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Parental substance use disorder and offspring not in education, employment or training: a national cohort study of young adults in Sweden

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Abstract

Aims: To test the hypothesis that exposure to parental substance use disorder is associated with an increased risk of being not in education, employment or training (NEET) in male and female offspring during young adulthood.

Design, setting and participants: A register-based, national cohort study of 797 376 individuals born between 1984 and 1990, residing in Sweden at age 17 years. Participants were followed from age 17 years to maximum age 32 years and assessed annually for being NEET.

Measurements: The exposure variable was binary, defined as any diagnosis of substance use disorder (alcohol and/or drug use disorder) in one or both parents, measured between offspring's birth and age 17 years. Cox regression analysis was used to obtain hazard ratios (HRs) for being NEET, assessed annually as a binary variable using income and employment data.

Findings: We found that 4.4% of individuals were exposed to parental substance use disorder. When adjusted for birth year, domicile, origin, psychiatric diagnosis, household income and parental psychiatric diagnosis, HRs for being NEET were HR = 1.13 (95% CI 1.09–1.16) for males, and HR = 1.15 (95% CI 1.12–1.19) for females. When stratified by age, adjusted HRs for experiencing the first episode of NEET peaked at age 17–19 years, HR = 1.37 (95% CI 1.25–1.50) for males, and HR = 1.31 (95% CI 1.18–1.44) for females.

Conclusions: In Sweden, exposure to parental substance use disorder before age 17 years is associated with increased risk of being not in education, employment or training during early adulthood. The risks were highest at age 17–19 years for both males and females, decreasing with greater age.

KEYWORDS
Alcohol use disorder, drug use disorder, labour market participation, NEET, parenting, substance use disorder

INTRODUCTION

Substance use disorder (SUD)—encompassing both drug and alcohol use disorders—is a common and debilitating condition that has negative consequences for individuals, families and communities [1]. Parental SUD has been associated with several negative outcomes among offspring, such as psychiatric disorders, including drug use disorder (DUD) and suicide [2–6].

Moreover, children who grow up with parental SUD may face disadvantages when they attempt to join higher education or the labour market. For example, a previous cohort study in Sweden reported that the children of parents with alcohol use disorder (AUD) achieved lower grades in school at age 15–16 years than their peers [7]. This is concerning, given that low education is strongly associated with youth unemployment, negative later life trajectories and adverse health outcomes [8–10]. It has also been suggested that parental SUD is detrimental to parent–child relationships and can mean that children are exposed to less positive socio-environmental contexts [7]. Parenting and the household environment are crucial for early cognitive development, as well as health, education and social outcomes in later life [11–18].

The concept of being 'not in education, employment, or training' (NEET) emerged in the United Kingdom in the 1990s [19]. NEET refers to a heterogeneous group of young people: those who are unemployed and inactive, not enrolled in any formal or non-formal education, affected by chronic illness or otherwise not available for work [20]. On average, across OECD (Organization for Economic Co-operation and Development) countries, the frequency of being NEET has decreased among 20–24-year-olds from almost 19% in 2009 to approximately 15% in 2019 [21]. In Sweden, the prevalence of NEET in 2019 among 15–29-year-olds was approximately 7% [21]. As in many OECD countries, migrant youths are more often NEET compared with their native peers [22–24]. Unlike in many other high-income countries, females in Sweden are no more likely to be NEET than males [24]. However, the odds of becoming NEET are substantially higher for young females with a child aged below 5 years than for males in the same position [8]. Gender may thus be particularly important to the risk of being NEET during young adulthood. Having poor self-perceived health and symptoms of poor mental health have also been associated with increased odds of being NEET [8, 25–27]. Additionally, the prevalence of NEET is higher in socio-economically disadvantaged neighbourhoods [8, 28].

The risks associated with being NEET may fluctuate among different stages of early adulthood. Individuals who have neither completed high-school education, which typically concludes at age 19–20 years in Sweden, nor transitioned into the labour market, may be at increased risk of being NEET repeatedly and experiencing mental health problems [29]. It is possible that, after completing high-school or university education, many choose to take a break for personal reasons, such as overseas travel []. For these individuals, being NEET may be voluntary in nature, or serve as a stepping-stone to labour-market attachment.

Overall, parental SUD is suggested to increase the risk of negative outcomes among offspring [7]. The impact of parenting and family relationships has been reported to peak before late adolescence (15–19 years) [30]. Given that the transition from school into work or further education is crucial for later life outcomes, it is important to understand whether or not exposure to parental SUD is associated with an increased risk of being NEET [30–33].

