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ORIGINAL ARTICLE

Familial factors and suicide: an adoption study in a Swedish National Cohort

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Background. Parental characteristics influence the risk of offspring suicide. In this study we wanted to separate the hereditary from the environmental influence of such factors by comparing their effects in the adopted *versus* non-adopted.

Method. A register study was conducted in a national cohort of 2 471 496 individuals born between 1946 and 1968, including 27 600 national adoptees, followed-up for suicide during 1987–2001. Cox regression was used to calculate hazard ratios (HR) for suicide of socio-economic indicators of the childhood household and biological parents' suicide, alcohol abuse and psychiatric morbidity separately in the adopted and non-adopted. Differences in effects were tested in interaction analyses.

Results. Suicide and indicators of severe psychiatric disorder in the biological parents had similar effects on offspring suicide in the non-adopted and adopted (HR 1.5–2.3). Biological parents' alcohol abuse was a risk factor for suicide in the non-adopted group only (HR 1.8 *v.* 0.8, interaction effect: $p=0.03$). The effects of childhood household socio-economic factors on suicide were similar in adopted and non-adopted individuals, with growing up in a single parent household [HR 1.5 (95% confidence interval 1.4–1.5)] as the most important socio-economic risk factor for the non-adopted.

Conclusions. The main familial effects of parental suicide and psychiatric morbidity on offspring suicide are not mediated by the post-natal environment or imitation, in contrast to effects of parental alcohol abuse that are primarily mediated by the post-natal environment. Social drift over generations because of psychiatric disorders does not seem likely to explain the association of socio-economic living conditions in childhood to suicide.

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Introduction

Childhood exposure to severe parental psychiatric problems such as suicidal behaviour, mental illness and substance abuse increases the risk of suicide later in life (see, for example, Dube *et al*. 2001). Evidence from family, twin and adoption studies supports the hypothesis that genetic factors contribute to this risk (Baldessarini & Hennen, 2004; Brent & Mann, 2005). Other studies demonstrate that childhood environmental adversities – such as sexual abuse, foster care, parental divorce and lone parenthood – also elevate the risk for suicidal behaviour in adult life (Dube *et al*. 2001, 2003; Hjern *et al*. 2004; Dong *et al*. 2005; Vinnerljung *et al*. 2006; Fergusson *et al*. 2007).

Parental suicide may be linked to offspring suicide in both ways. The risk is partly heritable (Brent & Mann, 2003) and various mechanisms have been suggested. The seemingly self-evident way via psychiatric illness most probably explains only a minor part of the heritability (Brent & Mann, 2005). However, early-onset major depression is one subtype of depression associated with an increased risk of suicide (Thompson, 2008) that has a well-established heritability (Lyons *et al*. 1998). Studies based on endophenotypes – internal phenotypes between gene and disease (Gottesman & Gould, 2003) – constitute a promising approach for identifying relevant genetic mechanisms. For instance, impulsive–aggressive traits,

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neurocognitive prerequisites and hypothalamic–pituitary–adrenal axis dysfunction all seem to be associated with suicide (Mann *et al*. 2009). The strongest evidence for specific genes involved in suicidal behaviour concerns serotonin transport and tryptophan hydroxylase (Roy *et al*. 2009).

Parental suicide also means a painful loss for the survivors and the environmental conditions may be changed dramatically for the afflicted child. Hypothetically, a parental suicide may inspire suicidal behaviour in the offspring even if there is little empirical support for such mechanisms (Burke *et al*. 2010). However, few attempts have been made to test this hypothesis in population data of mortal suicidal behaviour.

