Published in final edited form as:
Br J Psychiatry. 2014 October ; 205(4): 286–290. doi:10.1192/bjp.bp.113.136200.

Childhood family income, adolescent violent criminality and substance misuse: A quasi-experimental total population study

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Abstract

Background—Low socioeconomic status in childhood is a well-known predictor of subsequent criminal and substance misuse behaviors but the causal mechanisms are questioned.

Aims—To investigate whether childhood family income predicts subsequent violent criminality and substance misuse and whether the associations are in turn explained by unobserved familial risk factors.

Method—Nationwide Swedish quasi-experimental, family-based study following cohorts born 1989–1993 (n_{total}=526,167; $n_{cousins}$=262,267; $n_{siblings}$=216,424) between ages 15–21 years.

Results—Children of parents in the lowest income quintile experienced a seven-fold increased hazard rate of being convicted of violent criminality compared to peers in the highest quintile (HR=6.84, 95% CI: 6.28–7.44). This association was entirely accounted for by unobserved familial risk factors (HR=0.99; 95% CI: 0.46–2.13). Similar pattern of effects was found for substance misuse.

Conclusions—There are no associations between childhood family income and subsequent violent criminality and substance misuse once unobserved familial risk factors are adjusted for.

Poverty or low socioeconomic status (SES) during childhood is a well-known distal risk factor for subsequent criminal and substance misuse behaviors (1, 2). Recently, a Norwegian total population study found that children of parents in the lowest income decile were twice as likely to be convicted of a violent or drug crime compared to their peers in the fifth decile (3). Similarly, a number of longitudinal US studies have linked low income levels with substance use disorders (4, 5). Nevertheless, these findings could potentially result from inadequate adjustment of familial risk factors (6). Behavioral genetic investigations have found that the liabilities for both violent offending and substance misuse are substantially influenced by shared genetic and, to a lesser extent, family environmental factors (7, 8).

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The funders had no role in the design and conduct of the study; collection, management, analysis, and interpretation of the data; and preparation, review, or approval of the manuscript.

There were no financial or other conflicts of interest for any of the authors.

The lead author had full access to the data and takes responsibility for the integrity of the data and the accuracy of the data analysis.

Consequently, it has been proposed that quasi-experimental, genetically informative research designs that explicitly take such factors into account could be integral in elucidating the causal mechanisms further (9). A few smaller quasi-experimental studies have been performed to date and they suggest that the inverse associations between parental income during childhood and development of behavioral problems remain after such adjustments (10–13). The generalizability of these findings is still questioned due to potential selection bias. Determining the causal nature of these associations is crucial to inform policy and clinical preventive efforts.

Methods

Sample

We linked data from nine Swedish longitudinal, total population registers maintained by governmental agencies. The linkage was possible through the unique 10-digit civic registration number assigned to all Swedish citizens at birth and to immigrants upon arrival to the country. We were granted access to de-identified linked data after approval from the Regional Research Ethics Committee at Karolinska Institutet.

The following nine registers were utilized: (a) the *Total Population Register (TPR)* contained basic information (e.g., sex and date of birth) for all individuals registered as inhabitants of Sweden; (b) the *Multi-Generation Register* supplied data that linked index persons found in TPR to their biological parents, thus enabling us to interconnect siblings; (c) the *Medical Birth Register* included pregnancy data with close to full coverage (>99%) of all births in Sweden since 1973 (14) ; (d) the *Education Register*, contained information on highest level of completed formal education; (e) the *Cause of Death Register*, provided data on principal and contributing causes of death since 1958; (f) the *Migration Register* supplied data on dates for migration into or out of Sweden; (g) the *Integrated Database for Labour Market Research (LISA)* provided annual information on family disposable income and welfare recipiency since 1990 on all individuals 16 years of age and older that were registered in Sweden as of December 31 for each year; (h) the *National Patient Register* provided data on psychiatric inpatient care since 1973 (ICD-8, -9 and -10) and outpatient care since 2001 (ICD-10); and (i) the *National Crime Register* supplied detailed information on all criminal convictions in lower general court in Sweden since 1973. Plea bargaining is not allowed and conviction data include all persons who received custodial or non-custodial sentences; also those cases where the prosecutor decided to caution or fine. Only individuals age 15 or older are legally responsible in Sweden; hence, we were not able to study criminal offending prior to age 15.

