Article

Genetic and Family and Community Environmental Effects on Drug Abuse in Adolescence: A Swedish National Twin and Sibling Study

Kenneth S. Kendler, M.D.

Hermine H. Maes, Ph.D.

Kristina Sundquist, M.D., Ph.D.

Henrik Ohlsson, Ph.D.

Jan Sundquist, M.D., Ph.D.

Objective: Using Swedish nationwide registry data, the authors investigated genetic and environmental risk factors in the etiology of drug abuse by twin sibling modeling. The authors followed up with epidemiological analyses to identify shared environmental influences on drug abuse.

Method: Drug abuse was defined using public medical, legal, or pharmacy records. Twin and sibling pairs were obtained from the national twin and genealogical registers. Information about sibling pair residence within the same household, small residential area, or municipality was obtained from Statistics Sweden. The authors predicted concordance for drug abuse by years of co-residence until the older sibling turned 21 and risk for future drug abuse in adolescents living with parental figures as a function of family-level socioeconomic status and neighborhood social deprivation.

Results: The best twin sibling fit model predicted substantial heritability for drug abuse in males (55%) and females (73%), with environmental factors shared by siblings operating only in males and accounting for 23% of the variance in liability. For each year of living in the same household, the probability of sibling concordance for drug abuse increased 2%−5%. When not residing in the same household, concordance was predicted from residence in the same small residential area or municipality. Risk for drug abuse was predicted both by family socioeconomic status and neighborhood social deprivation. Controlling for family socioeconomic status, each year of living in a high social deprivation neighborhood increased the risk for drug abuse by 2%.

Conclusions: Using objective registry data, the authors found that drug abuse is highly heritable. A substantial proportion of the shared environmental effect on drug abuse comes from community-wide rather than household-level influences. Genetic effects demonstrated in twin studies have led to molecular analyses to elucidate biological pathways. In a parallel manner, environmental effects can be followed up by epidemiological studies to clarify social mechanisms.

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Psychoactive substance abuse is a worldwide public health problem (1). Since drug abuse is highly familial (2, 3), an important research goal has been to elucidate the nature of these familial risks. Previous twin studies of drug abuse have revealed an etiological role for genetic factors (4–8) and often shared environmental effects (e.g., 4, 5, 7, 8). These studies were not, however, fully representative; individuals had to agree to participate and accurately report socially undesirable behaviors.

In a Swedish nationwide adoption study of drug abuse (9), we found both genetic and familial environmental influences on risk. A follow-up nationwide study demonstrated that sibling resemblance for drug abuse risk was greater in pairs who were closer in age than in those more distant in age, and older siblings more strongly transmitted risk for drug abuse to their younger siblings than vice-versa (10). In the present study, we sought to clarify the magnitude of genetic effects on drug abuse in Sweden and gain further insight into the nature of shared environmental influences.

Using the national Swedish Twin Registry and the Multi-Generation Register to study drug abuse in twin, full, and half sibling pairs, we began by addressing four questions with twin sibling modeling. First, is the heritability of drug abuse estimated from public records similar to that found from population-based twin studies using personal interviews? Second, would we replicate evidence from an adoption sample (9) and several previous twin studies for familial-environmental effects on drug abuse? Third, our adoption study suggested differences in the transmission of drug abuse in males and females, but our sample size was too limited to address this question definitively.

This article is featured in this month's AJP Audio, is the subject of a CME course (p. 245), is an article that provides Clinical Guidance (p. 217), and is discussed in an Editorial by Dr. Hopfer (p. 140)

TABLE 1. Number of Twin and Sibling Pairs and the Tetrachoric Correlation for and Prevalence of Drug Abuse in these Pairs[a]

Sex	Pair type	Number of Complete Pairs	Number of Concordant Pairs	Number of Discordant Pairs	Tetrachoric Correlation	Standard Error	Prevalence of Drug Abuse (%)	
							Male	Female
Male-Male	Monozygotic twins	3,899	47	122	0.79	0.03	2.8	
Male-Male	Dizygotic twins	4,238	24	157	0.58	0.06	2.7	
Female-Female	Monozygotic twins	4,558	20	98	0.70	0.05		1.5
Female-Female	Dizygotic twins	4,313	8	110	0.45	0.09		1.5
Male-Female	Dizygotic twins	12,447	45	663	0.36	0.05	4.0	2.1
Male-Male	Full siblings	718,276	7,792	49,908	0.51	0.00	4.6	
Female-Female	Full siblings	641,506	1,139	20,133	0.37	0.01		1.8
Male-Female	Full siblings	1,347,324	4,063	76,392	0.32	0.01	4.3	1.7

[a] For full sibling pairs, we took all pairs within sibships up to four. With larger sibships, we picked four pairs at random.