Danish national register studies have reported inconclusive results about parental psychiatric illness and offspring suicide, one with no significantly increased risk in a study group followed up until middle adult life (Sørensen *et al*. 2009) and two with increased suicide risk in adolescents and young adults (Qin *et al*. 2002; Stenager & Qin, 2008). Any influence of a parental psychiatric disorder is dependent on the course and timing of the disorder and may be mediated in many ways in the family sphere (Goodman & Gotlib, 1999). The health of the other parent as well as the involvement of this parent with the child will moderate the risks (Goodman & Gotlib, 1999), illustrating the dilemma of the lone-parent family. The quality of the relationship between the child and the parent in focus is one potential mediator. In an experimental study using a problem-solving task design, mothers with current depressive symptoms displayed a low level of positivity toward their child (mean age 11.9 years) and a high level of negativity, which in turn was related to externalizing problems in the child (Foster *et al*. 2008). Psychiatric disorders in parents also have environmental consequences, for instance, by a downwards social drift of the living conditions of the household, which in turn may influence suicide risk (Agerbo, 2007). Goldberg even proposes that social drift may extend over several generations through hereditary factors (Goldberg & Morrison, 1963; Timms, 1998).

Most previous studies have reported an association between parental substance abuse and offspring suicide (Brent *et al*. 1994; Christoffersen & Soothill, 2003). One case-control study of young (<20 years of age) suicide victims is an exception, demonstrating no such association (Gould *et al*. 1996). Studies of genetic influence have not been conclusive. A genetic polymorphism associated with suicide and alcoholism has been described (Nielsen *et al*. 1998). In a children-of-twins study, Glowinski *et al*. (2004) found a relationship between paternal alcoholism and suicidal behaviour in the offspring, but their data did not support a purely genetic relationship. They concluded that without a high-risk environment, even a group at high or intermediate genetic risk was not at higher risk for suicide attempts than a group with low genetic risk. Parental alcohol abuse is also associated with – and may indeed be a causal agent for – various forms of environmental adversity such as abuse and neglect (Kelleher *et al*. 1994; Johnson & Leff, 1999; Lieberman, 2000) which in turn influence the risk of suicide (Christoffersen & Soothill, 2003).

Two methodological approaches are suitable for estimating the strength of genetic *versus* environmental factors: twin and adoption studies. In several twin studies a greater concordance for both suicide and suicide attempts in monozygotic than in dizygotic twins has been demonstrated (Roy *et al*. 1991, 1995; Statham *et al*. 1998; Cho *et al*. 2006). Adoption studies offer even better possibilities to separate genetic from environmental effects. Adopted children who are separated from their biological parents at birth or shortly afterwards share their genes, but – with exception of intra-uterine influence and influence during the usually short period before adoption – not the environmental effects of a parental suicide or a parental psychiatric disorder. So far two landmark adoption studies have addressed the topic of familial factors and suicide, both using a retrospective case-control design (Schulsinger *et al*. 1979; Wender *et al*. 1986). Wender *et al*. (1986) carried out an adoption study of affective disorders. They identified a 15-fold increased risk of suicide among family members in biological families of 71 national adoptees with affective disorder, compared with biological family members of 71 healthy adoptee controls. In a study of 57 adoptee suicides from the same Danish adoption registry, Schulsinger *et al*. (1979) found a six-fold elevated rate of suicide in the biological relatives of adoptees that had committed suicide compared with relatives of non-suicidal adoptee controls.

Scandinavian national registers have a unique potential in identifying risk factors for comparatively rare outcomes, such as suicide, because of the comprehensive coverage of entire populations with non-biased information. For adoption studies, however, the frequency of the risk factors is often too low in register studies to allow for a traditional adoption design. In this study we used a modified adoption design to address research questions about heritable and socioeconomic determinants of suicide by comparing the effect of these determinants in children raised by their biological parents with the effect in adopted children.

Method

This study was based on data from the national health databases held by the Swedish National Board of

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Health and Welfare and the national social databases held by Statistics Sweden linked through each individual's unique personal identification number. This personal number is awarded to each Swedish resident at birth or, for immigrants, after having received a permanent residency in Sweden, and is used in all national registers from cradle to death.