A total of 594,127 children were born in Sweden between 1989 and 1993 and registered in the Medical Birth Registry. We chose to exclude children from multiple births (n=14,670), those who had serious malformations at birth (n=20,905) or who could not be linked to their biological parents (n=3,956). Furthermore, we excluded data for children who had either died (n=2,525) or emigrated from Sweden before they reached 15 years of age (n=18,301). Last, we removed individuals with missing data on parental labour market exposures (n=7,603). Our final sample consisted of 88.6% of the targeted population (n=526,167). The

sample included 262,267 cousins and 216,424 siblings nested within 114,671 extended and 105,470 nuclear families.

Measures: Exposure variables

Childhood family income throughout ages 1 to 15 years—We calculated mean disposable family incomes (net sum of wage earnings, welfare and retirement benefits etc.) of both biological parents for each offspring and year between 1990 and 2008. Income measures were inflation-adjusted to 1990 values according to the consumer price index provided by Statistics Sweden. Econometric researchers have long recognized that single annual income exposure measures generally suffer from substantial measurement error due to their inability to accurately depict long-term SES, often leading to attenuation bias (15, 16). Therefore, annual variables were used to calculate the mean parental income throughout each offspring's childhood (ages 1 through 15).

Early critics challenged the linearity assumption used by studies adopting continuous income measures by contending that criminality is largely confined to the lowest social strata, often referred to as "the underclass" or "the poor", with little to no difference being found between the strata in the mid to upper ranges of the income distribution (17). Others have argued that the cause of the spurious correlations are due to separate mechanisms promoting deviant behaviors on both ends of the income distribution resulting in weak mean predictions (1). We decided, therefore, to test potential non-linear effects by categorizing our income measure in quintiles.

Confounders—Sex, birth year, and birth order (dichotomous; first born and other) were included in all models. We also adjusted for highest parental education (divided into primary, secondary and tertiary level qualifications) and parental ages (five age categories; <20, 20–25, 25–30, 30–35 and >35) at the time of the first-born child, and parental history of ever being hospitalized for a mental disorder (ICD-8/9: 290–315; ICD-10: F00–F99).

Measures: Outcome variables

Violent crime was defined as a conviction for homicide, assault, robbery, threats and violence against an officer, gross violation of a person's/woman's integrity, unlawful threats, unlawful coercion, kidnapping, illegal confinement, arson, intimidation, or sexual offences (rape, indecent assault, indecent exposure, or child molestation, but excluding prostitution, hiring of prostitutes, or possession of child pornography) (18). In line with previous studies using Swedish total population data (8, 19), we used an omnibus measure of substance misuse consisting of convictions of any drug-related crimes (defined as crimes against the Narcotic Drugs Act (SFS 1968:64) or driving under the influence of alcohol and/or illicit substances) or having been diagnosed with an alcohol- or drug misuse-related disease in inpatient or outpatient settings (ICD-8: 291, 303–4, 571, E853, E856.4, E859, E860, N980; ICD-9: 291, 303–5, 357.5, 425.5, 535.3, 571.0–571.3, E850, E854.1-2, E855.2, E860, N980; ICD-10: F10, G32.2, G62.1, G72.1, I42.6, K29.2, K70, K85, X41-2, X45, X61-2, X65, Y11 [with T43.6], Y12 [with T40] and Y15 [with T51]).

Statistical analyses

To account for time at risk, we calculated hazard ratios (HRs) with corresponding 95% confidence intervals (CIs) for adolescent violent crime or substance misuse by fitting Cox proportional hazards regression models to the data. The participants entered the study at their 15th birthday and were subsequently followed up for a median time of 3.5 years. The maximum follow-up time was 6 years. Those who emigrated or died during follow-up were censored.

