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Childhood adversity and psychiatric disorder in young adulthood: An analysis of 107,704 Swedes

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ABSTRACT

Childhood adversity (CA) is associated with increased risks of psychiatric disorder in young adulthood, but details in this association are less known. We aimed to explore the association of a range of CA indicators with psychiatric disorder in young adulthood, and the impact of age at exposure, disorder type and accumulation of indicators. We capitalized on Sweden's extensive and high-quality registers and analyzed a cohort of all Swedes (N = 107,704) born in Stockholm County 1987–1991. Adversities included familial death, parental substance misuse and psychiatric disorder, parental criminality, parental separation, public assistance recipiency and residential instability. Age at exposure was categorized as: 0–6.9 years (infancy and early childhood), 7–11.9 years (middle childhood), and 12–14 years (early adolescence). Psychiatric disorders after age 15 were defined from ICD codes through registers. Risks were calculated as Hazard Ratios (HR) with 95% confidence intervals (CI).

Results showed that exposure to at least one CA was associated with an increased risk of psychiatric disorder (HR 1.4, 95% CI: 1.3–1.4). Risks were increased for mood, anxiety, and psychotic disorders and ADHD but not for eating disorders. The risk varied with type of disorder but was similar for all exposure periods. Individuals with multiple (3+) CAs had a two-fold risk of psychiatric disorder (HR 2.0, 95% CI: 1.9–2.1). In conclusion, our findings support the long-term negative impact of CA on mental health, regardless of developmental period of exposure. Given that experience of CA is common, efforts should be put to alleviate the burden of childhood adversities for children, particularly among the most disadvantaged.

1. Introduction

There is accumulating evidence that childhood adversities (CAs) are associated with an increased risk of mental disorders in young adulthood, including substance misuse (Anda, 2008; Bellis et al., 2014; Dube et al., 2003; Kessler et al., 2010; McLaughlin et al., 2010a; Mersky et al., 2013), psychosis (Trauelsen et al., 2015), mood disorder (Anda, 2008; Björkenstam et al., 2015; Chapman et al., 2004; Kessler et al., 2010; McLaughlin et al., 2010a; Mersky et al., 2013; Sareen et al., 2013), and anxiety (Kessler et al., 2010; McLaughlin et al., 2010a; Mersky et al., 2013; Sareen et al., 2013). CAs may shape mental health through early life influences on neurodevelopment and psychosocial functioning.

Studies investigating adverse childhood experiences (ACE) as risk factors for psychiatric disorders have generally included adversities such as childhood abuse, neglect, and growing up in a dysfunctional household (Anda, 2008; Chapman et al., 2004; Felitti et al., 1998), the latter being characterized by substance misuse in the home, mentally ill family members, incarcerated parents etc. (Anda, 2008). CAs have been positively associated with the risk of psychiatric disorder in young adulthood in a number of recent studies. However, few studies have focused on to what extent the timing of exposure to CA affect subsequent onset of psychiatric disorder (Andersen and Teicher, 2008; Dunn et al., 2013; Heim and

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Binder, 2012; Kaplow and Widom, 2007; Khan et al., 2015) and whether the strength of the association depends on type of disorder and adversity. Furthermore, most previous studies have used retrospectively self-reported adversities and are, thus, limited by recall bias (Anda, 2008; Dube et al., 2003; Kessler et al., 2010; Mersky et al., 2013).

The results from the landmark ACE-study in San Diego (US) have suggested a framework for a new paradigm for medical, public health and social services (Felitti and Anda, 2010). The strong links between accumulations of childhood adversity, hypothesized to be indications of childhood traumas, and later psychiatric disorders have challenged a biological model of the etiology of psychiatric disorders (Skehan et al., 2012), but European replications based on the ACE-construct are still relatively few and far-in-between. Given that childhood adversities are common, any long-term detrimental effects on mental health would have an immense effect on both individuals and societies. Shedding light on the details of the CA and mental health relationship might inform the timing and targeting of public health interventions aiming to alleviate the burden of CA upon exposed children.

In the present study we capitalize on Sweden's extensive and high quality registers, by using a large sample of all individuals born between 1987 and 1991 in Stockholm County, Sweden. Our aim is to explore:

1. the association between different indicators of CA and the risk of psychiatric disorder in young adulthood, and whether the association differs by age at exposure and type of psychiatric disorder; and
2. the effects of cumulative exposure to multiple indicators of CA on the risk of psychiatric disorder

2. Methods

2.1. Study population

In total nine registers were merged to conduct the current analyses. The study population was defined as all individuals born in Stockholm County, Sweden between 1987 and 1991 (n = 116,087), recorded in the Medical Birth Register (Cnattingius et al., 1990). Stockholm County, with 11 municipalities, spans over an area of 2517 square miles. It has approximately 2.2 million people living in the metropolitan area. The Swedish health care system is publicly funded and there is universal access to it. Public mental health services comprise the large majority of mental health care since the percentage of population in contact with private psychiatrists is low (Dalman and Wicks, 2006). The depiction of the final analytical sample (n = 101,704) and exclusion criteria are illustrated in Fig. 1. Analyses not shown revealed that excluded individuals were more likely to have a foreign-born mother, to have been diagnosed with a psychiatric diagnosis before age 15 years, and to have experienced CA, as compared to the final analytical sample. The unique Swedish personal identity number was used to link this cohort to multiple health care and administrative registers:

The Causes of Death Register comprises information on all deaths of Swedish residents. The National Patient Register (NPR) includes all individuals admitted to psychiatric or general hospitals, with complete coverage for all care since 1987. In addition, an administrative health care database (VAL) was used containing individual data on utilization of publicly funded inpatient and outpatient health care in Stockholm County since 1997. The Total Enumeration Income Survey contains data on the income of and governmental benefits provided to all Swedish residents. The Total Population Register includes information on age, sex, place of residence, and other relevant demographic characteristics. The Longitudinal Integration Database for Health Insurance and Labor Market Studies (LISA) integrates existing data from the labor market, educational and social sectors. The Register of Court Convictions contains information on all court convictions in Sweden for persons 15 years of age or older. Families were linked together through the Multi-Generation register, which contains all known relationships between children and parents (born in 1932 or later) since 1961.