Using the large sample sizes available, could we detect sex differences in the etiological role of genetic and environmental risk factors for drug abuse? Fourth, do environmental factors specifically affect resemblance for drug abuse in twin pairs above and beyond their effect on siblings?

In typical genetic epidemiological studies, shared environment is treated as a latent variable estimated from patterns of resemblance in relatives. While this approach captures all environmental influences, it cannot identify the environmental processes involved. As findings of genetic influences from twin studies serve to stimulate molecular genetic investigations to identify risk genes, so should findings of environmental risk factors from twin studies be followed with more refined epidemiological methods to clarify the specific environmental processes involved.

Therefore, we also used information available in the entire Swedish population about cohabitation of full siblings and paternal and maternal half siblings during childhood and adolescence to examine whether resemblance for drug abuse is associated with years of residence together in the same household or community.

Finally, to gain further insight into the nature of environmental factors affecting risk for drug abuse at the household and neighborhood level during development, we followed up the seminal observations of Faris and Dunham (11) and Dohrenwend et al. (12) that low family socioeconomic status and neighborhood-level social deprivation increase the risk for drug abuse (13). We examined, in Swedish adolescents when still living with parents—and hence unable to select themselves into particular environments—the degree to which family socioeconomic status and neighborhood social deprivation predict future drug abuse registration.

Method

We linked comprehensive register and health care data from multiple nationwide Swedish sources to form a database using the unique individual Swedish 10-digit personal identification number assigned at birth for all residents.

Details of the databases, the statistical methods used to draw inferences, descriptions of the populations, tables of parameters, and potential limitations of the analyses are in the data supplement that accompanies the online edition of this article.

Results

Twin Sibling Models of Concordance for Drug Abuse

The tetrachoric correlations for drug abuse in our twin and full sibling pairs are listed in Table 1. Five findings are noteworthy. First, the correlations were substantially higher in the male-male and female-female monozygotic pairs than in the comparable dizygotic pairs, suggesting the importance of genetic factors in the etiology of drug abuse. Second, the correlations in both male-male and female-female dizygotic pairs were greater than half those observed in monozygotic pairs, which is consistent with important contributions of familial environmental effects. This pattern was more pronounced in males than in females, suggesting a greater shared environmental effect in males. Third, the correlations for drug abuse in male-male and female-female full sibling pairs were both modestly lower than those seen in comparable dizygotic pairs, with this difference being somewhat larger in males. This pattern is consistent with the importance of a special twin environment. Fourth, the opposite-sex dizygotic and full sibling correlations were modestly lower than those seen in the same-sex pairs but otherwise follow a similar pattern. Finally, as expected given large differences in sample size, correlations were known with much greater accuracy in full siblings than in twins.

We present in Table 2 the parameter estimates for the full ACTE model (additive genetic [A], shared or common environment [C], special twin environment [T], and unique environment [E] components) and best fit model 17 along with confidence intervals. The best fit model estimated the heritability of drug abuse to be 55% in males and 73% in females. Shared family environment and twin environment were present only in males, where they accounted for 23% and 3% of the variance in liability, respectively. The remainder of variance in risk in the two sexes resulted from individual specific environmental effects. Compared

TABLE 2. Parameter Estimates and 95% Confidence Intervals for the Full ACTE and Best Fit Model[a]

	Full Model	Best Fit Model
a^2_m		
Estimate	0.39	0.55
95% CI	0.30–0.48	0.52–0.58
c^2_m		
Estimate	0.30	0.23
95% CI	0.17–0.39	0.21–0.24
t^2_m		
Estimate	0.09	0.03
95% CI	0.00–0.14	0.00–0.08
e^2_m		
Estimate	0.23	0.20
95% CI	0.17–0.25	0.15–0.24
a^2_f		
Estimate	0.71	0.73
95% CI	0.61–0.75	0.71–0.76
c^2_f		
Estimate	0.01	—
95% CI	0.00–0.05	
t^2_f		
Estimate	0.00	—
95% CI	0.00–0.05	
e^2_f		
Estimate	0.27	0.27
95% CI	0.21–0.33	0.24–0.29

[a] a^2=additive genetic effects; c^2=shared environmental effects; t^2=special twin environmental effects; e^2=individual specific environmental effects; m=male; f=female