We fitted two separate models for the entire sample (n=526,167) that gradually adjusted for *observed* confounding variables. Model I adjusted for sex, birth year and birth order while Model II also adjusted for covariates highest parental education, parental ages at the time of the first-born child, and parental history of hospitalization for a mental disorder.

To assess the effects also of *unobserved* genetic and environmental factors, we fitted stratified Cox regression models to cousin (n=262,267) and sibling (n=216,424) samples with extended or nuclear family as stratum, respectively. The stratified models allow for the estimation of heterogeneous baseline hazard rates across families and thus capture unobserved familial factors (20). This also implies that exposure comparisons are made within families (21). Model III was fitted to the cousin sample and adjusted for observed confounders and unobserved within extended-family factors. Model IV was fitted on the sibling sample and accounted for unobserved nuclear family factors and for sex, birth year and birth order.

Cousin and sibling correlations on the exposure variable were calculated based on a varying-intercepts, mixed-effects model where the intercepts are allowed to vary across families (22). The magnitude of the variation was expressed as an intra-class correlation (ICC) (23). The ICC measures the degree to which observations are similar to one another within clusters; in this case cousins and siblings nested within extended and nuclear family clusters. The measure ranges between 0 and 1 where the latter implies that cousins and siblings have identical exposure values within families.

All models were fitted in Stata 12.1 IC (24).

Results

Demographic sample characteristics are presented in Table 1. Adolescent violent crime and substance misuse rates were inversely correlated with the childhood family income exposure. As an example, children of parents in the lowest income quintile experienced a rate of 11.05 per 1,000 person-years of being convicted of a violent crime while the same estimate was 1.77 for the children of parents in the highest income quintile.

Table 2 presents results from multivariable Cox regression models; children to parents in the lowest income quintile had an almost seven-fold increased hazard of being convicted of violent crime (crude HR=6.78; 95% CI: 6.23–7.38) and a two-fold increase of substance misuse (HR=2.45; 95% CI: 2.32–2.58) in adolescence compared to peers whose parents were in the fifth quintile (Model I).

When we made adjustments for observed family-wide risk factors (Model II), the effects of childhood family income on violent criminal convictions were significantly attenuated but remained strong (HR=3.93; 95% CI: 3.59–4.30). Controlling for family-wide risk factors also affected the association with substance misuse (HR=1.98; 95% CI: 1.86–2.10).

Model III expanded Model II by also accounting for unobserved familial risk factors within extended families (through cousin comparisons). This adjustment reduced the hazard ratios by 50% and 25% for adolescent violent crime and substance misuse, respectively. Lastly, we studied the effects of unobserved familial risk factors within nuclear families using sibling comparisons (Model IV). The associations between childhood family income and the outcomes disappeared completely; hazard ratios were 0.95 (95% CI: 0.44–2.03) for violent crime and 1.11 (95% CI: 0.62–1.98) for substance misuse, respectively. This suggested that unobserved familial factors fully accounted for the increased hazard ratios found in previous models.

Sensitivity analyses

Sibling correlations for childhood family income were, expectedly, rather high (Table 3), suggesting that the within-family variability was somewhat limited. Consequently, we re-fitted models presented in Table 2 to the childhood family income exposure variables covering single-year age periods (eTable 1). Sibling correlations for the latter were 0.57–0.74. Despite larger heterogeneity between siblings in these exposures, the results remained quite similar.

As suggested in Table 4, we could not find any period effects of the timing of exposures on substance misuse. The crude associations presented in Model I were high, but consistently appeared explained by familial factors (Model IV).

Differences between estimates for male-only and total population samples were small. In contrast, the females' estimates indicated low precision with wide confidence intervals, especially for violent convictions within families. The discrepancies across estimates for the different birth order subsamples and analyses excluding second-generation immigrants from non-Nordic countries and the total population sample were marginal.