Study population

The study population consisted of all persons born between 1946 and 1968, who were residents in Sweden on 31 December 1986 according to the Register of the Total Population (RTP), and had at least one biological parent recorded in the Multi-Generation Register identified as a Swedish resident in the census of 1960; 2 471 496 individuals. This population was followed prospectively in the Swedish national registers from 1987 until 2001. Such a long follow-up time is possible by linkage through each individual's unique personal identification number. The Multi-Generation Register covers the whole population and allows for linkages between adoptees and their adoptive as well as their birth parents.

Within this population a subgroup of 27 600 national adoptees was identified by records of an adoptive father and an adoptive mother and Sweden as a country of birth. Partner and other family adoptions (adoptions by an older sibling, grandparent, or sibling of the biological parents) were excluded, because of the genetic links between the adoption and the biological family in these cases. No data were available about age at adoption and the actual environmental circumstances of the children prior to adoption (e.g. time in infant residential care). From other sources, however, we have information that most adoptions during this time period were made from unwanted pregnancies in young unmarried women (Bohman, 1970; Nordlöf, 2001). Since private adoptions are prohibited by Swedish law, the children were taken into institutional care by the municipalities shortly after birth to be adopted at a median age of 6 months, and very few children were adopted at a later age than 12 months according to these same sources (Bohman, 1970; Nordlöf, 2001). The quality of the care in the infant homes varied but was judged to be 'fairly satisfactory' according to the standards of the time. Boys tended to be adopted at a later age than girls and children with early signs of disability were often exempted from adoption and cared for in foster care or institutions. There was no policy to match adoptive with biological parents, but the possibility that such matching was done in individual cases cannot be completely excluded (Bohman, 1970; Nordlöf, 2001).

Sociodemographic variables

Year of birth, sex and geographic location of the home (residency) of the study population were identified in the RTP in 1986.

Data on the families' socioeconomic background were retrieved from the latest Swedish Population and Housing Census before the study subjects turned 18 years. For those born between 1946 and 1952 it was the 1960 census, for those born between 1953 and 1962 it was the 1970 census, for those born between 1963 and 1968 it was the 1980 census, and for those born in 1968 it was the 1985 census. Variables used for describing childhood household socio-economic background were socio-economic status (SES), residency, housing, single parenthood and maternal age (the mother's age at the time of the child's birth). For the adoptees these variables refer to their adoptive families.

SES was defined according to a classification used by Statistics Sweden, which is based on occupation but also takes educational level of occupation, type of production and position at work of the head of the household into account (Statistics Sweden, 1982). Unfortunately, however, the categories in the Census of 1960 on these circumstances were crude, which forced us to use a simplified categorization of SES consisting of blue collar, white collar and unclassified (including missing, unemployed and others that do not fit into the first two categories, e.g. self-employed).

Indicators of parental psychiatric morbidity/suicide

Data on parental risk factors were obtained through individual record linkage to the Swedish Hospital Discharge Register for the years 1987 to 2002 and the National Cause of Death Register (NCDR) from 1960 to 2001 with the aid of the 6th, 7th, 8th, 9th and 10th revision of the World Health Organization International Classification of Diagnosis (ICD-6–10). Parental risk factors were dichotomized as having at least one parent or having no parent that fulfilled the criteria for each variable. 'Suicide death' in parents was defined in the same way as for the study population (see below). 'Psychotic and affective disorders' was defined as at least one discharge or/and an underlying or contributing cause of death diagnosis of a psychotic disorder (F20–F29 in ICD-10) or a mood disorder (F30–F39 in ICD-10). 'Alcohol abuse' was defined as at least one discharge or/and an underlying or contributing cause of death diagnosis indicating a psychiatric or somatic disorder associated with alcohol addiction or excessive alcohol consumption.

Outcome

The outcome variable 'suicide death' was created with data from the NCDR. During the years 1987–1996

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it was defined according to ICD-9 as an underlying cause of death of E950–E959 or E980–E989, thus including both certain and uncertain suicides, with the corresponding categories from ICD-10 during 1997–2001: X60–X84 or Y10–Y34. All suicide diagnoses were based on forensic autopsies.