We therefore aimed to test the following hypotheses: (1) that exposure to parental SUD during childhood is associated with an increased risk of being NEET in male and female offspring during young adulthood and (2) that these risks would be greatest prior to age 20 years.

METHODS

Study population

This was a register-based, longitudinal cohort study of 797 376 individuals who were born between 1984 and 1990 and were alive and residing in Sweden on their 17th birthday. Participants were followed-up from age 17 years to a maximum age of 32 years (range = 26–32 years) and assessed annually, between January 2001 and December 2016, for the study outcome (NEET). Participants were censored at the first recorded episode of NEET, in the event that three or more consecutive years of income data were missing (due to possible emigration, $n = 29\,615$), at death ($n = 2107$) or at the end of the follow-up period on 31 December 2016.

Sweden's national registers provide individual-level, sex-disaggregated, longitudinal population data that have been reported to offer high internal validity and completeness across multiple domains [34]. We used an existing database ('Psychiatry Sweden'), which comprises the total population of Sweden (including migrants with resident permits) born 1932–2016. We utilized data linked from the following national registers: the Multi-Generation Register, the Longitudinal Integration Database for Health Insurance and Labour Market Studies (LISA), the cause of death register, STATIV (a longitudinal database for integration studies) and the National Patient Register, which includes all in- and outpatient psychiatric care since 1973. Missing baseline data for household income and domicile comprised less than 4% of the study population.

This study adhered to the Reporting of Observational Studies in Epidemiology (STROBE) statement (Supporting information, Table S1). As the study protocol was not pre-registered, our analyses should be considered exploratory in nature. The Regional Ethics Committee in Stockholm approved the study before any records were linked (decision number: 2016/987–32).

Exposure

The exposure variable was parental SUD. This was measured between offspring's birth and seventeenth birthday and defined as any

diagnosis of AUD or DUD in one or both biological parents. Definitions of AUD and DUD were based on the 8th, 9th and 10th editions of the World Health Organization's International Classification of Diseases (ICD-8: 291, 303, 304; ICD-9: 291, 292, 303, 304, 305, 967, 969, 980; and ICD-10: F10–F19). Exposure to parental SUD was coded in binary format using a dummy variable with the value 1 (exposed) or 0 (not exposed).

Outcome

The outcome variable was offspring NEET, defined by the Swedish Agency for Youth and Civil Society (MUCF) as living and registered in Sweden for an entire calendar year with annual income below the price basic amount—a national statistic calculated annually from the consumer price index—while not receiving study grants or being registered for more than 60 hours of education [35]. We hypothesized that this definition of NEET would introduce an optimal balance of sensitivity and specificity to identify those at risk of the negative outcomes associated with NEET, while minimizing the incorrect labelling of individuals who are on short-term study breaks, working holidays, parental leave or sabbaticals. Outcome status was coded in binary format as a dummy variable with the value 1 (one or more episodes of NEET) or 0 (no episodes of NEET).

Covariates

We included the following covariates, shown to be associated with parental SUD and negative consequences in offspring [7, 36, 37], and available in our data.

Individual factors included sex (male or female), birth year and domicile, which consisted of three categories, according to the Swedish Association of Local Authorities and Regions: large cities with populations of at least 200 000 people, medium-sized towns with at least 50 000 inhabitants and small towns/rural areas. Origin was categorized into three groups: (i) native Swedish, referring to individuals born in Sweden with both parents born in Sweden; (ii) offspring of migrants, referring to individuals born in Sweden with at least one parent born abroad; and (iii) migrants, defined as individuals born outside Sweden with both parents also born abroad. Psychiatric diagnosis was captured as a binary variable from birth until 17 years of age. This referred to any psychiatric diagnosis (ICD-10, F01–F99) registered in in- or outpatient health care.

Parental factors included parental psychiatric diagnosis, a binary variable defined as any psychiatric diagnosis other than SUD (ICD-8: 290, 292–302, 305–315; ICD-9: 290, 293–302, 306–319 and ICD-10: F01–F09, F20–F99) in one or both parents, before the offspring reached age 17 years, and household income, which was defined as total annual household income for the study population at offspring age 16 years. This was converted into quintiles, where the lowest quintile represented the lowest income group. A time-line showing the measurement of study variables is available in the Supporting information, Fig. S1.

