years. Parental reports were collected from the child's primary caregiver, who had to have been living with the child. In over 90% of cases, this is the child's biological mother. The children were interviewed from age 3.

Of the 3563 children included in CDS in 1997, we excluded those born after 1995 (n=588), as they were too young to be eligible to respond to the adolescent depression measures at follow-up. In addition we excluded those for whom we did not have information about adolescent depressive symptoms (n=752). Thus, our final analytical sample included 2223 children born between 1985 and 1995. Of these individuals, 96% were re-interviewed during 2002–2003 (CDS-II), and 58% during 2007–2008 (CDS-III).

2.2. Measures

2.2.1. Childhood social adversity

Information on childhood social adversity when the children were between the ages of 0 and 12 years was obtained from the original PSID studies. Five adversities chosen because of their association with poor mental health in prior research (Duncan and Brooks-Gunn, 1997; Gilman et al., 2003; Najman et al., 2010; Wadsworth and Butterworth, 2006; Weitoft et al., 2003) were included in the analyses. Based on the child developmental stage theories (Schaffer and Kipp, 2014), and given the size of the sample, we chose two exposure periods: 0–6.9 years (infancy and early childhood), and 7–12 years (middle childhood).

2.2.1.1. Single-parent household. In each PSID wave, the family head was asked whether or not she/he lived with a partner. If the respondent was a primary caregiver with no husband/wife/cohabiter, this indicator was coded as 1.

2.2.1.2. Poverty. The PSID consistently collects data on all income sources. A common practice to capture poverty is to use a size-adjusted measure of family income, typically the “income-to-needs-ratio”. This measure is obtained by dividing total household income by the official US poverty threshold corresponding to the size of the given household (Duncan and Brooks-Gunn, 1997; Institute for Research on Poverty, 2016). The conventional income-to-needs ratio is based only on wages and salaries and therefore a ratio of 1.00 in PSID can result in an underestimate of the number of poor households (Stevens, 1994). Therefore we used a higher poverty threshold of 1.25 and below to indicate a household in poverty.

2.2.1.3. Long-term household public assistance reciprocity. A child was classified as a recipient if her/his family had received public assistance, including food stamps, AFDC/TANF, Supplementary Security Income (SSI), Social Security Income or other welfare for at least 6 months in the preceding year.

2.2.1.4. Long-term parental unemployment. In the PSID study, parents were asked annually if they had been unemployed and, if so, for how many weeks. Unemployment was defined as a spell of at least six months in the past year.

2.2.1.5. Residential instability. Based on the number of family moves since birth that were assessed by questions in the PSID study, we defined this indicator as three or more family moves. Prior research guided us to choose this threshold (Jelleyman et al., 2008).

2.2.2. Adolescent depressive symptoms

Follow-up data on adolescent depressive symptoms were obtained from the three CDS survey waves, according to birth year of the study participants: The CDS-I was used for those born in 1987; CDS-II for those born 1987–1990, and CDS-III for those born 1990–1995. Thus, adolescent depressive symptoms were assessed when all study participants were 12–17 years old.

Adolescent depressive symptoms were measured in two ways. The first measure was based on the caregiver's assessment of the adolescent's behavior using a 13-item Internalizing Index (II), based on work by Peterson and Zill (1986) using items derived from Achenbach's Behavioral Problem Index. Internalizing behaviors are an indicator of depressive symptoms in children. The index scale range from 0 (low) to 26 (high). The internal reliability for this scale is 0.82 (PSID, 2010).

The second measure is the short form of the Children's Depression Inventory (CDI). The CDI, included in the second and third wave of the CDS, is a widely used assessment of depressive symptoms with good reliability and validity (Cronbach's α of 0.80) (Allgaier et al., 2012). The CDI rates the severity of symptoms related to depression and/or dysthymic disorder in children and adolescents. Adolescents were given 10 sets of three statements and were asked to select one statement from each set to indicate how they had felt over the last two weeks. Measured symptoms included being bothered by things; having no friends; hating oneself; disliking how one looks; and feeling: sad, alone, like crying, unloved, that things never work out, and that they do things wrong. Responses were totaled, resulting in possible scores that range from a low of 0 (no depressive symptoms) to a high of 20.