The extent to which other non-linear categorizations (i.e. tertiles and deciles) of childhood family income impacted the results was tested and we found negligible differences (data not presented; available upon request).

In addition, we explored whether results were due to the relatively low rates of our outcome variables by re-fitting models to the following alternative outcomes: (a) any criminal conviction and (b) any property conviction. Corresponding rates were 25.27 and 10.70 per 1,000 person-years, respectively. The general pattern of effects found in the main analyses remained here (eTable 2), and the magnitudes of estimates were also very similar to those of models predicting substance misuse (cf. Tables 2 and 4).

Finally, we investigated if the results were specific to the childhood SES exposure variable by re-fitting the models to an alternative indicator; parental welfare recipiency between ages 1 through 15. Individuals who receive means-tested welfare benefits in Sweden are not

primarily characterized by their lack of financial means; they are a selected group with a wide range of psychosocial issues (25). The results nonetheless matched those exploring childhood family income (eTable 3).

Discussion

Using traditional epidemiological methods, we found that low income in one's family of origin was indeed associated with higher risk of violent offending and substance misuse during adolescence. However, the excess risks became marginal or disappeared completely when we gradually adjusted for familial risk factors of these associations by studying within-extended family and within nuclear-family estimates (with cousin and sibling controls, respectively). This held true when childhood SES was defined either as parental disposable income or welfare recipiency throughout child ages 1–15 years. Sensitivity analyses proved the results robust across sex, ethnicity and age periods and not influenced by limited within-family variability in the exposure variables.

Overall, our finding that the associations between childhood family income and adolescent violent criminality and substance misuse are unlikely to be causal has been suggested in prior systematic reviews on SES and criminality (26–29). On the contrary, smaller US-based quasi-experimental studies on behavioral problems have indicated causal effects (10–12). The diverging results may have at least two plausible explanations. First, outcome variables are not directly comparable between studies; while we have focused on severe criminal offending and substance misuse, earlier studies addressed less severe antisocial behaviors and conduct problems. Second, it could be that Sweden's comprehensive welfare state actually mitigates possible adverse effects of growing up with limited material resources (30).

Our results indicate therefore that prevention efforts that specifically aim to decrease rates of violent offending and substance misuse should target a wider range of familial risk factors than merely parental income. This recommendation is in line with contemporary research that defines early socioeconomic exposures as distal risk factors due to their lack of direct associations with delinquency and antisocial behaviors while familial risk factors (e.g. quality of the parent-child relationship, family dissolution and parental criminality) are instead viewed as proximal risk factors because they tend to explain the majority of the variance in such outcomes (31, 32). Further large-scale genetically informative quasi-experimental studies are thus going to be crucial in identifying and determining potentially causal familial predictors of violent criminality and substance misuse.

Strengths and weaknesses

In addition to this being the largest study of childhood SES, adolescent violent crime and substance misuse; a total population study of five birth cohorts of children born 1989–1993, we addressed and ruled out possible effects from various methodological weaknesses pointed out previously (1, 15–17, 33). Measurement error was minimized by the use of well-defined, prospectively and objectively gathered family income measures spanning 15 years (16). The extensive 15-year exposure period made it possible to study potential temporal variability in effects including both the timing and persistence of low childhood SES.

Three methodological considerations are important when interpreting the present findings. First, we cannot exclude potential bias from cohort effects that might have affected the associations between childhood family SES and outcome because included cohorts were infants or pre-school children when Sweden underwent a major economic recession in the mid-1990s with quadrupling unemployment rates and substantially rationalized welfare programs (34). We were unable to explore such bias because we did not have access to yearly parental income data prior to 1990. However, if anything, cohort effects bias may have led to an overestimation of unadjusted effects seen before accounting for unobserved familial risk factors.

Second, our approach of utilizing nationwide registry data confined our analyses to arguably more severe cases that had been registered by the legal and clinical services for their actions. It is obviously an empirical question whether the results for non-diagnosed cases would be similar.