Statistical analyses

To test our main hypothesis, analyses were based on person-time measured from 1 January 2001 to whichever occurred first: the first recorded episode of NEET, death or the end of the follow-up period on 31 December 2016. First, we compared the incidence of NEET by socio-demographic characteristics of the study population from age 17 years to maximum age 32 years. Results were presented as incidence rates per 100 000 person-years with 95% confidence intervals (CIs). We then used Cox's regression analyses of person-years, with age as the underlying time-scale, to estimate univariate hazard ratios (HRs) of NEET. Thirdly, we examined adjusted HRs for the first episode of being NEET. Results were presented in four different models, as HRs with 95% CIs: model 1 adjusted for birth year, domicile and origin; model 2 added psychiatric diagnosis; model 3 added household income; and model 4 adjusted for all aforementioned variables and other parental psychiatric diagnosis. All analyses were stratified by sex. To test our second hypothesis, we examined HRs for the first episode of NEET at ages 17–19, 20–22, 23–25 and 26–32 years. All models were tested for proportional hazards using Schoenfeld residuals. The assumption of proportional hazards was fulfilled in the general test. Stata version 16 MP was used for all statistical analyses. The data sets analysed for the current study are not publicly available due to Swedish data protection laws that restrict public sharing of data.

Sensitivity analyses

To further assess the association between parental SUD and the risk of offspring NEET in native Swedish offspring, offspring of migrants and migrants, we tested interaction effects among males and females. On the basis of significant interaction effects ($P < 0.05$), we stratified the analyses between exposure to parental SUD and the first episode of NEET by origin. In addition, to further assess the association between exposure to parental SUD and the risk of NEET, we tested interaction effects for offspring psychiatric diagnosis and parental psychiatric diagnosis among males and females. Based on significant interaction effects ($P < 0.05$), we stratified our analyses by offspring psychiatric diagnosis and parental psychiatric diagnosis in both males and females.

RESULTS

Among 797 376 individuals included in the analyses, 48.5% were female (Table 1). Just fewer than half of the study population resided in medium-sized towns, approximately 30% in large cities and the rest

TABLE 1 Socio-demographic and clinical characteristics of the study population, by parental substance use disorder (SUD) exposure (n = 797 376)

Socio-demographic and clinical characteristics	Total (%)	Exposure to parental SUD before age 17 years, n (%)	
		No parental SUD	Any parental SUD
All individuals	797 376 (100)	762 434 (95.6)	34 942 (4.4)
Sex			
Male	410 081 (51.4)	392 224 (51.4)	17 857 (51.1)
Female	387 295 (48.6)	370 210 (48.6)	17 085 (48.9)
Birth year			
1984	100 544 (12.6)	95 966 (12.6)	4578 (13.1)
1985	105 144 (13.2)	100 459 (13.2)	4685 (13.4)
1986	108 598 (13.6)	103 901 (13.6)	4697 (13.4)
1987	111 236 (14.0)	106 606 (14.0)	4630 (13.3)
1988	119 019 (14.9)	113 977 (15.0)	5042 (14.4)
1989	122 506 (15.4)	116 991 (15.3)	5515 (15.8)
1990	130 329 (16.3)	124 534 (16.3)	5795 (16.6)
Domicile			
Large city	229 131 (29.7)	218 643 (29.7)	10 488 (30.5)
Medium-sized town	361 524 (46.9)	345 588 (46.9)	15 936 (46.3)
Smaller town/rural area	180 861 (23.4)	172 859 (23.5)	8002 (23.2)
Origin			
Native Swedish	605 909 (76.0)	580 110 (76.1)	25 799 (73.8)
Offspring of migrants	132 812 (16.7)	125 100 (16.4)	7712 (22.1)
Migrants	58 655 (7.3)	57 224 (7.5)	1431 (4.1)
Psychiatric diagnosis			
No	777 168 (97.5)	744 397 (95.8)	18 037 (89.3)
Yes	20 208 (2.5)	32 771 (4.2)	2171 (10.7)
Parental psychiatric diagnosis			
No	751 728 (94.3)	744 740 (97.7)	6988 (20.0)
Yes	45 648 (5.7)	17 694 (2.3)	27 954 (80.0)
Household income (quintiles)			
1 (low income)	154 091 (20.0)	139 789 (19.0)	14 302 (41.5)
2	153 962 (20.0)	145 563 (19.8)	8399 (24.4)
3	154 005 (20.0)	149 171 (20.3)	4834 (14.0)
4	154 012 (20.0)	150 183 (20.4)	3829 (11.1)
5 (high income)	153 900 (20.0)	150 824 (20.5)	3076 (8.9)