2.2.3. Confounders

We considered several potential confounders including the child's sex, birth year, race (categorized as White Non-Hispanic, Black Non-Hispanic, Hispanic, and Other), whether or not parents had completed high school and college when the child was born, and mother's age at the child's birth. In an attempt to take genetic factors into account, we also controlled for parental serious mental illness. We used the K6-scale, a self-rated 6-item scale that screens for mood and anxiety disorders (Kessler et al., 2010). This scale has been validated in the United States and around the world as a measure of psychological distress and mental illness (Kessler et al., 2010). The K6 score ranges from 0 to 24, and as previous research has shown that dichotomous scoring of responses in the range 13+ vs. 0–12 discriminates between respondents with and without serious mental illness with good accuracy, we chose this cut-off point.

2.3. Statistical analysis

The dependent variables were discrete and thus treated as count data (Affi et al., 2007). The dependent variables had a positive skew, making a Poisson model (which forces the mean and the variance to be equal) inappropriate (Affi et al., 2007). A negative binomial model fitted the data better than either a Poisson or a zero-inflated Poisson model. With a Vuong test, the negative binomial model was tested

against the zero-inflated negative binomial (Affi et al., 2007), and was found best suited to this data. The negative binomial model models the log of the expected count as a function of the predictor variables. The coefficients can be interpreted as follows; for a one unit change in the predictor variable, the difference in the logs of expected counts of the response variable is will change by the respective regression coefficient, holding the other predictor variables in the model constant (UCLA Statistical Consulting Group, 2016). From the negative binomial regression, associations are presented as Incidence Rate Ratios (IRR) with 95% confidence intervals (CI). The IRR is interpreted as the estimated rate ratio comparing exposed with unexposed, given the variables are held constant in the model.

When examining the effect of each childhood social adversity on adolescent depressive symptoms, three multivariate models were analyzed for each exposure and outcome: Model I adjusted for sex and birth year. Model II added adjustments for race, mother's age at the child's birth, parental serious mental illness, and parental education. In the third model, all childhood social adversities were included simultaneously.

To assess the effects of cumulative exposure to childhood social adversity, the total number of exposures to different adversities were summed up and grouped into: 0, 1, 2, and 3 or more adversities. In the analysis of cumulative effects, only the first exposure for each adversity was considered. In these analyses, Models I and II were repeated.

As the PSID oversampled for African American and low-income families, we used weights provided by the PSID to allow the sample to approximate a representative sample of the US population, i.e. to adjust analyses for attrition and oversampling of low-income families.

Statistical analyses were conducted using SAS v.9.4 and Stata v. 13.1.

2.4. Ethical approval

The UCLA Institutional Review Board for Human Subjects has determined that this research is exempt from human subjects review because it involves the secondary analysis of public use, de-identified survey data.

3. Results

Table 1 provides weighted descriptive characteristics for the 2223 sample members. Nearly 60% were exposed to at least one indicator of childhood social adversity. The most common indicators were single-parent household and household public assistance. Compared to children with no history of social adversity, those exposed tended to have slightly younger mothers, and parents with serious mental illness. Their parents more often had lower levels of education, in particular parents of children growing up in poverty.

All studied childhood adversities were highly inter-correlated (Table 2). Specifically single-parent household and public assistance were highly correlated, as were poverty and public assistance.

Children exposed to childhood social adversity reported higher levels of adolescent depressive symptoms captured by the two depression scales. For the entire sample, the Internalizing Index mean was 3.8 (SE: 0.1). Children not exposed to social adversity reported a lower mean score (3.1 (SE: 0.1)) compared to children who experienced social adversity (mean scores spanning from 4.2 to 4.7 depending on type of adversity). The overall mean of the CDI score was 2.8 (0.1), with higher means in children exposed to adversity.