Third, the sibling-comparison design makes several important assumptions and requires a large sample size (9, 35, 36). In principle, only sibling pairs discordant on both exposure and outcome contribute to the analyses. We identified 116,875 siblings in 56,551 families who were discordant for childhood family income (measured in deciles). Among these discordant siblings, 3,195 were further discordant for violent criminal convictions and 5,507 for substance misuse. Nevertheless, these sample sizes are still larger than in most of the previous studies. Moreover, the sibling-comparison design assumes that the results of discordant siblings are generalizable to the total population. We found no income differences when comparing the discordant siblings to the total population; $t(526165)=1.25$, $p=0.21$. Thus, our findings do not seem to follow from poor statistical power or that results from discordant siblings are not generalizable.

In conclusion, the present study highlights the importance of adjusting for unobserved familial risk factors when studying the impact of childhood SES on later adverse outcomes, such as violent crime and substance misuse; hence, claims of causal effects after only adjusting for *observed* covariates should be viewed with caution. We found strong inverse correlations that were explained fully by unobserved familial risk factors shared by children growing up in low SES households. Future research is needed to validate these results in other contexts and elucidate the nature of the mechanisms, including the relative contributions of genes or environments.

Acknowledgments

The study was supported by the Swedish Council for Working Life and Social Research, the Swedish Research Council (2010-3184; 2011-2492) and the National Institute of Child Health and Human Development (HD061817).

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Table 1

Demographic characteristics for all children born in Sweden 1989–1993 that were included in a study of childhood family income (ages 1–15 years) and violent crime convictions and substance misuse during adolescence (ages 15–21 years).

Variable	N	%	Adolescent violent crime Rate per 1,000 person-years	Adolescent substance misuse Rate per 1,000 person-years
Total sample	526,167	100	5.12	7.36
Sex				
Male	269,625	51.24	8.20	8.28
Female	256,542	48.76	1.91	6.40
Birth year				
1989	102,687	19.52	4.31	6.93
1990	108,641	20.65	5.21	7.87
1991	108,897	20.70	5.39	7.51
1992	106,435	20.23	6.00	7.47
1993	99,507	18.91	5.60	6.79
Birth order				
First born	215,598	40.98	4.48	6.73
Other	310,569	59.02	5.56	7.81
Mean parental disposable income				
Quintile 1 (lowest)	105,234	20.00	11.05	12.45
Quintile 2	105,233	20.00	6.04	8.03
Quintile 3	105,234	20.00	3.60	5.60
Quintile 4	105,233	20.00	2.86	5.25
Quintile 5 (highest)	105,233	20.00	1.77	5.30

Table 2

Relative risks for adolescent violent crime and substance misuse as a function of childhood (ages 1–15) family income by quintiles.

	Adolescent violent crime Hazard ratio [95% CI]				Adolescent substance misuse Hazard ratio [95% CI]			
	Model I	**Model II**	**Model III**	**Model IV**	**Model I**	**Model II**	**Model III**	**Model IV**
Quintile 1 (low)	6.78 [6.23; 7.38]	3.93 [3.59; 4.30]	1.89 [1.40; 2.56]	0.95 [0.44; 2.03]	2.45 [2.32; 2.58]	1.98 [1.86; 2.10]	1.53 [1.24; 1.90]	1.11 [0.62; 1.98]
Quintile 2	3.66 [3.35; 4.00]	2.50 [2.28; 2.74]	1.46 [1.09; 1.95]	0.81 [0.41; 1.61]	1.56 [1.47; 1.66]	1.39 [1.31; 1.48]	1.19 [0.97; 1.45]	1.31 [0.81; 2.13]
Quintile 3	2.14 [1.95; 2.36]	1.61 [1.46; 1.77]	1.04 [0.78; 1.40]	0.76 [0.42; 1.39]	1.08 [1.02; 1.15]	1.02 [0.95; 1.08]	0.96 [0.79; 1.18]	1.12 [0.76; 1.67]
Quintile 4	1.64 [1.48; 1.81]	1.34 [1.21; 1.48]	0.80 [0.60; 1.06]	0.64 [0.39; 1.05]	1.00 [0.93; 1.06]	0.96 [0.90; 1.03]	0.98 [0.81; 1.17]	1.09 [0.81; 1.47]
Quintile 5 (high)	Reference	Reference	Reference	Reference	Reference	Reference	Reference	Reference