in smaller towns or rural areas. Migrants and offspring of migrants accounted for approximately 7 and 17% of the total study population, respectively. In total, 34 942 (4.4%) individuals were exposed to parental SUD. Among individuals who were exposed to parental SUD, approximately 80% were exposed to an additional parental psychiatric diagnosis. In comparison, among the study population who were not exposed to parental SUD, just 2.3% were exposed to any other parental psychiatric diagnosis. Approximately 41% of offspring exposed to parental SUD were in the lowest income quintile compared to approximately 18% of those who were not exposed.

Incidence rates (IRs) of NEET were higher among males (6390, 95% CI = 6260–6524) and females (7086, 95% CI = 6945–7229) who

were exposed to parental SUD than among those who were not (Table 2). In general, IRs of NEET were higher among young adults with a psychiatric diagnosis or a low family income and among females from smaller towns or rural areas.

Overall, the risk of being NEET was greater among males and females who were exposed to parental SUD than among individuals who were not (Table 3). After adjusting for year of birth, domicile and origin (model 1), HRs were 1.46 (95% CI = 1.42–1.48) among males exposed to parental SUD and 1.45 (95% CI = 1.42–1.47) among females exposed to parental SUD. When psychiatric diagnosis was taken into account (model 2), these estimates were attenuated. HRs were further attenuated when adjusting for household income (model

TABLE 2 Incidence rates (IRs) of being not in education, employment or training (NEET) among males and females, by parental substance use disorder (SUD) exposure, per 100 000 person-years, 2001–16 (n = 797 376)

| | IR (95% CI) of NEET per 100 000 person-years | | | |
| | Female (n = 387 295) | | Male (n = 410 081) | |
Socio-demographic characteristics	No parental SUD	Any parental SUD	No parental SUD	Any parental SUD
All individuals	4865 (4842–4889)	7086 (6945–7229)	4251 (4230–4273)	6390 (6260–6524)
Origin				
Native Swedish	4698 (4673–4725)	7006 (6844–7171)	3925 (3903–3949)	6181 (6032–6334)
Offspring of migrants	5760 (5662–5859)	7044 (6382–7774)	5733 (5639–5829)	7161 (6496–7895)
Migrants	5301 (5240–5362)	7371 (7065–7690)	5234 (5175–5296)	6972 (6680–7278)
Domicile				
Large city	4656 (4613–4699)	6498 (6259–6746)	4497 (4456–4539)	6310 (6075–6554)
Medium-sized town	5052 (5016–5079)	7511 (7295–7733)	4342 (4311–4375)	6706 (6507–6915)
Smaller town/rural area	5252 (5200–5303)	7473 (7174–7785)	4139 (4096–4184)	6372 (6100–6656)
Parental psychiatric disorder				
No	4830 (4807–4854)	7340 (7022–7672)	4216 (4194–4237)	6174 (5891–6470)
Yes	6548 (6362–6739)	7023 (6868–7183)	5927 (5755–6104)	6645 (6299–6597)
Psychiatric diagnosis				
No	4787 (4764–4810)	6819 (6677–6963)	4186 (4165–4208)	6175 (6044–6309)
Yes	8779 (8558–9007)	12 043 (11 252–12 889)	8624 (8375–8880)	12 120 (11 199–13 117)
Household income (quintiles)				
1 (low income)	6669 (6602–6737)	8325 (8081–8576)	5974 (5911–6037)	7625 (7394–7864)
2	5732 (5673–5791)	7127 (6841–7424)	5020 (4966–5074)	6351 (6090–6623)
3	4929 (4877–4982)	6352 (6011–6714)	3879 (3834–3925)	5427 (5119–5754)
4	4302 (4253–4351)	5887 (5520–6280)	3597 (3554–3641)	5326 (4984–5692)
5 (high income)	3606 (3562–3651)	5471 (5083–5891)	3579 (3536–3622)	5447 (5054–5871)

CI = confidence interval.