Table 3 shows the crude and multi-adjusted IRRs with 95% CIs for the association between childhood social adversity and total scores of Internalizing Index and Children's Depression Inventory respectively. The results for the Internalizing Index suggest a positive association between several of the adversities and adolescents' internalizing behavior (Model I). Single-parent household, household public assistance, and residential instability were associated with significantly

Table 1
Sample characteristics of the participants in the Child Development Supplement of the Panel Study of Income Dynamics, weighted numbers and column percent.

	All	No childhood social adversity	Single-parent household	Poverty	Household public assistance	Long-term parental unemployment	Residential instability
n (%)	2223	910	826	210	751	66	503
Characteristics							
Females	1102 (50)	448 (49)	390 (47)	101 (48)	374 (48)	26 (50)	240 (48)
Race							
White non-Hispanic	1422 (64)	691 (76)	400 (48)	41 (19)	320 (43)	32.7 (50)	324 (64)
Black non-Hispanic	370 (17)	39 (4)	288 (35)	100 (48)	262 (35)	28.2 (43)	122 (24)
Hispanic	278 (13)	110 (12)	70 (9)	49 (23)	118 (16)	3.0 (5)	28 (6)
Other	153 (7)	70 (8)	67 (8)	20 (10)	50 (7)	2.0 (3)	29 (6)
Parental characteristics							
Mother's age at child birth (mean (SE))	27.8 (0.2)	29.9 (0.3)	26.8 (0.4)	25.8 (0.5)	26.0 (0.4)	26.6 (0.4)	24.9 (0.4)
Parental serious mental illness (PCG/OCG K6 > 12)							
No	2056 (92)	863 (95)	747 (90)	181 (86)	652 (87)	56 (85)	454 (90)
Yes	167 (8)	47 (5)	79 (10)	29 (14)	99 (13)	10 (15)	49 (10)
Parental high school completion at child's birth							
No	397 (18)	113 (12)	188 (23)	111 (53)	221 (29)	10 (14)	76 (15)
Yes	1826 (82)	797 (88)	637 (77)	99 (47)	530 (71)	56 (86)	428 (85)
Parental college completion at child's birth							
No	1403 (63)	412 (45)	696 (84)	206 (98)	646 (86)	46 (69)	402 (80)
Yes	820 (37)	498 (55)	130 (16)	4 (2)	105 (14)	20 (31)	101 (20)
Adolescent depressive symptoms							
Internalizing Index (mean, SE)	3.8 (0.1)	3.1 (0.1)	4.4 (0.2)	4.2 (0.2)	4.7 (0.2)	4.6 (0.5)	4.3 (0.3)
Children's Depression Inventory (mean, SE)	2.8 (0.1)	2.5 (0.1)	3.1 (0.1)	2.7 (0.2)	3.0 (0.1)	1.9 (0.3)	3.3 (0.2)

increased IRR. Controlling for mother's age, parental serious mental illness, and parental education reduced the estimates markedly, and only single-parent household and public assistance remained significant (IRR: 1.3 (95% CI 1.1–1.4), and IRR 1.2 (95% CI 1.1–1.4) respectively). When adjustments were made for all adversities simultaneously, the IRRs were slightly reduced further (Table 3, Model III).

The timing of exposure to the different adversities was of less importance (Table 3), as the risk estimates for the different exposure periods were similar, with overlapping CIs.

Similarly to the results for the Internalizing Index, we found a positive association between single-parent household, and residential instability and adolescents' CDI scores (Table 3). These associations declined slightly in Model II and Model III.

Duration of exposure had only a small impact on the association (Supplemental Table 1).