2.2. Measures

2.2.1. Indicators of childhood adversity

CA indicators were selected based upon prior research demonstrating them to have significant adverse health or social implications (Anda, 2008; Björkenstam et al., 2013; Farrington and Welsh, 2007; Rasmussen et al., 2014; Ringback Weitoft et al., 2008; Siegenthaler et al., 2012; Vinnerljung et al., 2010; Wood et al., 1993), and measured between birth and age 14. Based on the child developmental stage theories (Schaffer and Kipp, 2014), we chose the following three exposure periods: 0–6.9 years (infancy and early childhood), 7–11.9 years (middle childhood), and 12–14 years (early adolescence). These exposure periods were informed by previous developmental timing research (Schaffer and Kipp, 2014) and encompass major transitions in a child's life. If an individual was exposed to the same indicator more than once, one indicator in each sensitive period was considered.

Familial death: Death of a parent or a sibling.

Parental substance misuse: At least one parent hospitalized with a main diagnosis for alcohol and/or drug-related substance misuse (International Classification of Disease (ICD-9) codes: 291–292, 303–3050, 3570, 4255, 5353, 5710, 5711–5713, 6483, 6555, 9650, 9696–9697; ICD-10: E244, F10–F16, F18–F19, G312, G621, G721, I426, K292, K70, K852, K86, O354–355, P044, T40, T436, T51, Z502–503, Z714, Z721–Z722) and/or received an alcohol or drug-related conviction.

Parental psychiatric disorder (excluding substance misuse related diagnoses): At least one parent hospitalized with a main diagnosis of psychiatric disorder (ICD-9: 290–319; ICD10: F00–F99).

Severe parental criminality: A parent sentenced to prison, probation, or forensic psychiatric care.

Parental separation: Parental marital status was measured when the child was between the ages of 3 and 14. This indicator was coded as 1 if the parent's marital status changed from married to divorced between two years.

Household living on public assistance: This indicator, used as a proxy for relative poverty, was defined as at least one parent having received public assistance that constituted more than 50% of the yearly income during a year or more (when the child was between 3 and 14 years of age). In Sweden, public assistance is a form of cash income allowance from local social authorities after a thorough individual means test, designed to guarantee people a minimum standard of living (Hessle and Vinnerljung, 2000).

Residential instability: Two or more changes in place of residency.

2.2.2. Psychiatric disorder

The study population was prospectively followed for onset of psychiatric disorder after age 15 (from 2002 if born 1987 through 2006 if born 1991) until at most December 31st 2011. Psychiatric disorder was defined as a register-based diagnosis of any psychiatric disorder (ICD-10 codes F00–99) during psychiatric inpatient care, psychiatric outpatient care, and/or primary care, as recorded in the NPR and VAL Additionally, the following types of disorders were considered separately: substance misuse (ICD-10: F10–19);

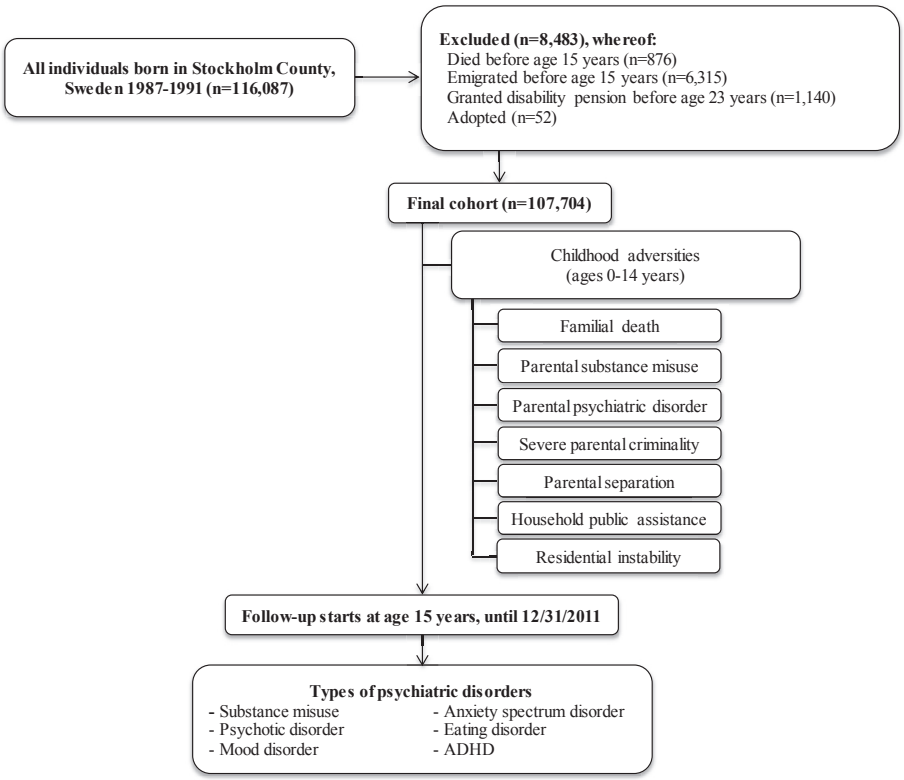

Fig. 1. Flow chart depicting study design and the derivation of study population.

psychotic disorders (ICD-10: F20–29); mood disorders (ICD-10: F30–39); anxiety, dissociative, stress-related and somatoform disorders (ICD-10: F40–48); eating disorders (ICD-10: F50), and Attention deficit hyperactivity disorder (ADHD), (ICD-10: F90).