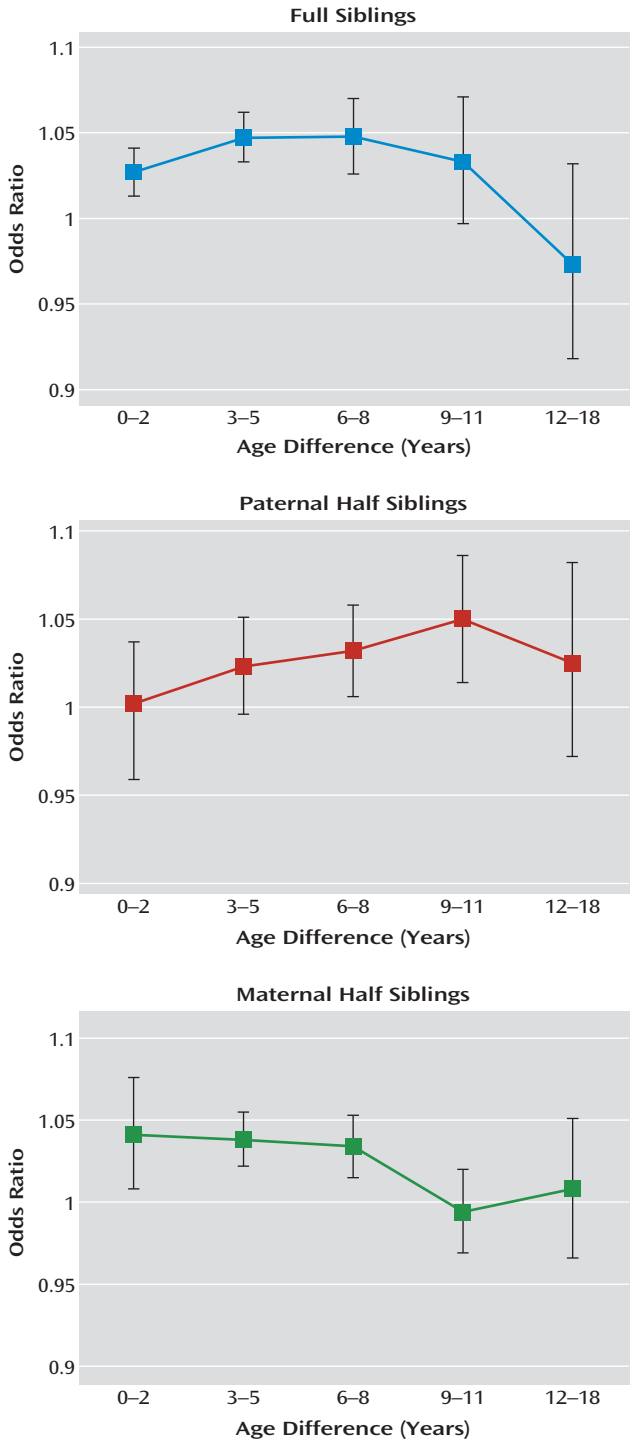
with the best fit model, the full model estimated nearly identical parameters for females, but in males it produced higher estimates for the shared family and twin environments and lower estimates for heritability.

Impact of Years of Residence in the Same Household or Community on Resemblance for Drug Abuse

Years of cohabitation in male-male sibling pairs. Among sibling pairs with at least one member with drug abuse, we predicted the probability that the pair was concordant for drug abuse as a function of the number of years cohabitating in the same household (Figure 1) and their age difference. For full siblings, concordance for drug abuse was significantly predicted by years of cohabitation for those born 0–2 years apart (odds ratio per year=1.03, 95% confidence interval [CI]=1.01–1.04, p=0.0002), 3–5 years apart (odds ratio=1.05, 95% CI=1.03–1.06, p<0.0001), and 6–8 years apart (odds ratio=1.05, 95% CI=1.03–1.07, p<0.0001). A trend was seen in the same direction for those born 9–11 years apart (odds ratio=1.03, 95% CI=0.99–1.07, p=0.07) but not for those born more than 12 years apart.