As shown in Table 4, 15% of the individuals had been exposed to three or more adversities. Both the crude and multi-adjusted models revealed a relationship between cumulative number of adversities and the risk of adolescent depression. Analyses of timing of exposure to cumulative number of adversities showed that, if exposed to multiple

indicators (i.e. ≥3 indicators), the elevated risk for adolescent depression remained significant only among those who experienced multiple indicators in different childhood periods. Thus, the timing of cumulative exposures appeared to have little effect on the risk of adolescent depression.

4. Discussion

4.1. Summary

This study of 2223 adolescents shows that childhood social adversity is associated with an elevated risk of adolescent depressive symptoms. Individuals exposed to childhood social adversity scored higher on the Children's Depression Inventory scale, and their parents rated them higher on the Internalizing Index scale. Among the studied social adversities, single-parent household and residential instability particularly increased the risk of adolescent depressive symptoms regardless of timing of the exposure. Lastly, cumulative exposure to childhood social adversity was associated with increased risk of adolescent depression, also regardless of timing.

Table 2
Pearson correlation matrix of sample characteristics and childhood social adversities.

	1	2	3	4	5	6	7	8	9	10
1. Sex	1.00									
2. Race	-0.03	1.00								
3. Mother's age at child's birth	0.01	-0.10**	1.00							
4. Parental serious mental illness	-0.01	0.07**	-0.15**	1.00						
5. Parental education	0.03	-0.24**	0.39**	-0.16**	1.00					
6. Single-parent household	-0.03	0.18**	-0.32**	0.07**	-0.34**	1.00				
7. Poverty	0.00	0.16**	-0.14**	0.13**	-0.23**	0.28**	1.00			
8. Public assistance	-0.01	0.22**	-0.29**	0.20**	-0.34**	0.50**	0.40**	1.00		
9. Long-term parental unemployment	-0.02	0.03	-0.08**	0.05*	-0.05*	0.10**	0.14**	0.16**	1.00	
10. Residential instability	-0.03	-0.01	-0.33**	0.07*	-0.16**	0.30**	0.10**	0.22**	0.07**	1.00

* p < 0.01;

**p < 0.001.

Table 3

Associations between timing of childhood social adversity, and adolescent depressive symptoms. Weighted incidence rate ratios (IRR) from negative binomial regression models with 95% confidence intervals (CI).