2.2.3. Covariates

We considered a range of potential confounders.

People from disadvantaged family backgrounds are more likely to experience childhood adversities than those born in more privileged families (Anda, 2008; Kuh et al., 2004). Thus, we adjusted for parental education and income. Parental education was defined as the highest educational level among the parents when the child was 15 years old i.e., between 2002 and 2006, and classified in three categories: 1) Nine years of compulsory school, 2) 10–12 years of education (equivalent to senior high school), and 3) ≥13 years of education (i.e., university). Disposable income when the child was 15 years was obtained by calculating the sum of all the family members' incomes multiplied by the individual consumption weights, then divided by their aggregate consumption weight. We categorized disposable income into quartiles.

Parental country of birth was categorized as mother born in Sweden or not.

Psychopathology before age 15: was defined as having had a psychiatric in- or outpatient contact before age 15.

2.3. Statistical analysis

Statistical analyses were conducted using SAS v.9.4 and Stata v. 13. Multivariate analyses were performed using Cox hazards models of time to first diagnosis of psychiatric disorder. We assessed person-years at risk by adding up the years the individuals were alive and living in Sweden during the follow-up period (entry date defined as the date of the 15th birthday and exit date as the date of the first psychiatric diagnosis, date of death from the Causes of Death Register, date of emigration from the Register of the Total Population or the end of follow-up (i.e. December 31st 2011)). We examined the association between indicators of CA and psychiatric disorder in adulthood, in two regression models: Model I was adjusted for sex and birth year; and Model II was further adjusted for mother's country of birth, parental SEP (income and education), and psychopathology before age 15. Disorder-specific outcomes were analyzed using the same regression models. When we examined the different sensitive periods, we applied the exposure periods described above, and used the two regression models. To assess the effects of cumulative exposure to CA indicators, the total number of exposures to different adversities were summed up and grouped into: 0, 1, 2, and 3 + indicators. In the analysis of cumulative effects, only the first exposure for each indicator was considered. Because of the inter-correlations among the independent variables, we performed analysis to detect multicollinearity. First, we started by examining the bivariate correlations among independent variables. No pair of variables was highly correlated. The correlation coefficients ranged from 0.02 to 0.45; values in chi-square tests were $p < .0001$. We then evaluated the multicollinearity diagnostic statistics, the Variance Inflation Factor (VIF), produced by linear regression analysis (PROC REG with options VIF in SAS). The VIFs ranged from 1.01 to 1.35, indicating no presence of multicollinearity.

Sensitivity analyses: Parental psychiatric disorder is a proxy for genetic liability for psychiatric disorder and may increase risk of experiencing other CA indicators. Thus, we repeated the analyses of the remaining seven CA indicators in the subset of study participants without parental psychiatric disorder. CA increases the risk of childhood psychopathology which, in turn, is a powerful risk factor for psychopathology in adulthood. In order to examine the effect of CA on adult onset psychiatric disorder, we stratified

the cohort into two groups depending on history of childhood psychiatric disorder or not and repeated the analyses. We used parental psychiatric diagnoses as a proxy for parental psychopathology. Thus, we did not have detailed information on parental psychopathology.

Additional sensitivity analyses were carried out in which we incorporated a Bonferroni correction for multiple comparisons (Armstrong, 2014). These analyses yielded almost identical results, indicating consistent results.

Lastly, we performed a sensitivity analysis to examine the possibility of effect modification by parental education. Stratifications were made based on parental educational level.

3. Results

3.1. Prevalence of indicators of CA and psychiatric disorders

42% of the study participants (N = 107,704, 49% women and 51% men) were exposed to at least one indicator of CA (Table 1). Parental separation was the most common exposure (22%), followed by household public assistance (20%). Having a mother born outside Sweden, and low parental SEP were more common in children exposed to CA.

One in every five study participants received a diagnosis of psychiatric disorder during the follow-up period. The most common disorder types were anxiety spectrum disorders (46% of all psychiatric diagnoses), substance misuse (30%) and mood disorders (30%). Individuals exposed to CAs were more commonly diagnosed with psychiatric disorder during the follow-up period, compared to non-exposed.

3.2. Associations of indicators of CA with psychiatric disorder by sensitive exposure periods

Table 2 presents the crude and multi-adjusted HRs with 95% confidence intervals for risk of psychiatric disorder by exposure to CA indicators in sensitive periods. Initially we analyzed the sensitive periods for each of the psychiatric outcomes, revealing that there were no differences between the periods (data not shown). All indicators of CA were associated with an elevated risk of psychiatric disorder regardless of sensitive period. HRs ranged from 1.3, (95% CI 1.2–1.3) for familial death (any period) and residential instability (any period), to 1.6 (95% CI 1.5–1.7) for parental psychiatric disorder (any period), and 1.6, (95% CI 1.5–1.7) for household public assistance (any period). The risk appeared to further increase in those exposed to CA in more than one sensitive period. Adjustments for additional potential confounders (Model II) had only little effect on the estimates.