The pattern was similar in paternal half siblings except that no significant effect was seen in those born 0–2 years or more than 12 years apart. For those born 3–5 years apart, a trend was evident (odds ratio=1.02, 95% CI=0.99–1.05, p=0.09) while significant effects were seen for those born

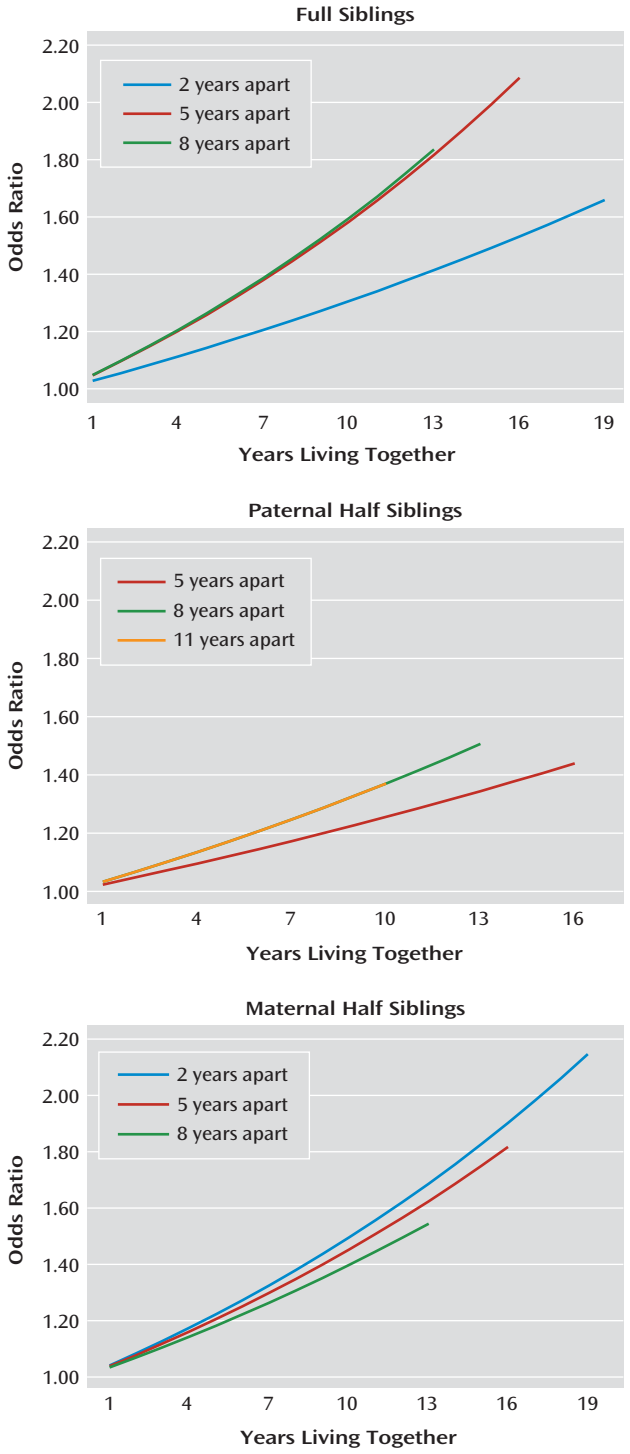
FIGURE 1. Relationship Between Living in the Same Household (Number of Years), Age Difference, and Drug Abuse Among Both Siblings in the Pair



6–8 years (odds ratio=1.03, 95% CI=1.01–1.06, p=0.0143) and 9–11 years apart (odds ratio=1.05, 95% CI=1.01–1.09, p=0.05).

For maternal half siblings, significant effects were seen for those pairs born 0–2 years (odds ratio=1.04, 95% CI=1.01–1.08, p=0.0158), 3–5 years (odds ratio=1.04, 95% CI=1.02–1.06, p<0.0001), and 6–8 years apart (odds

FIGURE 2. Relationship Between Living in the Same Household and Drug Abuse Among Both Siblings in the Pair[a]



[a] The figure illustrates the odds ratio at different number of years living together for different birth year differences.

ratio=1.03, 95% CI=1.02–1.05, p=0.0004). No trend, however, was seen for those born more than 9 years apart.

Figure 2 depicts the cumulative impact of the effects of cohabitation on resemblance for drug abuse for those analyses demonstrating a statistically significant effect. The figure illustrates the odds ratio for concordance for drug abuse in sibling pairs as a function of the number of years living together for varying age differences. Thus, for full siblings born 2 years apart, cohabitating together for the entire follow-up period of 19 years produced an aggregate odds ratio of 1.027^{19}=1.66. Note that the effects of cohabitation on concordance for drug abuse were estimated to be slightly stronger for full siblings born 5 or 8 years apart. Compared with full siblings, the cumulative effects of cohabitation were slightly greater in maternal half siblings and somewhat less in paternal half siblings.