Indicators of social adversity	n (unweighted)	weighted (%)	Outcome measures							
			Internalizing Index			Children's Depression Inventory				
			Model I ^a	Model II ^b	Model III ^c	Model I ^a	Model II ^b	Model III ^c		
Single-parent household										
Any time	1092	37.1	1.4 (1.2–1.6)	1.3 (1.1–1.5)	1.2 (1.1–1.4)	1.3 (1.1–1.5)	1.3 (1.1–1.5)	1.2 (1.0–1.4)		
Infancy and early childhood	185	6.6	1.3 (1.1–1.6)	1.1 (0.9–1.3)	1.1 (0.9–1.3)	1.4 (1.1–1.8)	1.4 (1.1–1.8)	1.3 (1.0–1.7)		
Middle childhood and early adolescence	239	9.3	1.4 (1.1–1.7)	1.3 (1.1–1.6)	1.3 (1.0–1.5)	1.2 (1.0–1.4)	1.2 (1.0–1.4)	1.1 (1.0–1.4)		
Both periods	668	21.3	1.4 (1.2–1.7)	1.3 (1.1–1.6)	1.3 (1.1–1.5)	1.3 (1.0–1.5)	1.3 (1.0–1.5)	1.2 (1.0–1.5)		
Poverty										
Any time	301	9.4	1.1 (1.0–1.4)	1.0 (0.8–1.2)	0.9 (0.7–1.1)	1.0 (0.8–1.2)	0.9 (0.7–1.1)	0.9 (0.7–1.1)		
Infancy and early childhood	184	6.4	1.1 (0.9–1.4)	1.0 (0.8–1.2)	0.9 (0.7–1.1)	0.9 (0.7–1.2)	0.9 (0.7–1.1)	0.9 (0.6–1.1)		
Middle childhood and early adolescence	57	1.7	1.3 (0.9–1.9)	1.0 (0.7–1.4)	1.0 (0.7–1.3)	1.1 (1.0–1.5)	1.1 (0.9–1.3)	1.2 (0.9–1.6)		
Both periods	60	1.3	1.1 (0.7–1.7)	0.9 (0.5–1.4)	0.8 (0.5–1.3)	0.8 (0.6–1.1)	0.7 (0.5–0.9)	0.7 (0.5–1.0)		
Household receiving public assistance										
Any time	949	33.8	1.5 (1.3–1.7)	1.2 (1.1–1.4)	1.2 (1.0–1.3)	1.2 (1.0–1.4)	1.1 (0.9–1.2)	1.0 (0.8–1.2)		
Infancy and early childhood	242	9.1	1.4 (1.2–1.8)	1.2 (0.9–1.5)	1.1 (0.9–1.4)	1.3 (1.0–1.6)	1.2 (0.9–1.5)	1.1 (0.9–1.4)		
Middle childhood and early adolescence	185	7.3	1.4 (1.2–1.7)	1.3 (1.0–1.5)	1.2 (1.0–1.4)	1.2 (1.0–1.4)	1.1 (0.9–1.3)	1.0 (0.8–1.3)		
Both periods	522	17.4	1.6 (1.4–1.9)	1.3 (1.1–1.4)	1.2 (1.0–1.4)	1.1 (0.9–1.3)	1.0 (0.8–1.2)	0.9 (0.7–1.1)		
Long-term parental unemployment										
Any time	109	3.0	1.4 (0.9–2.0)	1.1 (0.8–1.5)	1.1 (0.8–1.5)	0.7 (0.5–1.0)	0.7 (0.5–1.0)	0.7 (0.5–1.0)		
Infancy and early childhood	52	1.2	1.3 (0.6–2.8)	1.1 (0.6–1.9)	1.1 (0.6–2.0)	0.7 (0.4–1.3)	0.7 (0.4–1.1)	0.7 (0.5–1.2)		
Middle childhood and early adolescence	48	1.5	1.5 (1.1–2.1)	1.2 (0.8–1.7)	1.1 (0.8–1.6)	0.7 (0.4–1.3)	0.7 (0.4–1.3)	0.7 (0.4–1.2)		
Both periods	9	0.3	0.5 (0.3–1.1)	0.5 (0.3–0.9)	0.6 (0.3–1.0)	0.5 (0.3–0.9)	0.5 (0.3–1.1)	0.6 (0.3–1.1)		
Residential instability										
Any time	573	22.6	1.3 (1.1–1.5)	1.1 (1.0–1.3)	1.1 (1.0–1.2)	1.4 (1.2–1.6)	1.4 (1.2–1.6)	1.3 (1.2–1.5)		
Infancy and early childhood	177	8.0	1.3 (1.0–1.7)	1.1 (0.8–1.4)	1.0 (0.8–1.3)	1.4 (1.0–1.8)	1.4 (1.0–1.8)	1.3 (1.0–1.7)		
Middle childhood and early adolescence	89	2.9	1.5 (1.2–1.9)	1.4 (1.1–1.8)	1.3 (1.0–1.7)	1.5 (1.2–2.0)	1.6 (1.3–2.1)	1.6 (1.2–2.1)		
Both periods	307	11.1	1.2 (1.1–1.4)	1.1 (1.0–1.3)	1.1 (0.9–1.2)	1.3 (1.1–1.6)	1.4 (1.1–1.6)	1.3 (1.1–1.6)		

Reference group: No social adversity

^a Adjusted for sex, birth year, and race.

^b Model I with additional adjustments for mother's age at the child's birth, parental serious mental illness, and parental education.