Statistical analysis

In the multivariate analysis, hazard ratios (HR) were calculated using Cox regressions of person days with suicide death as the dichotomized outcome variable (definition above). Person time in the study was calculated with data on death from the NCDR and data on immigration during 1987–89 and the Total Enumeration Income Survey during 1990–2001.

Statistical analysis was performed separately for socio-economic indicators of the childhood household and indicators of psychiatric morbidity, including suicide, of the biological parents. In a first model the analysis was adjusted for age and sex only, where birth year was entered as a continuous variable, since suicide death tended to decrease by year of birth in a linear fashion. Model 2 added indicators of parental morbidity to the analysis of socio-economic indicators and vice versa.

The analysis was stratified into the adopted and non-adopted. Differences in effects of determinants between adopted and non-adopted individuals were calculated in interaction effects between these two strata in a model, adjusted for age and sex only, that included the whole study population. SPSS 15.0 for Windows software package (SPSS Inc., USA) was used in all statistical analyses (SPSS Inc., 2004).

Results

A total of 8635 (0.4%) non-adopted and 180 (0.7%) adopted individuals had committed suicide during the follow-up period ($p<0.001$). Of all suicides, 73.3% were committed by men. The adopted had higher rates of suicide compared with the non-adopted (8.5 *v.* 5.2/1000 for men and 3.9 *v.* 2.0/1000 for women).

Suicide risk varied considerably in the whole study population for all demographic and socio-economic determinants investigated in this study. Biological parents' suicide, affective/psychotic disorders and alcohol abuse also influenced the suicide risk. This influence tended to be higher for maternal morbidity indicators compared with paternal indicators for women, while this was true only for suicide and alcohol abuse for men (Table 1).

Table 2 shows the multivariate analysis of the socio-economic determinants of the childhood household. Growing up in a single parent household (HR 1.46) was found to have the highest risk for suicide in the non-adopted but tended to have a lower effect for the adopted (HR 1.46 *v.* 0.92, $p=0.25$). Parental SES, housing and low maternal age contributed less (HR 1.15, 1.14 and 1.16, respectively) with similar effects in the adopted and non-adopted. HRs decreased marginally with adjustment for biological parents' morbidity.

Table 3 shows the multivariate analysis of the indicators of psychiatric morbidity and suicide in the biological parents. Biological parents' suicide, as well as psychotic and affective disorders, had a similar influence on the suicide risk for adoptees and the non-adopted. Biological parents' alcohol abuse had a greater effect in the non-adopted than in the adopted (HR 1.88 *v.* 0.82, $p=0.03$).

Discussion

In this register study of a national cohort we compared the effect of socio-economic and heritable risk factors for 180 suicides in the adopted with more than 8000 suicides in young and middle-aged non-adopted individuals within the same age range. Suicide and severe psychiatric disorder in the biological parents had similar effects in the non-adopted and adopted. Alcohol abuse in biological parents, however, was found to be a risk factor for suicide in non-adoptees only. Socio-economic determinants had a similar effect in the adopted and the non-adopted, indicating that social drift because of parental psychiatric morbidity or suicide did not explain the major effects of these variables.

It is notable that the risk of suicide in individuals having experienced parental suicide was almost the same for those raised by adoptive and biological parents. This speaks against the imitation hypothesis as an explanation of the increased risk of suicide associated with parental suicide. It is also in line with the findings of Burke *et al*. (2010) who reported a four-fold increased likelihood for suicide attempt in a study of offspring of depressed parents with suicidal behaviour. This increase, however, was eliminated if the exposure was restricted to exposure antecedent to the suicidal behaviour. The authors found only two previous studies on the topic with an adequate design, both reporting similar findings (Burke *et al*. 2010).