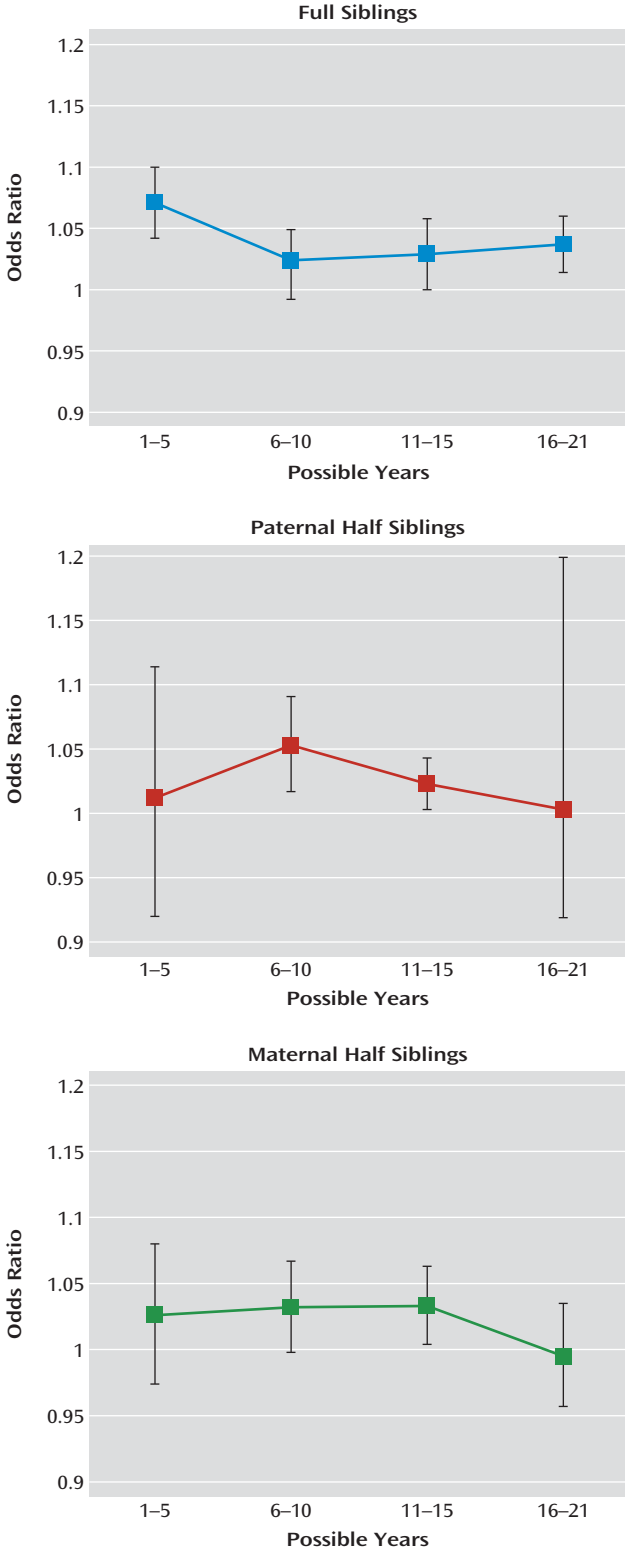
Years of living in the same small residential area in male-male sibling pairs. Within the follow-up period, we next asked whether, when not living in the same home, concordance for drug abuse was influenced by living in the same small residential area (Figure 3). For these analyses, we divided sibling pairs as a function of the number of years that they were not residing together at home and hence could have been living within the same small residential area. For full siblings, concordance for drug abuse was significantly predicted by years of residence in the same small residential area for those eligible for ≤5 years (odds ratio=1.07, 95% CI=1.04–1.10, p<0.0001), 11–15 years (odds ratio=1.03, 95% CI=1.00–1.06, p=0.05), and 16–21 years (odds ratio=1.04, 95% CI=1.01–1.06, p=0.0014), and a trend in that direction was seen for those eligible to live in the same small residential area for 6–10 years (odds ratio=1.02, 95% CI=0.99–1.05, p=0.058).

For paternal half siblings, sample sizes were quite limited for those eligible for 1–5 and 16–21 years, and we could see no effect of living in the same small residential area on concordance for drug abuse (Figure 3). However, years of living in the same small residential area did significantly affect concordance for drug abuse in paternal half siblings eligible for 6–10 years (odds ratio=1.05, 95% CI=1.02–1.09, p=0.004) and 11–15 years (odds ratio=1.02, 95% CI=1.00–1.04, p=0.02).