^c Model II with additional adjustments for other childhood social adversities at any time.

Several of the adversities used in this study were associated with increased scores of adolescent depressive symptoms, captured with the

Internalizing Index scale. This is consistent with a recent meta-analysis on socioeconomic inequalities and mental health in children and

Table 4

Associations between cumulative number of indicators of childhood social adversity, and adolescent depressive symptoms. Weighted incidence rate ratios (IRR) from negative binomial regression models with 95% confidence intervals (CI).

Total number of indicators of social adversity, and timing	n (unweighted)	weighted (%)	Internalizing Index		Children's Depression Inventory	
			Model I ^a	Model II ^b	Model I ^a	Model II ^b
0	791	44.5	1 (REF)	1 (REF)	1 (REF)	1 (REF)
1	490	24.2	1.4 (1.2–1.6)	1.3 (1.1–1.5)	1.3 (1.1–1.5)	1.3 (1.1–1.5)
2	446	15.9	1.6 (1.4–1.9)	1.5 (1.2–1.7)	1.4 (1.1–1.7)	1.3 (1.1–1.6)
3+	496	15.3	1.7 (1.4–2.1)	1.4 (1.1–1.7)	1.4 (1.2–1.7)	1.4 (1.1–1.7)
<i>1 indicator occurring in:</i>						
Infancy and early childhood	150	7.5	1.4 (1.1–1.7)	1.1 (0.9–1.4)	1.4 (1.1–1.7)	1.3 (1.1–1.7)
Middle childhood and early adolescence	153	8.2	1.4 (1.1–1.7)	1.2 (1.0–1.5)	1.2 (0.9–1.4)	1.1 (0.9–1.4)
Both periods	187	8.5	1.5 (1.2–1.9)	1.4 (1.1–1.8)	1.3 (1.0–1.7)	1.3 (1.0–1.8)
<i>2 indicators, whereof:</i>						
Both occurring in infancy and early childhood	45	1.5	1.6 (1.1–2.2)	1.5 (1.0–2.2)	1.7 (1.0–2.9)	1.7 (1.0–3.0)
Both occurring in middle childhood and early adolescence	46	1.8	1.6 (1.1–2.3)	1.7 (1.1–2.5)	1.1 (0.7–1.7)	1.2 (0.8–1.8)
Both occurring in different periods	355	12.6	1.7 (1.4–2.0)	1.4 (1.2–1.7)	1.4 (1.1–1.7)	1.3 (1.0–1.6)
<i>3+ indicators, whereof:</i>						
At least three occurring in infancy and early childhood	28	0.9	2.1 (1.2–3.9)	1.3 (0.8–2.4)	1.2 (0.6–2.6)	1.0 (0.5–2.2)
At least three occurring in middle childhood and early adolescence	7	0.2	1.2 (0.8–1.8)	0.9 (0.6–1.3)	1.2 (0.4–3.6)	1.1 (0.3–4.2)
The indicators occurred in different periods	461	14.3	1.7 (1.4–2.0)	1.4 (1.1–1.7)	1.5 (1.2–1.8)	1.4 (1.2–1.7)

^a Adjusted for sex, birth year, and race.

^b Model I with additional adjustments for mother's age at the child's birth, parental serious mental illness, and parental education.

adolescents that concluded that socioeconomically disadvantaged children and adolescents were much more likely to develop mental health problems, and particularly children growing up in long-term social adversity (Reiss, 2013).