TABLE 3 Cox regression models for being not in education, employment or training (NEET), by parental substance use disorder (SUD) exposure, stratified by sex, 2001–16 (n = 797 376)

| | NEET at age 17–32 years | HR (95% CI) | | | | |
Parental SUD exposure	Total	Univariate	Model 1	Model 2	Model 3	Model 4
Males						
No parental SUD	152 610	1	1	1	1	1
Any parental SUD	9020	1.48 (1.45–1.51)	1.46 (1.42–1.48)	1.43 (1.39–1.45)	1.28 (1.26–1.31)	1.13 (1.09–1.16)
Females						
No parental SUD	166 595	1	1	1	1	1
Any parental SUD	9656	1.47 (1.44–1.50)	1.45 (1.42–1.47)	1.41 (1.38–1.44)	1.27 (1.24–1.29)	1.15 (1.12–1.19)

HR = hazard ratio; CI = confidence interval.
Model 1 adjusted for birth year, domicile and origin.
Model 2 adjusted for birth year, domicile, origin and psychiatric diagnosis.
Model 3 adjusted for birth year, domicile, origin, psychiatric diagnosis and family household income.
Model 4 adjusted for birth year, domicile, origin, psychiatric diagnosis, family household income and parental psychiatric diagnosis.

3). Lastly, when other parental psychiatric diagnosis was also considered (model 4), HRs decreased to 1.13 (95% CI = 1.09–1.16) for males and 1.15 (95% CI = 1.12–1.19) for females.

In estimating the HRs of NEET within different age groups we found that the hazards decreased with older age, both in univariate analyses and in adjusted models (Tables 4–5). In model 4, among

TABLE 4 Cox regression models for being not in education, employment or training (NEET) among males, by parental substance use disorder (SUD) exposure, stratified by age group, 2001–16 (n = 410 081)

Parental SUD exposure Males	First episode of NEET at age 17–32 years Total	HR (95% CI)				
		Univariate	Model 1	Model 2	Model 3	Model 4
Age 17–19 years						
No parental SUD	22 789	1	1	1	1	1
Any parental SUD	2180	2.12 (1.99–2.26)	2.17 (2.04–2.31)	1.95 (1.83–2.08)	1.58 (1.48–1.68)	1.37 (1.25–1.50)
Age 20–22 years						
No parental SUD	92 810	1	1	1	1	1
Any parental SUD	5224	1.43 (1.38–1.48)	1.40 (1.35–1.45)	1.38 (1.33–1.43)	1.24 (1.20–1.28)	1.08 (1.02–1.13)
Age 23–25 years						
No parental SUD	26 521	1	1	1	1	1
Any parental SUD	1274	1.36 (1.28–1.46)	1.34 (1.26–1.44)	1.33 (1.24–1.42)	1.21 (1.13–1.29)	1.09 (1.00–1.20)
Age 26–32 years						
No parental SUD	1659	1	1	1	1	1
Any parental SUD	758	1.32 (1.21–1.43)	1.29 (1.19–1.40)	1.27 (1.17–1.38)	1.22 (1.12–1.32)	1.09 (0.97–1.22)

HR: hazard ratio; CI: confidence interval.
Model 1 adjusted for birth year, domicile and origin.
Model 2 adjusted for birth year, domicile, origin and psychiatric diagnosis.
Model 3 adjusted for birth year, domicile, origin, psychiatric diagnosis and family household income.
Model 4 adjusted for birth year, domicile, origin, psychiatric diagnosis, family household income and parental psychiatric diagnosis.

TABLE 5 Cox regression models for being not in education, employment or training (NEET) among females, by parental substance use disorder (SUD) exposure, stratified by age group, 2001–16 (n = 387 295)

Parental SUD exposure Females	First episode of NEET at age 17–32 years Total	HR (95% CI)				
		Univariate	Model 1	Model 2	Model 3	Model 4
Age 17–19 years						
No parental SUD	18 848	1	1	1	1	1
Any parental SUD	1807	2.18 (2.04–2.33)	2.22 (2.08–2.38)	1.97 (1.84–2.11)	1.60 (1.49–1.70)	1.31 (1.18–1.44)
Age 20–22 years						
No parental SUD	73 914	1	1	1	1	1
Any parental SUD	4576	1.52 (1.47–1.59)	1.50 (1.44–1.55)	1.46 (1.41–1.51)	1.31 (1.26–1.36)	1.16 (1.10–1.22)
Age 23–25 years						
No parental SUD	36 245	1	1	1	1	1
Any parental SUD	1904	1.40 (1.32–1.48)	1.38 (1.31–1.46)	1.36 (1.28–1.43)	1.22 (1.15–1.29)	1.12 (1.03–1.21)
Age 26–32 years						
No parental SUD	41 742	1	1	1	1	1
Any parental SUD	1741	1.17 (1.11–1.23)	1.15 (1.09–1.21)	1.14 (1.08–1.20)	1.07 (1.02–1.13)	1.06 (1.00–1.14)