3.3. Differential associations of indicators of CA with type of psychiatric disorder

Exposure to CA significantly predicted all types of psychiatric disorders, except eating disorders (Table 3). The risk tended to be higher for substance misuse and psychotic disorders. Parental substance misuse, severe parental criminality, and household public assistance were particularly strong predictors of substance misuse in the study population. Parental psychiatric disorder was

Table 1
Selected cohort characteristics, by exposure to childhood adversity for the 107,704 individuals included in the study.

	All	Unexposed to childhood adversities	Familial death	Parental substance misuse	Parental psychiatric disorder	Severe parental criminality	Parental separation[a]	Household public assistance[a]	Residential instability
n (%)	107,704	62,725 (58)	3470 (3)	9216 (9)	5455 (5)	5180 (5)	23,265 (22)	21,924 (20)	5324 (5)
Characteristics									
Females	52,619 (49)	30,511 (49)	1703 (49)	4565 (50)	2651 (49)	2542 (49)	11,547 (50)	10,686 (49)	2594 (49)
Parental educational level									
9 years of education	6376 (6)	2072 (3)	626 (18)	1220 (13)	560 (10)	809 (16)	1617 (7)	3212 (15)	439 (8)
10–12 years of education	46,194 (43)	23,543 (38)	1625 (47)	5516 (60)	2656 (49)	3232 (62)	10,943 (47)	12,866 (59)	2631 (49)
>12 years of education	55,015 (51)	37,097 (59)	1147 (33)	2442 (26)	2212 (41)	1120 (22)	10,662 (46)	5777 (26)	2241 (42)
Missing	119 (0)	13 (0)	72 (2)	38 (0)	27 (0)	19 (0)	43 (0)	69 (0)	13 (0)
Parental income									
Quartile 1 (<91,999)	24,197 (22)	10,022 (16)	726 (21)	3162 (34)	1687 (31)	2003 (39)	6566 (28)	8970 (41)	1777 (33)
Quartile 2 (92,000–119,749)	27,926 (26)	14,775 (24)	788 (23)	2977 (32)	1663 (30)	1695 (33)	6808 (29)	7255 (33)	1566 (29)
Quartile 3 (119,750–154,699)	27,998 (26)	18,263 (29)	892 (26)	1963 (21)	1255 (23)	1001 (19)	5240 (23)	4014 (18)	1065 (20)
Quartile 4 (>154,700)	27,583 (26)	19,665 (31)	1064 (31)	1114 (12)	850 (16)	481 (9)	4651 (20)	1685 (8)	916 (17)
Mother born outside of Sweden	20,680 (19)	9533 (15)	823 (24)	2212 (24)	1315 (24)	1514 (29)	5164 (22)	7239 (33)	992 (19)
Psychiatric disorder before age 15	3268 (3)	1445 (2)	147 (4)	455 (5)	320 (6)	258 (5)	818 (4)	1090 (5)	242 (5)
Psychiatric disorder after age 15									
Any	21,156 (20)	10,160 (16)	852 (25)	2713 (29)	1651 (30)	1556 (30)	5512 (24)	6080 (28)	1296 (24)
Substance misuse	6300 (6)	2626 (4)	289 (8)	1107 (12)	587 (11)	623 (12)	1733 (7)	2168 (10)	461 (9)
Psychotic disorders	400 (0)	149 (0)	27 (1)	65 (1)	54 (1)	44 (1)	134 (1)	149 (1)	40 (1)
Mood disorders	6426 (6)	3043 (5)	263 (8)	835 (9)	580 (11)	494 (10)	1754 (8)	1844 (8)	399 (7)
Anxiety spectrum disorders	9715 (9)	4584 (7)	369 (11)	1248 (14)	795 (15)	717 (14)	2624 (11)	2832 (13)	594 (11)
Eating disorder	1502 (1)	884 (1)	46 (1)	127 (1)	83 (2)	62 (1)	349 (2)	256 (1)	62 (1)
ADHD	2149 (2)	853 (1)	100 (3)	338 (4)	199 (4)	212 (4)	615 (3)	767 (3)	205 (4)

[a] Data available from age 3.

Table 2
Cox proportional Hazard Ratios (HR) with 95% confidence intervals (CI) for the associations between indicators of childhood adversity, categorized in exposure windows reflecting the age at exposure, and psychiatric disorders after age 15 years.