The analyses in the current study enabled us to distinguish between the effects of different social adversities. Single-parent household and public assistance were associated with the highest IRR for the Internalizing Index score. Although highly correlated, the association between single-parent household and depressive symptoms remained after taking public assistance into account (Table 3, Model III). Prior studies have also shown these indicators to play a major part in the increased risk of depression (Amato and Keith, 1991; Duncan and Brooks-Gunn, 1997; Weitoft et al., 2003; McLanahan and Sandefur, 1994; Daryanani et al., 2016). A large-scale Swedish study examining mortality and morbidity in children with single parents showed that, although growing up in a single-parent family often is associated with a wide range of other social adversities including lack of household resources the increased risk of mental health problems for children of single parents remained even after taking other adversities into account (Weitoft et al., 2003). A number of explanations have been proposed to account for why lone parenthood may have negative effects on children's mental health (Amato and Keith, 1991; McLanahan and Sandefur, 1994; Daryanani et al., 2016). For example, the total amount of time devoted to the child may be reduced since one parent is missing (McLanahan and Sandefur, 1994; Daryanani et al., 2016; Kendig and Bianchi, 2008). The family conflict perspective proposes that the conflict between parents before and during the separation period leading up to single-parenthood is a severe stressor for children (Amato and Keith, 1991). With respect to parenting style, a recent study examining single mother parenting and adolescent psychopathology showed that single mothers were more likely to engage in rejecting parenting behaviors, e.g. offering minimal emotional support and little compassion (Daryanani et al., 2016). This in turn predicted adolescent psychopathology (Daryanani et al., 2016).

Further, our results showed that residential instability was associated with increased risk for adolescent depression. This is consistent with studies showing that frequent relocation is associated with higher risk of depression onset both in childhood, adolescence and adulthood (Gilman et al., 2003).

On the other hand, poverty and long-term parental unemployment were not independently associated with increased depression scores in our study. Findings from prior studies examining the relationship between these indicators of childhood adversity and adolescent depression have been inconclusive. Although some studies have found a positive association between childhood poverty and adolescent depression (Duncan and Brooks-Gunn, 1997; Najman et al., 2010), a recent study that also used the PSID found that the association between poverty and adolescent depression was explained by other family characteristics and by mother's depression (Butler, 2014). Another possible explanation, also shown in other settings, is that other factors that often accompany poverty explains most of the association (e.g. the family environment) (Butler, 2014; Tracy et al., 2008). Studies have demonstrated that, adjusting for other stressful life events often attenuate the association between poverty and adolescent depression (Tracy et al., 2008). With respect to parental unemployment, unlike findings in previous studies (Sleskova et al., 2006; Torikka et al., 2014), we found no significant association with adolescent depression. One explanation may be that it is not the parental unemployment per se that predicts adolescent depression, but rather poverty as a result of job loss that leads to family stress. Not all families suffer from poverty as a result of job loss, which could be seen as one explanation to the discrepancy between our results and earlier studies. Also, we focused on unemployment of at least one parent without distinguishing between mother's and father's unemployment. Prior studies have demonstrated that the effect of father's unemployment is worse on children (Artazcoz et al., 2004; Piko and Fitzpatrick, 2001), and that

mother's unemployment in fact may have a positive role in adolescent's mental health (Piko and Fitzpatrick, 2001).

We found that age at exposure to social adversity was not differentially related to the risk of adolescent depressive symptoms. We recently examined the effect of childhood adversity on psychiatric disorder, including depression, in a Swedish cohort, and found that timing of exposure to childhood adversity had very little impact on the risk (Björkenstam et al., 2016). These findings are in contrast to earlier studies that found timing of exposure to childhood adversity to be important for subsequent adolescent depression (Andersen and Teicher, 2008; Dunn et al., 2013; Heim and Binder, 2012; Kaplow and Widom, 2007; Reiss, 2013). However those earlier studies examined child maltreatment exclusively. They showed that early exposure is worse for later mental health outcomes. Childhood maltreatment, including abuse and neglect, is a type of childhood adversity that may have more severe effects than the adversities used in our study which may explain the discrepancy in our results. Another explanation for the inconsistency in findings is that the associations may differ depending on the outcome studied. One recent meta-analysis on socioeconomic inequalities and mental health in children and adolescents found that the impact of social adversity on mental health was stronger in early childhood than in adolescence (Reiss, 2013). However, this study included both internalizing and externalizing disorders.