The effect on residing within the same small residential area on concordance for drug abuse was of similar magnitude in maternal half siblings but less significant, reaching the threshold for only one group—those eligible for 11–15 years (odds ratio=1.03, 95% CI=1.00–1.06, p=0.027). A trend in the same direction was seen for those eligible for 6–10 years (odds ratio=1.03, 95% CI=0.99–1.07, p=0.064).

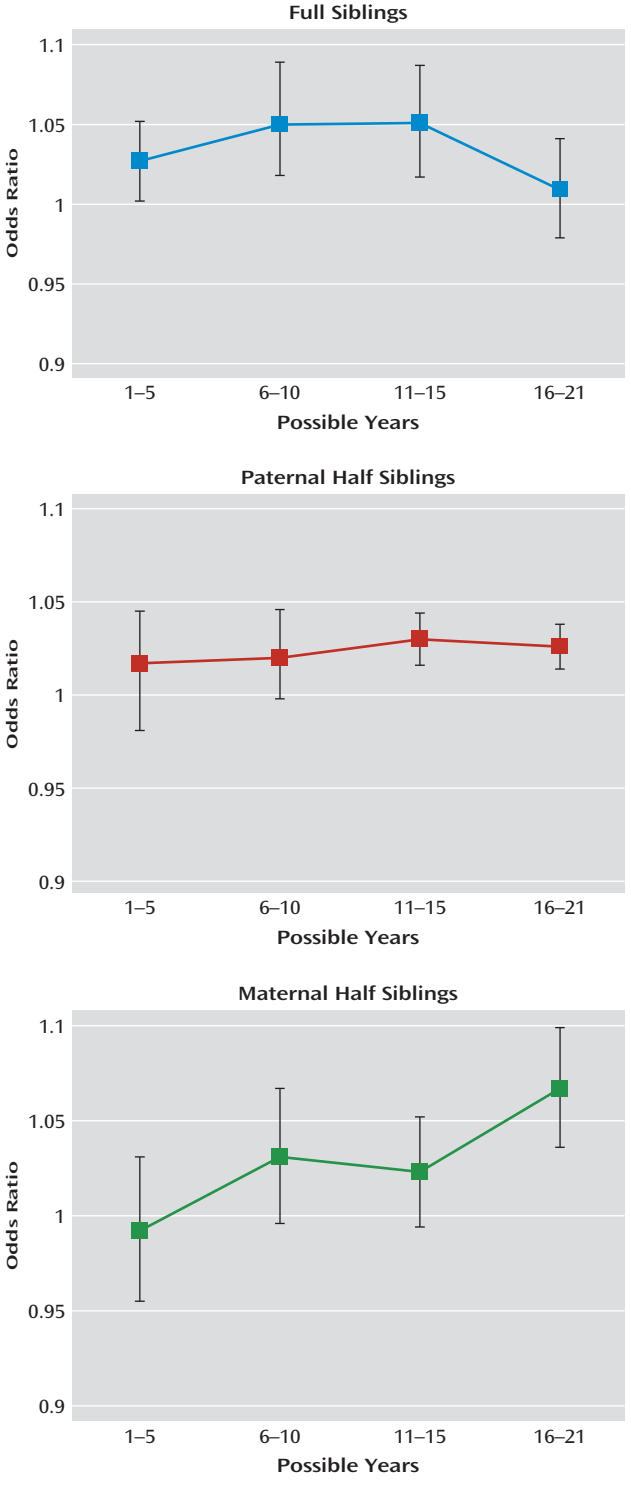
Years of living in the same municipality in male-male sibling pairs. Within the follow-up period, we next inquired whether, when not living in the same home or small residential area, concordance for drug abuse was influenced by living in the same municipality (Figure 4). For full siblings, years of residing in the same municipality were significantly associated with increased concordance for drug abuse for those eligible for 1–5 years (odds ratio=1.03, 95% CI=1.00–1.05, p=0.0305), 6–10 years (odds ratio=1.05, 95% CI=1.02–1.09, p=0.0028), and 11–15 years (odds ratio=1.05, 95% CI=1.02–1.09, p=0.0035). For paternal half siblings, years of residing in the same municipality were

FIGURE 3. Relationship Between Living in the Same Small Residential Area (Number of Years) and Drug Abuse Among Both Siblings in the Pair[a]



[a] We divided sibling pairs as a function of the number of years that they were not residing together at home and hence could have been living within the same small residential area.