The indicator for parental psychiatric morbidity did not mediate the main effects of parental suicide on suicide in the adoptees in this study. Results from quite a few previous studies point in the same direction, highlighting the specific genetic contribution linked to parental suicide (Roy *et al*. 1991; Fu *et al*. 2002; Brent & Mann, 2005). It is reasonable to believe that this contribution reflects specific endophenotypes,

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Table 1. *Incidence of suicide death in the study population by adoption status, childhood household characteristics and psychiatric morbidity of the biological parents*

	Men			Women		
	n	No. of suicides	Incidence, per 1000	*n*	No. of suicides	Incidence, per 1000
Adoption status						
Adopted	17 227	147	8.5	16 013	63	3.9
Non-adopted	1 491 789	7706	5.2	1 417 903	2898	2.0
Childhood household						
Parental SES						
White collar	542 546	2438	4.5	515 774	1050	2.0
Blue collar	620 740	3493	5.6	593 523	1203	2.0
Missing or other	345 730	1922	5.6	324 619	708	2.2
Residency						
Metropolitan regions	451 741	2315	5.1	449 080	1078	2.4
Smaller city	760 320	3969	5.2	718 772	1431	2.0
Rural area	296 955	1569	5.3	266 064	452	1.7
Housing						
Apartment	548 848	3244	5.9	535 676	1311	2.4
Own house	949 443	4553	4.8	886 876	1610	1.8
Single parent household						
Yes	220 281	1573	7.1	211 290	554	2.6
No	1 279 178	6229	4.9	1 212 371	2370	2.0
Maternal age, years						
<25	483 006	2580	5.3	460 287	932	2.0
25–34	767 039	3473	4.5	726 387	1314	1.8
>34	250 170	1307	5.2	239 329	504	2.1
Biological parents						
Suicide						
Any parent	20 671	204	9.9	19 465	76	3.9
Mother	6108	71	11.6	5741	34	5.9
Father	14 726	135	9.2	13 859	43	3.1
Alcohol abuse						
Any parent	51 762	443	8.6	48 959	163	3.3
Mother	12 842	140	10.9	12 233	52	4.2
Father	40 469	322	8.0	38 321	119	3.1
Psychotic disorders						
Any parent	18 990	155	8.2	18 228	66	3.6
Mother	9363	86	9.2	8914	43	4.8
Father	9706	70	7.2	9410	23	2.4
Affective disorder						
Any parent	14 961	135	9.0	14 045	78	5.5
Mother	9688	86	8.9	9002	54	6.0
Father	5329	51	9.6	5109	24	4.7
All	1 509 016	7853	5.2	1 433 916	2961	2.1

SES, Socioeconomic status.

such as impulsive–aggressive traits (Carballo *et al*. 2008; Brent, 2009), which in turn may be related to aberrant hereditary serotonin patterns (Rujescu *et al*. 2007).

The absence of an increased risk for suicide in children born to parents with alcohol problems was unexpected, considering the established association between substance abuse and suicide (Harris & Barraclough, 1997). A lower level of serotonin has been found in both and has also been proposed as a possible shared genetic factor (Nielsen *et al*. 1998). Roy *et al*. (2007), in a gene–environment study, found that a

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Table 2. *Cox regression of sociodemographic determinants of the childhood household and suicide*