HR = hazard ratio; CI = confidence interval.
Model 1 adjusted for birth year, domicile and origin.
Model 2 adjusted for birth year, domicile, origin and psychiatric diagnosis.
Model 3 adjusted for birth year, domicile, origin, psychiatric diagnosis and family household income.
Model 4 adjusted for birth year, domicile, origin, psychiatric diagnosis, family household income and parental psychiatric diagnosis.

those exposed to parental SUD, the HRs for NEET were highest at age 17–19 years in both males (1.37, 95% CI = 1.25–1.50) and females (1.31, 95% CI = 1.18–1.44). The HRs for males and females decreased to 1.08 (95% CI = 1.02–1.13) and 1.16 (95% CI = 1.10–1.22) at age 20–22 years and 1.09 (95% CI = 1.00–1.20) and 1.12 (95% CI = 1.03–1.21) at age 23–25 years, respectively, after adjusting for year of birth, domicile, origin, psychiatric diagnosis, household income and other parental psychiatric diagnosis. Exposure to parental SUD prior to age 17 years was not associated with a first episode of NEET among 26–32-year-olds.

Our sensitivity analyses (Supporting information, Table S2) suggested that the association between exposure to parental SUD and the risk of offspring being NEET was greater among native Swedish individuals than among migrants and offspring of migrants. In addition, the association between exposure to parental SUD and the risk of NEET was similar among offspring who had a psychiatric diagnosis and those who did not (Supporting information, Table S3). However, the relative impact of parental SUD on offspring's risk of being NEET was higher among those with parents without any additional psychiatric diagnosis (Supporting information, Table S4).

DISCUSSION

We found that exposure to parental SUD prior to age 17 years was associated with an increased risk of being NEET during young adulthood among both male and female offspring. This increased risk peaked at around age 17–19 years, then decreased gradually. Our findings showed that these increased risks of being NEET were partially, but not fully, attenuated after adjusting for birth year, domicile, origin, psychiatric diagnosis, household income and other parental psychiatric diagnosis.

While we are not aware of any previous study of parental SUD and offspring risk of being NEET, the overarching importance of parenting is well documented [11, 14, 17, 30, 38]. A previous Swedish cohort study reported that children exposed to parental AUD achieved lower grades in school than their peers [7]. Further, cross-sectional data from across the European Union suggest that low education is strongly associated with being NEET [19, 39]. In addition, a longitudinal study of adopted and non-adopted offspring of parents with DUD in Sweden suggested that high-quality child-rearing environments can substantially reduce the risk of DUD, even among offspring at high genetic risk [2]. Overall, our findings corroborate existing literature that highlights the importance of parenting and suggests that exposure to parental SUD may have a psychosocial 'scarring' effect on offspring [18, 40].

Our finding that the association between exposure to parental SUD and offspring being NEET is strongest at age 17–19 years is consistent with previous reports that the impact of parenting and other family relationships peaks during adolescence [30]. Adolescents, who are still developing their capacity for autonomy, must negotiate a transitional period of labour-market integration that may involve precarious employment, low wages and other challenges [24, 41, 42]. Previous longitudinal research has suggested that being NEET during this stage may increase the risk of later NEET episodes [29]. Importantly, in the Swedish context, being NEET at this age implies that an individual has not completed secondary school, which is likely to have negative implications and increase the risk of being NEET later in life [8, 19, 39]. While our findings suggest that age 17–19 years is a particularly important window for vocational engagement among individuals who are exposed to parental SUD, it should be noted that these results corresponded only to the first episode of being NEET. It is therefore likely that lower HRs reported among older age groups may

be partly explained by the fact that individuals were censored at the first instance of being NEET, as later occurrences of the same individuals being NEET were not included.

In addition, our findings suggest that the risks of being NEET were greatly reduced after adjusting for household income and, in particular, other parental psychiatric disorders. One previous cohort study reported that additional parental psychiatric disorders may partially explain the association between parental AUD and educational disadvantage among offspring [7]. Other studies have reported associations between socio-economic status (SES), SUD and being NEET [43–49]. Our findings were consistent with those reports, adding evidence that other parental psychiatric disorders and low household income may contribute to the association found between parental SUD and offspring risk of NEET.