Childhood adversity	n (cases + non-cases)	n (cases)	Any psychiatric disorder	
			Model I[a]	Model II[b]
Familial death				
Any period	3470	852	1.3 (1.2–1.4)	1.2 (1.2–1.3)
First and only occurrence 0–6 years	1321	318	1.3 (1.1–1.4)	1.2 (1.1–1.3)
First and only occurrence 7–11 years	1165	302	1.4 (1.2–1.6)	1.3 (1.2–1.5)
First and only occurrence 12–14 years	909	217	1.3 (1.1–1.4)	1.2 (1.1–1.4)
>1 exposure period	75	15	1.1 (0.7–1.1)	0.9 (0.6–1.6)
Parental substance misuse				
Any period	9216	2713	1.7 (1.6–1.8)	1.5 (1.5–1.6)
First and only occurrence 0–6 years	3008	768	1.4 (1.3–1.5)	1.3 (1.2–1.4)
First and only occurrence 7–11 years	1958	564	1.6 (1.5–1.8)	1.5 (1.4–1.6)
First and only occurrence 12–14 years	1429	424	1.7 (1.6–1.9)	1.6 (1.4–1.7)
>1 exposure period	2821	957	2.0 (1.9–2.2)	1.8 (1.7–1.9)
Parental psychiatric disorder				
Any period	5455	1651	1.7 (1.6–1.8)	1.6 (1.5–1.7)
First and only occurrence 0–6 years	1762	500	1.6 (1.5–1.7)	1.5 (1.3–1.6)
First and only occurrence 7–11 years	1607	480	1.7 (1.6–1.9)	1.6 (1.4–1.7)
First and only occurrence 12–14 years	1051	326	1.8 (1.6–2.0)	1.6 (1.4–1.8)
>1 exposure period	1035	345	2.0 (1.8–2.2)	1.8 (1.6–2.0)
Severe parental criminality				
Any period	5180	1556	1.7 (1.6–1.8)	1.5 (1.4–1.6)
First and only occurrence 0–6 years	2258	652	1.6 (1.5–1.7)	1.4 (1.3–1.6)
First and only occurrence 7–11 years	1167	342	1.6 (1.5–1.8)	1.5 (1.4–1.7)
First and only occurrence 12–14 years	581	182	1.8 (1.6–2.1)	1.6 (1.4–1.8)
>1 exposure period	1174	380	1.9 (1.7–2.1)	1.7 (1.5–1.8)
Parental separation[c]				
Any period	23,265	5512	1.3 (1.3–1.4)	1.3 (1.2–1.3)
First and only occurrence 0–6 years	8226	1962	1.3 (1.3–1.4)	1.3 (1.2–1.4)
First and only occurrence 7–11 years	9876	2272	1.3 (1.2–1.3)	1.2 (1.2–1.3)
First and only occurrence 12–14 years	3544	847	1.3 (1.2–1.4)	1.3 (1.2–1.4)
>1 exposure period	1619	431	1.5 (1.4–1.7)	1.4 (1.3–1.6)
Household public assistance[c]				
Any period	21,924	6080	1.7 (1.6–1.7)	1.6 (1.5–1.6)
First and only occurrence 0–6 years	3960	937	1.4 (1.3–1.5)	1.3 (1.3–1.4)
First and only occurrence 7–11 years	2690	711	1.6 (1.4–1.7)	1.5 (1.4–1.6)
First and only occurrence 12–14 years	1018	282	1.7 (1.5–1.9)	1.6 (1.4–1.8)
>1 exposure period	14,256	4150	1.8 (1.7–1.9)	1.7 (1.6–1.8)
Residential instability				
Any period	5324	1296	1.3 (1.2–1.4)	1.2 (1.2–1.3)
First and only occurrence 0–6 years	3130	720	1.2 (1.1–1.3)	1.2 (1.1–1.2)
First and only occurrence 7–11 years	1444	348	1.3 (1.2–1.5)	1.2 (1.1–1.3)
First and only occurrence 12–14 years	366	102	1.6 (1.4–2.0)	1.4 (1.2–1.7)
>1 exposure period	384	126	1.9 (1.6–2.2)	1.7 (1.4–2.0)

Reference group: No childhood adversity.
[a] Adjusted for sex and birth year.
[b] Model I with additional adjustments for mother's country of birth, parental income and education, and own psychopathology before age 15.
[c] Data available from age 3.

the CA indicator more strongly associated with psychotic and mood disorders (HR 2.6, 95% CI: 2.0–3.5 and HR 1.8, 95% CI: 1.8–2.0, respectively). Household public assistance and frequent moves in childhood appeared to be the strongest predictors for ADHD (HR 1.8, 95% CI: 1.6–2.0; and HR 1.7, 95% CI: 1.5–2.0 respectively). Adjusting for potential confounding factors (Model II) produced only minor attenuations of the risk estimates.

3.4. The effect of cumulative exposure to CA

Co-occurrence of adversities was common (Table 4). The most common combination of adversities in those exposed to two adversities (10%) was parental separation and public assistance (33%) (data not shown). Exposure to parental substance abuse, parental criminality and public assistance simultaneously was common (25% of those exposed to three adversities had been exposed to these adversities (data not shown)). There was a graded relationship between the number of CA indicators and the risk of psychiatric disorder. HRs ranged from 1.4 (95% CI 1.3–1.4) for those exposed to one adversity to 1.6 (95% CI 1.6–1.7) and 2.0 (95% CI 1.9–2.1) for those exposed to 2 and 3 or more adversities, respectively.

Results for separate types of disorder (Supplementary Table 4) followed the same pattern, with risks being particularly increased for those exposed to more than 3 adversities (HR for substance misuse 2.8, 95% CI: 2.6–3.0; for psychotic disorder 4.0, 95% CI: 3.0–5.4; for mood disorders 2.1, 95% CI: 2.0–2.3, anxiety disorders 2.0, 95% CI: 1.8–2.1, and finally for ADHD 2.5, 95% CI: 2.2–2.9). However, cumulative exposure to CAs did not increase the risk of eating disorders.

Lastly, the timing of cumulative exposures appeared to have little effect on the risk of psychiatric disorder, regardless of type of disorder.