	Non-adopted ($n = 2\,443\,897$)			Adopted ($n = 27\,600$)			Interaction effect[c]
	Incidence, per 1000	Model I[a] HR (95% CI)	Model II[b] HR (95% CI)	Incidence, per 1000	Model I[a] HR (95% CI)	Model II[b] HR (95% CI)	*p*
Sex							
Male	5.1	2.59 (2.47–2.70)	2.62 (2.50–2.75)	8.8	2.15 (1.59–2.90)	2.67 (1.56–3.57)	0.31
Female	1.9	1	1	3.9	1	1	
Parental SES							
White collar	3.1	1	1	5.9	1	1	
Blue collar	3.8	1.15 (1.10–1.21)	1.13(1.08–1.19)	7.4	1.20 (0.87–1.66)	1.10 (0.61–2.00)	0.79
Missing or other	3.7	1.14 (1.08–1.21)	1.12 (1.06–1.19)	6.4	1.20 (0.83–1.74)	2.01 (1.12–3.60)	0.67
Residency							
Metropolitan regions	3.6	1.05 (0.99–1.11)	1.04 (0.98–1.11)	6.3	0.99 (0.65–1.52)	1.11 (0.54–2.27)	0.52
Other city	3.5	1.02 (0.97–1.08)	1.03 (0.97–1.09)	6.6	1.04 (0.71–1.52)	1.04 (0.55–1.97)	0.82
Rural area	3.5	1	1	6.6	1	1	
Housing							
Apartment	4.1	1.16 (1.11–1.21)	1.13 (1.07–1.18)	8.2	1.45 (1.07–1.97)	1.82 (1.09–3.06)	0.40
Own house	3.2	1	1	5.8	1	1	
Single parent household							
Yes	4.9	1.46 (1.39–1.54)	1.33 (1.25–1.42)	6.5	0.92 (0.57–1.47)	0.88 (0.35–1.75)	0.25
No	3.3	1	1	6.8	1	1	
Maternal age, years							
<25	3.6	1.16 (1.11–1.21)	1.13 (1.07–1.19)	9.1	1.56 (1.05–2.30)	1.21 (0.63–2.32)	0.30
25–34	3.1	1	1	5.9	1	1	
>34	3.6	1.11 (1.05–1.17)	1.14 (1.07–1.21)	6.2	1.10 (0.80–1.49)	0.92 (0.54–1.56)	0.60

HR, Hazard ratio; CI, confidence interval; SES, socio-economic status.

[a] Adjusted for age.

[b] Adjusted for age and biological parents' morbidity.

[c] *p* Value in interaction analysis in model I in the entire study population.

Table 3. *Cox regression of indicators of psychiatric morbidity of the biological parents and offspring suicide*

	Non-adopted ($n = 24\,443\,897$)			Adopted ($n = 27\,600$)			Interaction effect[c]
	Incidence, per 1000	Model I[a] HR (95% CI)	Model II[b] HR (95% CI)	Incidence, per 1000	Model I[a] HR (95% CI)	Model II[b] HR (95% CI)	*p*
Suicide of parent(s)							
Yes	6.9	1.99 (1.78–2.23)	1.88 (1.68–2.10)	16.4	2.25 (1.63–3.03)	2.37 (1.19–4.69)	0.53
No	3.5	1	1	6.3	1	1	
Any severe psychiatric disorder of parent(s)							
Yes	6.1	1.51 (1.35–1.70)	1.50 (1.33–1.69)	11.8	1.82 (1.00–3.27)	1.82 (1.01–3.29)	0.51
No	3.5	1	1	6.3	1	1	
Alcohol abuse by parent(s)							
Yes	6.0	1.88 (1.73–2.04)	1.71 (1.57–1.86)	6.0	0.82 (0.36–1.88)	0.81 (0.36–1.85)	0.03
No	3.5	1	1	6.5	1	1	

HR, Hazard ratio; CI, confidence interval.

[a] Adjusted for sex and age.

[b] Adjusted for sex, age and socio-economic indicators.

[c] *p* Value in interaction analysis in model I in the entire study population.

low expression of the promoter region of the serotonin transporter gene increased the risk of a suicide attempt associated with childhood trauma in patients with substance dependence. Further, alcohol-dependent individuals with suicidal behaviour score high on impulsive and aggressive behaviours (Koller *et al*. 2002), suggesting a link to impulsive–aggressive traits from both these clinical phenomena. To some degree our results challenge these hypotheses about common genetic mechanisms for substance abuse and suicide or at least suggest that the impact of such genetic influence is considerably lower than could be expected. Instead, the main influence of parental substance abuse on suicide seems to be mediated by the family environment.