Lastly, consistent with prior research in various settings (Björkenstam et al., 2015; Hazel et al., 2008; Mersky et al., 2013), we found indications of an association between cumulative exposure to childhood social adversity and adolescent depressive symptoms.

As discussed by others, there are several pathways through which childhood social adversity may contribute to the development of depressive symptoms in adolescence. In our study, the results suggested a positive association between several of the adversities and adolescent depressive symptoms even after controlling for parental mental health and other factors. These findings indicate support for the social causation hypothesis.

Childhood is the time in life when the brain is most sensitive to experience, and therefore most easily influenced in positive and negative ways. It is during these times in life when social, emotional, cognitive and physical experiences will shape neural systems in ways that influence functioning for a lifetime. In particular, home and family environments are powerful determinants of emotional and behavioral functioning later in life (Schaffer and Kipp, 2014). Prior studies have shown that childhood social adversities, such as those included in our study, are major contributors to increased stress levels during childhood (Mossakowski, 2015; Schaffer and Kipp, 2014). These increased stress levels may cause disruptions in cognition, coping styles and behavior through various pathways (Schaffer and Kipp, 2014). Studies have shown that the increased stress from social adversity may lead to emotional dysfunction, and children with poor emotional competence may have higher likelihood of adolescent depression (Shapiro and Steinberg, 2013; Wadsworth and Butterworth, 2006). Further, the increased stress often leads to lower self-esteem and self-worth, which in turn may increase the risk of depressive symptoms (Mossakowski, 2015; Wadsworth and Butterworth, 2006).

Furthermore, studies have suggested that the influence of social adversity on adolescent depressive symptoms is mediated by adolescent stress burden, and thus that social adversities, rather than being independent risk factors for depression in adolescence, contribute to risk for early depression due to its association with adolescent stress burden (Hazel et al., 2008; Mossakowski, 2015). However, our finding that age of exposure was not differentially associated with risk of adolescent depressive symptoms appears not to support this notion. It is a plausible hypothesis that adolescent stress burden may be increased by more proximate adversity during pre-adolescence. However, even adversity during early childhood increased risk for adolescent depressive symptoms in our study. It is the total load of

adversities that affects the risk of depressive symptoms rather than the timing of each adversity.

The study has methodological weaknesses that need to be addressed in future research. Because of the structure of the data, alternative causal pathways cannot be fully discounted. The findings in our study are also limited by the difficulty in fully capturing the concepts being measured. While the measures we used are well-tested and used widely, they are based on self- or parent-reported survey questions and not professional diagnostic tools. Further, the prevalence of some of the indicators may be underreported, which would bias our findings towards the null. Another limitation is that we have not examined the fluidity of social adversities, but rather, as done by others (Felitti and Anda, 2010), treated them as discrete life events. Furthermore, in an attempt to take genetic factors into account, we used parental serious mental illness, measured with the K6 screening scale, as a proxy for genetic liability. This measure only partly captures genetic liability for depression. Finally, we only had information every other year, i.e. we were not able to capture what happened between the waves with respect to social adversity. This is especially important when examining the effect of long-term exposure to childhood social adversity.

5. Conclusion

This study demonstrated that childhood social adversity, and especially single-parent household and residential instability, is likely to increase the risk of depressive symptoms in adolescence, and that children exposed to a larger number of adversities are a particularly vulnerable group for the development of adolescent depression. Our findings further revealed that age at exposure to social adversity is of less importance with respect to risk of adolescent depressive symptoms. Our findings stress the importance of increased and extended support to disadvantaged children. This should be part of any strategy aiming at reducing the negative effects of social adversity on adolescent mental health outcomes. Continued work is also needed to reduce inequalities in depressive symptoms. Policies and interventions to reduce adolescent depression need to consider the social background of the family as an important risk or protective factor.

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.jad.2017.01.035.

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