CAs increased the risk of psychiatric disorder in adulthood in the subset of study participants without childhood psychopathology and the risks were similar compared to the whole study population (sensitivity analyses, Supplement Tables 1–3). By contrast, most of the magnitudes of effects were lower in the subset of study participants with a history of childhood psychopathology.

The sensitivity analysis in which we examined the association

Table 3
Cox proportional Hazard Ratios (HR) with 95% confidence intervals (CI) for the associations between indicators of childhood adversity and types of psychiatric disorders after age 15 years.

Childhood adversity	Substance misuse		Psychotic disorders		Mood disorders		Anxiety spectrum disorders		Eating disorder		ADHD	
	Model I[a]	Model II[b]	Model I[a]	Model II[b]	Model I[a]	Model II[b]	Model I[a]	Model II[b]	Model I[a]	Model II[b]	Model I[a]	Model II[b]
Familial death	1.5 (1.3–1.6)	1.4 (1.2–1.5)	2.2 (1.5–3.2)	2.1 (1.4–3.2)	1.3 (1.1–1.4)	1.3 (1.1–1.4)	1.2 (1.1–1.3)	1.1 (1.0–1.3)	0.9 (0.7–1.3)	1.0 (0.8–1.4)	1.5 (1.2–1.8)	1.3 (1.1–1.6)
Parental substance misuse	2.4 (2.2–2.5)	2.1 (2.0–2.3)	2.1 (1.6–2.7)	1.9 (1.4–2.5)	1.6 (1.5–1.7)	1.5 (1.4–1.7)	1.6 (1.5–1.7)	1.5 (1.4–1.6)	1.0 (0.8–1.2)	1.0 (0.9–1.2)	2.0 (1.8–2.3)	1.6 (1.4–1.8)
Parental psychiatric disorder	2.0 (1.8–2.2)	1.8 (1.7–2.0)	2.9 (2.2–3.9)	2.6 (2.0–3.5)	1.9 (1.8–2.1)	1.8 (1.7–2.0)	1.7 (1.6–1.9)	1.6 (1.5–1.8)	1.1 (0.9–1.4)	1.1 (0.8–1.3)	1.9 (1.7–2.2)	1.5 (1.3–1.7)
Severe parental criminality	2.3 (2.1–2.4)	1.9 (1.8–2.1)	2.4 (1.8–3.3)	2.1 (1.5–2.9)	1.7 (1.5–1.8)	1.6 (1.4–1.7)	1.6 (1.5–1.7)	1.5 (1.4–1.6)	0.8 (0.7–1.1)	0.9 (0.7–1.2)	2.2 (1.9–2.6)	1.7 (1.5–2.0)
Parental separation[c]	1.4 (1.3–1.5)	1.3 (1.3–1.4)	1.8 (1.5–2.2)	1.7 (1.4–2.1)	1.4 (1.3–1.4)	1.3 (1.3–1.4)	1.3 (1.3–1.4)	1.3 (1.3–1.4)	1.1 (1.0–1.2)	1.1 (1.0–1.2)	1.5 (1.4–1.6)	1.4 (1.3–1.5)
Household public assistance[c]	2.1 (2.0–2.2)	2.0 (1.8–2.1)	2.3 (1.9–2.8)	2.1 (1.7–2.6)	1.6 (1.5–1.7)	1.6 (1.5–1.7)	1.7 (1.6–1.7)	1.6 (1.5–1.6)	0.8 (0.7–0.9)	0.9 (0.8–1.0)	2.2 (2.0–2.4)	1.8 (1.6–2.0)
Residential instability	1.5 (1.4–1.7)	1.4 (1.3–1.6)	2.1 (1.5–2.9)	2.0 (1.4–2.8)	1.3 (1.1–1.4)	1.2 (1.1–1.3)	1.3 (1.2–1.4)	1.2 (1.1–1.3)	0.8 (0.6–1.1)	0.8 (0.6–1.1)	2.1 (1.8–2.4)	1.7 (1.5–2.0)

Reference group: No childhood adversity.
[a] Adjusted for sex and birth year.
[b] Model I with additional adjustments for mother's country of birth, parental income and education, and own psychopathology before age 15.
[c] Data available from age 3.

between CAs and psychiatric disorder, excluding individuals with indications of parental psychiatric morbidity revealed that the exclusion of these individuals only marginally attenuated the association between CA and psychiatric disorders in young adulthood (data not shown).

The sensitivity analysis examining the potential effect modification of parental education revealed that parental educational level did not seem to modify the CA and young adulthood psychopathology association (data not shown).

4. Discussion

4.1. Key results

In this large cohort study of 107,704 young adults in Sweden, we found a significantly elevated risk of psychiatric disorder related to exposure to childhood adversity. All studied CA indicators appeared to increase the risk of psychiatric disorder regardless of timing for the exposure and the association persisted across different types of psychiatric disorders, including mood, anxiety, substance misuse and psychotic disorders and ADHD. By contrast, CA appeared unrelated to the risk of eating disorders. Lastly, the risk of psychiatric disorder grew higher with increasing numbers of adversities, regardless of timing for the cumulative exposure.