Having grown up in a single parent household stood out as a risk factor within the childhood household characteristics. Similar results have been shown in previous studies by Weitoft *et al*. (2002, 2003). It has been suggested that there may be an accumulation of other risk factors in single parents nested into these findings, which in turn has a negative influence on the children (Weitoft *et al*. 2002, 2003). In our study, neither sociodemographic factors nor parental psychiatric morbidity explained much of this increased risk in the regression analysis.

The presence of heritable factors that, apart from increasing the risk of a specific event such as suicide, also may influence the interaction of the individual with the labour market suggests a possible 'social drift' over generations (Goldberg & Morrison, 1963; Timms, 1998). The lack of a major interaction between adoption and socio-economic indicators of the childhood household found in this study indicates that social drift does not explain the association between low social status and suicide. It should be noted, however, that this presumes that matching between adoptive and biological parents on socio-economic grounds was not a common practice, a possibility which cannot altogether be discarded.

A higher risk for suicidal behaviour in national as well as international adoptees in Sweden has been reported previously (Hjern *et al*. 2004; von Borczyskowski *et al*. 2006). This higher risk was confirmed in this study. Adoptees tend to grow up under better than average rearing conditions (hence a slightly less favourable risk after adjustment for childhood household characteristics), but heritable risk factors for suicide are considerably more common in adoptees than the non-adopted (von Borczyskowski *et al*. 2006). It is also possible that adoption in itself, or other exposures involved in the adoption process, such as experiences of institutional rearing in infancy, may lead to an increased risk of suicide in adulthood.

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Methodological issues

Previous studies have shown a good validity for register data on suicide in Western countries (Moscicki, 1997). In Sweden all unexpected deaths outside of hospitals are investigated through a forensic autopsy (National Board of Forensic Medicine, 2004). The accuracy of diagnoses in the Cause of Death Register is examined every second year in independent studies of random samples (Official Statistics Sweden, 2004). Furthermore, in our definition of suicide, undetermined deaths were included, to minimize spatial and secular trends in detecting cases of suicide (Linsley *et al*. 2001).

The validity of a diagnosis of schizophrenia in the Swedish National Discharge Register has been found to be quite high and stable over time and between clinics or regions (Dalman *et al*. 2002). A more important methodological problem is probably that indicators of psychiatric morbidity based on psychiatric diagnoses at death or at hospital discharge implies that only a small – and quite extreme – fraction of the total morbidity will be recorded. In comparison with these hospital-based morbidity indicators, the precision of the parental suicide variable can be expected to be much higher. This in turn may lead to an underestimation of the true effects of the psychiatric risk factors in comparison with the effect of parental suicide.

A specific issue in this context concerns the strength of the heritability effect. Early-onset suicidal behaviour has a greater genetic component than later suicidal behaviour (Brent *et al*. 2003; Brent & Mann, 2005). In the present study, cases of parental suicides before 1960 and offspring suicides before 1987 are not included in the analysis. Therefore, the effects of suicide-related heritable factors may be underestimated in our results.

Ideally, we would have liked to perform a pure cross-adoption analysis like Tienari *et al*. (1985) in their classic study on schizophrenia. But due to the low rate of our indicator of psychiatric morbidity in adoptive parents, we would have needed an even bigger dataset, or other indicators of psychiatric morbidity than hospital discharges, suicide and cause of death. The low rate of indications of serious psychiatric morbidity in adoptive parents is not surprising as such since the adoption process in Sweden included 'vetting' applicants for mental health problems.

Conclusions

This study indicates that the association of suicide over generations in the same family is explained by heritable factors rather than through the post-natal environment or imitation. Alcohol abuse, on the other hand, is a risk factor for suicide only when children are exposed to this abuse in their post-natal environment. Social drift over generations because of heritable psychiatric disorders does not seem likely to explain the association of socio-economic living conditions in childhood to suicide.

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Declaration of Interest

None.

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