FIGURE 4. Relationship Between Living in the Same Municipality (Number of Years) and Drug Abuse Among Both Siblings in the Pair[a]



[a] We divided sibling pairs as a function of the number of years that they were not residing together at home and hence could have been living within the same municipality.

significantly associated with increased concordance for drug abuse only for those eligible for 11–15 years (odds ratio=1.03, 95% CI=1.02–1.04, p<0.0001) and 16–21 years (odds ratio=1.03, 95% CI=1.01–1.04, p<0.0001). For maternal half siblings, years of residence in the same municipality were significantly associated with increased concordance for drug abuse only for those eligible for 16–21 years (odds ratio=1.07, 95% CI=1.04–1.10, p<0.0001) with a trend in the same direction in those eligible for 6–10 years (odds ratio=1.03, 95% CI=0.99–1.07, p=0.088).

Validation of cohabitation effects. Resemblance for drug abuse was substantially higher in maternal than paternal male-male half siblings (see Table S3 in the online data supplement). Although the genetic relationship is similar in the two kinds of half siblings, maternal half siblings were more likely to cohabit than paternal half siblings. Could we explain the excess resemblance for drug abuse in the maternal half siblings from their cohabitation history? In all half siblings, being a maternal half sibling pair strongly predicted higher concordance for drug abuse (see Table S5 in the online data supplement). These differences were significant for three of these five groups and at a trend level for a fourth. However, when we controlled for years of residence in the same household or for residence in the same household, the same small residential area, or the same metropolitan area the comparisons did not reach significance, with the observed odds ratios clustering around unity.

Examination of female-female and male-female pairs. In our final analyses, we jointly examined the effects of cohabitating at the level of the household, small residential area, and municipality on male-male compared with female-female pairs and on male-male compared with male-female full siblings and maternal and paternal half-sibling pairs. In each case, we examined a total of 45 comparisons (five groups differing in year of birth times three sibling types times three residential units [household, small residential area, and municipality]). The degree of resemblance for drug abuse was smaller in female-female pairs than in male-male pairs in 34 of 45 comparisons (sign test p=0.0008). However, as indicated by the interaction terms, this difference was significant in only one of these analyses in the direction of less effect in female-female pairs. The degree of resemblance for drug abuse was smaller in male-female pairs than in male-male pairs in 37 of 45 comparisons (sign test p<0.0001). This difference was significant in nine analyses, always in the direction of less effect of co-residence in male-female sibling pairs than in male-male pairs.

Family socioeconomic status and neighborhood social deprivation on future risk for drug abuse. In individuals born from 1970 to 1985 and living with a parent or grandparent, a multivariate Cox model showed that future risk for drug abuse was strongly predicted by both measures of family socioeconomic status (family income hazard ratio=1.16, 95% CI=1.12–1.19, p<0.0001), parental education (hazard ratio=1.18, 95% CI=1.16–1.21, p<0.0001), and small residential area level social deprivation (hazard ratio=1.09, 95% CI=1.07–1.11, p<0.0001) assessed when the individual was 15. We then examined the impact of years lived in a high social deprivation small residential area through age 15 on the risk for future drug abuse registration. This variable was highly significant when examined on its own (hazard ratio per year=1.03, 95% CI=1.03–1.04, p<0.0001) or when controlling for family income and parental education (hazard ratio per year=1.02, 95% CI=1.02–1.02, p<0.0001). Controlling for family socioeconomic status, an individual spending the first 15 years of his life in a high social deprivation neighborhood had an odds ratio for eventual drug abuse registration of 1.35.

Discussion

We conducted three sets of analyses that sought to clarify the etiological role of familial and community factors in drug abuse. All analyses used Swedish national samples, including twin and sibling pairs, and objective methods of drug abuse diagnosis using medical, legal, and pharmacy records.

Twin Sibling Models

Our twin sibling modeling results addressed four questions. First, despite substantial differences in methodology, the estimates for the heritability of drug abuse in this sample were within the range found in the previous twin studies, where heritability estimates for drug abuse or drug dependence varied from 31% to 74% (4, 5, 7, 14). The largest of these studies (14) produced a heritability estimate for abuse or dependence of 63%, midway between our estimates in males and females. Using ascertainment methods that did not depend on subject cooperation or on accurate long-term recall of socially undesirable behaviors, we provided an important confirmation of the results of previous twin studies that genetic factors contribute substantially to risk for drug abuse.