4.2. Comparison with other studies

Indications of at least one CA were common in our population. Over 40% were exposed to at least one adversity, and nearly 20% to two or more adversities. These proportions are consistent with prior research in various settings, both from the US and Europe (Anda, 2008; Björkenstam et al., 2013; Kessler et al., 2010; McLaughlin et al., 2010a). Our finding that CA increases the risk of psychiatric disorder is in agreement with several previous studies (Anda, 2008; Bellis et al., 2014; Björkenstam et al., 2013, 2015; Chapman et al., 2004; Dube et al., 2003; Kessler et al., 2010; McLaughlin et al., 2010a; Mersky et al., 2013; Slopen et al., 2014). However, most of these studies have been based on cross-sectional or retrospectively reported data (Anda, 2008; Bellis et al., 2014; Mersky et al., 2013; Sareen et al., 2013; Slopen et al., 2014), and long-term prospective studies have been warranted (Kessler et al., 2010; Sareen et al., 2013). We found that age of exposure to CA had no impact on the risk of psychiatric disorder in adulthood. This is in contrast to some earlier studies that found timing of CAs to be of importance for subsequent psychiatric morbidity (Andersen and Teicher, 2008; Heim and Binder, 2012). These studies have focused on adversities such as abuse and neglect that were not considered in the current study, which might explain the inconsistency with our results.

Furthermore, our large cohort enabled us to examine differential effects of CA with type of psychiatric disorders, including common mental disorders (mood and anxiety) but also substance misuse, psychosis and ADHD. A recent multinational retrospective investigation of CAs and adult psychopathology found little evidence of specificity of the effects of adversities with type of disorder (Kessler et al., 2010). This study included mood and anxiety disorders, substance misuse and behavioral disorders but not psychosis and eating disorders CA appeared to particularly increase the risk of psychosis and substance misuse, in our study, while the excess risk was somewhat lower for anxiety disorders. By contrast, we found no evidence of an association between CA and eating disorders. This is in discrepancy with an earlier study of a small sample from the US (Johnson et al., 2002). However, our finding is in line with a recent study from Mexico that found CAs not to be related to eating disorders, opposing results for other types for disorders (Benjet

Table 4
Cox proportional Hazard Ratios (HR) with 95% confidence intervals (CI) for the association between cumulative number of indicators of childhood adversity, categorized in exposure windows reflecting the age at exposure, and risk of psychiatric disorders after age 15 years.

Total number of childhood adversities	n (cases + non-cases)	Any psychiatric disorder	
		n (cases)	HR (95% CI)[a]
0	62,725	10,160	1 (REF)
1	26,499	5777	1.4 (1.3–1.4)
2	11,193	2913	1.6 (1.6–1.7)
3+	7287	2306	2.0 (1.9–2.1)
1 adversity occurring:			
0–6 years	14,118	3168	1.4 (1.3–1.5)
7–11 years	8813	1818	1.3 (1.2–1.3)
12–14 years	3568	801	1.4 (1.3–1.5)
2 adversities, whereof:			
Both occurring 0–6 years	4344	1103	1.6 (1.5–1.7)
Both occurring 7–11 years	1424	350	1.5 (1.3–1.7)
Both occurring 12–14 years	397	110	1.7 (1.4–2.1)
Both occurring in different periods	5028	1350	1.7 (1.6–1.8)
3+ adversities			
At least three occurring 0–6 years	2998	991	2.1 (2.0–2.2)
At least three occurring 7–11 years	505	166	2.1 (1.8–2.4)
At least three occurring 12–14 years	97	26	1.7 (1.1–2.5)
The indicators occurred in different periods	3687	1123	1.9 (1.8–2.0)

[a] Adjusted for sex, birth year, mother's country of birth, parental income and education, and own psychopathology before age 12.

et al., 2015).

Lastly, our study adds that recurrence of CA exposure rather than developmental timing is important for mental health later in life. This is consistent with some studies that found that cumulative exposure to CAs increases the risk of psychiatric disorders (Mersky et al., 2013). Accumulated exposure to CA increased the risk of psychosis by four-fold in our study, a finding that is in agreement with a recent Danish study on CA and the risk of psychosis (Trauelsen et al., 2015).

4.3. Interpretation and possible explanations for our findings

Exposure to CA is a major contributor to increased stress levels during childhood (Anda, 2008; Wadsworth and Butterworth, 2006). Earlier studies have found that environmental influences including emotional stress can have a major and long-lasting negative effect on the development of mental health (Schaffer and Kipp, 2014). From pregnancy throughout early adolescence, the environments in which children live and learn have a significant impact on their cognitive, emotional and social development. In particular, home and family environments and the characteristics of the parents and persons to whom children are exposed are powerful determinants of emotional and behavioral functioning later in life (Schaffer and Kipp, 2014). Researchers in the field of psychology suggest that the stress that comes with growing up in a dysfunctional household may lead to lower self-esteem and lower self-worth, which in turn may increase the risk for psychiatric disorder (Dube et al., 2003; Wadsworth and Butterworth, 2006). Growing up in an adverse family environment may lead to elevated stress reactivity that in turn has been shown to predict psychiatric disorder (McLaughlin et al., 2010b). The allostatic load theory suggests CA to be associated with enduring changes in the nervous, endocrine and immune systems; changes that in turn may lead to allostatic overload, resulting in adverse health outcomes and psychiatric disorder (Beckie, 2012; Danese and McEwen, 2012).

CAs were more common among children with lower socioeconomic background in our study, and lower socioeconomic background is strongly associated with psychiatric disorder (Kosidou et al., 2011). However, CA remained strongly associated with development of psychiatric disorders in young adulthood after we adjusted for parental education and income, suggesting that only a small part of the association is confounded by parental socioeconomic background.