Regarding the importance of origin in this association, a lower prevalence of SUD has previously been reported among migrants in Sweden, along with an increased prevalence of being NEET [22–24, 50, 51]. While the hazards of being NEET in our study were similar after adjustments for origin, there are likely to be other confounding factors involved in this relationship. For example, parental SUD is likely to interact with many different risk factors linked to both migration and being NEET (such as adverse life experiences, family structures, neighbourhood deprivation, etc.). Moreover, in our study parental SUD referred to individuals who had been in contact with specialized health-care and had thus been formally diagnosed. Among those facing barriers to seeking or receiving health-care in Sweden, such as migrants, psychiatric diagnoses made in the country of origin were not necessarily recorded in the Swedish registers [52].

Strengths and limitations

This was, to our knowledge, the first study to investigate the association between parental SUD and offspring being NEET. Crucially, a longitudinal design ensured that all exposures to parental SUD occurred before assessment of NEET began. Other methodological strengths within our study are related to the use of register data. Sweden's health registers provide individual-level, sex-disaggregated data with a high level of completeness and internal validity across multiple domains [34]. The study included a national cohort of all individuals born between 1984 and 1990, ensuring a large study population and minimizing any issues of sample representativeness or selection bias.

Despite these strengths, our study had important limitations. The primary limitation was that the study data did not satisfy the assumptions necessary for causal inference. In particular, there is a strong possibility for both residual confounding and unmeasured confounders. Possible confounding effects of parental separation, childhood trauma, adoption and early parenthood, for example, must be considered, but such data were not available for analysis. Furthermore, we recognize that the relationship between SUD and other psychiatric disorders is complex, and that these disorders may be

mutually exacerbating [53]. Similarly, household income may be affected by parental SUD, rather than operating strictly as a confounder. It is therefore possible that adjusting for these covariates may have introduced bias. Given this potential overadjustment, it may be that our maximally adjusted models underestimated the risks associated with parental SUD.

The use of register data is not without limitations. Psychiatric disorders tend to be underestimated within Swedish registries [54]. The reliance upon formally diagnosed SUD and other psychiatric disorders is likely to have resulted in an underestimation of actual cases. Additionally, undocumented migrants, who are generally vulnerable to being NEET, were not included in this study [22–24]. Importantly, these data only include biological parents and not adoptive or step-parents. Furthermore, cases in which birth parents do not have custody of—or indeed contact with—their child, were not differentiated from other households in this study.

Lastly, the MUCF definition of NEET used in this study may have introduced heterogeneity within the NEET population. For example, professionals who worked abroad for periods of up to 3 years at a time—and thus paid taxes overseas—may have been recorded as NEET. The characteristics of these individuals are likely to have differed considerably from those who neither worked nor studied during the study period. A further limitation was that the applied definition did not consider the underlying reason for being NEET. Thus, individuals who were voluntarily NEET were not differentiated from those who truly experienced labour-market disengagement. This is not necessarily the case in other OECD countries, where self-reported measures of NEET are used [19, 24]. Furthermore, our study did not differentiate individuals who accumulated multiple episodes of NEET from those who experienced a single episode. However, no consensus definition of NEET has been established to date [23, 55, 56]. Thus, it is likely that subgroups with increased vulnerability for the adverse outcomes associated with being NEET exist within the wider NEET population.

CONCLUSIONS

Our study found that exposure to parental SUD during childhood was associated with an increased risk of being NEET during young adulthood. This risk was highest at age 17–19 years in both males and females, but decreased with older age. The risks of being NEET were substantially reduced when household income and, in particular, other parental psychiatric diagnosis were adjusted for. We suggest that children who are exposed to parental SUD may require additional support, and as they approach the transitional period may be targeted for evidence-based programmes to improve labour market participation.

ACKNOWLEDGEMENT

AUTHOR CONTRIBUTIONS

Paul Welford: Conceptualization; data curation; formal analysis; investigation; methodology; project administration; resources; software; validation. **Anna-Karin Danielsson:** Conceptualization; funding acquisition; investigation; methodology; project administration; resources; supervision; validation. **Helio Manhica:** Conceptualization; data curation; formal analysis; funding acquisition; investigation; methodology; project administration; resources; software; supervision.

DECLARATION OF INTERESTS
None.

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