Notes: Figures express hazard ratios (HR:s) with corresponding 95% confidence intervals (CI:s) within brackets. Model I: Adjusted for sex, birth year and birth order; Model II: Model I + adjusted for parental highest achieved education, age at birth and history of mental disorder; Model III: Model II + adjusted for unobserved within extended-family risk factors (through cousin comparisons); Model IV: Model I + adjusted for unobserved within nuclear-family risk factors (through sibling comparisons).

Table 3

Cousin and sibling intra-class correlations (ICCs) of childhood family income exposure by age periods.

Childhood family income	Cousins		Siblings	
	ICC	95% confidence	ICC	95% confidence
Ages 1–5 years	0.62	[0.62; 0.63]	0.90	[0.90; 0.90]
Ages 6–10 years	0.65	[0.65; 0.65]	0.90	[0.90; 0.90]
Ages 11–15 years	0.65	[0.64; 0.65]	0.91	[0.91; 0.91]
Ages 1–15 years	0.69	[0.69; 0.70]	0.96	[0.96; 0.96]

Table 4

Sensitivity analyses. Relative risks as a function of childhood family income stratified by exposure age periods, sex, number of children in household, birth order and parental immigrant status.

	Adolescent violent crime Hazard ratio [95% CI]		Adolescent substance misuse Hazard ratio [95% CI]	
	Model I	**Model IV**	**Model I**	**Model IV**
Ages 1–15 yrs (reference)	6.78 [6.23; 7.38]	0.95 [0.44; 2.03]	2.45 [2.32; 2.58]	1.11 [0.62; 1.98]
Ages 1–5 yrs	4.36 [4.06; 4.68]	0.76 [0.46; 1.24]	2.10 [2.00; 2.22]	0.98 [0.65; 1.48]
Ages 6–10 yrs	5.93 [5.46; 6.45]	0.91 [0.54; 1.55]	2.32 [2.20; 2.45]	1.17 [0.77; 1.79]
Ages 11–15 yrs	6.06 [5.57; 6.59]	0.59 [0.34; 1.03]	2.28 [2.16; 2.40]	1.03 [0.67; 1.59]
Males only, ages 1–15 yrs	6.39 [5.82; 7.01]	0.51 [0.24; 1.12]	2.42 [2.25; 2.60]	0.84 [0.37; 1.92]
Females only, ages 1–15 yrs	8.95 [7.24; 11.08]	1.32 [0.24; 7.19]	2.48 [2.28; 2.70]	1.15 [0.46; 2.86]
Single children households only, ages 1–15 yrs	5.87 [3.70; 9.29]	N/A	2.45 [1.89; 3.17]	N/A
First-born only, ages 1–15 yrs	7.26 [6.19; 8.53]	N/A	2.57 [2.34; 2.83]	N/A
Other birth order, ages 1–15 yrs	6.45 [5.83; 7.13]	1.44 [0.49; 4.22]	2.35 [2.20; 2.52]	0.82 [0.32; 2.06]
Nordic-born parents only, ages 1–15 yrs	6.72 [6.17; 7.32]	0.98 [0.45; 2.11]	2.45 [2.32; 2.59]	1.11 [0.62; 1.99]

Note: Figures express hazard ratios (HR:s) with corresponding 95% confidence intervals (CI:s) within brackets. Model I: Adjusted for sex, birth year and birth order; Model IV: Model I + adjusted for unobserved within nuclear-family risk factors (through sibling comparisons). N/A: Not applicable.