Furthermore, adjustment for childhood psychopathology only slightly attenuated the association of CA with psychiatric disorder in adulthood and effects of CAs were similar in those with onset of disorder in adulthood compared to the whole study population. These findings support the long term effects of CA on mental health, perhaps through enduring neurocognitive changes, heightened stress sensitivity and a higher probability of negative events also in adult life. The association of CA with adult psychiatric disorder was weaker in those experiencing childhood psychopathology. This is somewhat expected since childhood psychopathology is a strong competing risk factor for psychiatric disorder in adulthood, obscuring the effect of other risk factors.

The relatively low specificity of the effects of CA by type of psychiatric disorder in our study suggests the existence of general causal pathways relating CA to disorders, including elevated stress during neurodevelopment and the long-term effects of CA on cognition and behavior. However, the risk was particularly increased for psychosis and substance misuse, indicating that childhood adversities may be particularly detrimental for the development of this type of disorders. Interestingly, eating disorders appear to not to be related to CA, a finding that indicates partly differential etiology for these disorders.

The studied adversities examined in the current study reflect social disadvantage in childhood, though may also capture a degree of genetic susceptibility to morbidity. It is difficult to disentangle hereditary risk for mental disorder from social disadvantage in childhood. It is also well-known that the presence of a family history of psychiatric morbidity is a predictor of offspring psychiatric morbidity (Siegenthaler et al., 2012). In an attempt to take genetic factors into account, we repeated the analyses excluding individuals with history of parental psychiatric inpatient care, which, however, yielded little effect on risk estimates.

4.4. Implications

The results presented in this study show that exposure to CA is common. Our findings give further support to the research underlining the importance of the childhood environment for mental

health later in life. The fact that CAs often co-occur has important implications for intervention because it means that prevention of single adversities among individuals exposed to many is unlikely to have important effects. Several studies have stressed the importance of preventive interventions (Cuijpers et al., 2011; Dube et al., 2003; Kessler et al., 2010; Slopen et al., 2014; Trauelsen et al., 2015). From a practice perspective, detecting CAs early is crucial in order to determine whether children exposed to stressful environments are on a trajectory that could lead to increased risk for later psychiatric disorder. Extended support to children growing up in adversity should be part of any strategy aiming for reducing the effects they have on mental health outcomes. If detected early enough, the impact of CA on mental health can be reversed, or at least attenuated. Early intervention to reduce exposure to multiple CAs, and later intervention to address long-term adult negative psychological consequences of having been exposed to CAs would seem to hold the most promise. It has been argued that the public health implications of CA are greater for primary than for secondary prevention because the associations of CA with disorder onset are much stronger than the associations with persistence (Cuijpers et al., 2011; McLaughlin et al., 2010a).

4.5. Strengths and limitations

This study has several methodological strengths, including the longitudinal population-based design, use of national registers with high completeness and no loss to follow up. The large cohort size allowed for detailed analyses demonstrating the cumulative effect of childhood adversity on specific types of psychiatric disorder. Other quantitative studies with a similar design and research questions, but with other kinds of data sources often struggle with insufficient power and missing data that due to the sensitive nature of the topic might be substantial. Also, we considered timing and persistence of exposure, information that many studies lack. The study also has some methodological weaknesses, some of which have been addressed above. Relying solely on register data, there are CAs that we were not able to study, including witnessing domestic violence. Another limitation is that, with respect to treatment-seeking, we do not know whether the associations between CA and psychiatric disorder vary among those who seek treatment and those who do not. Also, it has been shown that socioeconomic factors play a part in treatment-seeking (Kosidou et al., 2011), but also with respect to psychiatric morbidity, such that lower socioeconomic background is a risk factor for most psychiatric disorders. In Sweden, lower socioeconomic background predicts higher mental health service use (Kosidou et al., 2011), which is probably due to the higher psychiatric morbidity in this population. If the associations between CA and psychiatric disorder vary among those who seek treatment and those who do not, this might have biased our results. With respect to follow-up, the youngest individuals were followed until age 20 years and the oldest until age 24 years. Although many psychiatric disorders are known to have their onset in adolescence, certain disorders including psychosis typically have their onsets later (Kessler et al., 2007). Thus, the younger birth cohorts may include individuals who have not yet received a diagnosis although they in fact suffer from a psychiatric disorder. This potential outcome misclassification would lead to underestimation of the true risk. Another limitation is that we have not examined the fluidity of CAs but rather, as done by others (Felitti and Anda, 2010) treated them as discrete life events. Probably the most decisive limitation is the lack of data on child maltreatment (abuse and neglect). Swedish registers do not contain this kind of information. This effectively excludes possibilities to study the interaction of childhood adversities and child maltreatment, related to different age stages during childhood.

5. Conclusion

In conclusion, our findings support the long-term negative impact of childhood adversity on mental health, particularly when accumulating. It also demonstrates that, regardless of when in childhood exposure to CA occurs it is detrimental for the child's subsequent health. Given that experience of CA is common, early and efficient detection of CA is of importance for preventing interventions targeted to improve long term mental health outcomes among disadvantaged children.

Ethical considerations

This study is approved by the ethical committee in Stockholm, Sweden (dnrs: 2010/1185-31/1 and 2013/1118-32).

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Conflicts of interest

On behalf of all authors, the corresponding author states that there is no conflict of interest.

Contributors

Emma Björkenstam, and Kyriaki Kosidou originated the idea. Emma Björkenstam analyzed the data and wrote the manuscript draft. All authors contributed to the interpretation of the results and to the writing of the final article.

Appendix A. Supplementary data

Supplementary data related to this article can be found at http://dx.doi.org/10.1016/j.jpsychires.2016.02.018.

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