Second, most of the earlier twin studies (4, 5, 7, 8) and our recent adoption (9) and sibling studies (10) found familial-environmental influences on drug abuse. We found robust evidence for shared environmental effects in males. One small twin study of drug abuse explicitly examined this question and found larger estimates for shared environmental effects (c^2) in males than in females (9% and 4%) (5). Interestingly, when examining the most common substance of abuse, cannabis, their results were similar to those reported here, with a c^2 estimate of 24% in males and zero in females (5). Our results are also consistent with evidence of shared environmental effects on alcohol abuse in Swedish males (15). While the pattern of correlations suggested modest shared environmental effects in females, this effect was not detectable in twin modeling, perhaps because of the lower power resulting from the rarity of drug abuse in females.

Third, we had hoped to clarify sex differences in the patterns of risk factors for drug abuse. We found robust evidence for quantitative differences—genetic factors were considerably more important in the etiology of drug abuse in females than in males. We did not, however, find qualitative sex effects, but the presence of large shared environmental effects in only one sex makes the detection of qualitative sex effects much more difficult, because both effects predict lowered correlations in opposite-sex compared with same-sex dizygotic and sibling pairs.

Fourth, because we could study sibling pairs as well as twins, we could evaluate whether, for environmental reasons, dizygotic twins resembled one another more for their risk for drug abuse than did full siblings. Looking at the raw correlations (Table 1), we saw such a trend in both male-male and female-female pairs. In model fitting, however, we detected evidence for a special twin environment only in males. This is also likely a result of the lower power of our analyses in females. However, the impact of the special twin environment in males (3% of variance) was much smaller than that seen for shared environmental effects (23%). This result suggests that experiences unique to twin siblings had a less important impact on resemblance for drug abuse than the general background family, school, and community environment shared by all siblings. Furthermore, expanding beyond twins to include a large sample of sibling pairs in our modeling renders our findings more generalizable, as siblings are among the most common of human relationships.

In a previous analysis of resemblance for drug abuse in Swedish siblings (10), we reported that resemblance was significantly related to age difference. Those born within 2 years of one another were consistently more similar with respect to risk for drug abuse than those born more than 5 years apart. Our present findings of greater resemblance for drug abuse in dizygotic twins than nontwin siblings are consistent with this finding, as dizygotic twins are essentially siblings born at the same time.

In our Swedish studies of drug abuse, we are in the relatively unique position of being able to directly compare our findings from twins and siblings to those from an adoption study using identical diagnostic procedures in the same population (9). Heritability estimates can be obtained from our adoption findings by doubling the tetrachoric correlation for drug abuse between the adoptee and their biological parent or full sibling. These estimates agree closely with one another (34% and 29%, respectively) and are considerably lower than those estimated from our twin sample. One way to estimate shared familial environment from adoption data is to estimate the correlation in risk between the adoptee and their adoptive siblings. This equaled 0.19, close to our estimate of shared environmental effects in males.

Lower heritability estimates from adoption studies compared with twin samples has been seen for other phenotypes (16–18) and might have several causes. The degree to which this discrepancy reflects chance factors (our twin sibling sample was much larger than our adoption sample), upward biases on our estimation of heritability from the twin sample or downward biases on our estimation of heritability from our adoption sample, will require further investigation.

Impact of Years of Residence in the Same Household or Community

Our second set of analyses used the detailed information available in Swedish registries to clarify the nature of shared environmental influences on drug abuse. A critical feature of our design was to hold the degree of genetic resemblance constant and then examine whether years of residence in the same household, small residential area, or metropolitan area predicted resemblance for drug abuse in sibling pairs. In so doing, we isolated the impact of the environment that, in typical family studies, is confounded with genetic effects. We performed these analyses in three sibling groups, treating them as replicate experiments.

Five findings from these analyses are noteworthy. First, confirming results from our twin sibling modeling, in male-male full siblings, the number of years of living together in the same home was systematically related to resemblance for drug abuse. For each year of living in the same household, the probability that sibling pairs with at least one member affected with drug abuse would be concordant typically increased between 2% and 5%. Extrapolated over the expected years of cohabitation, this produced odds ratios ranging from 1.6–2.0. When compared with the total odds ratios for drug abuse among full siblings (~5.0–8.5), cohabitation accounts for a modest component of resemblance, consistent with estimates from our twin sibling models.

Second, the impact of cohabitation effects was similar in the three sibling groups. If our measures were confounded with genetic effects, they should have been much stronger in full siblings than in half siblings.

Third, the effect of cohabitation on resemblance for drug abuse was less potent in sibling pairs of very different ages. Across our three sibling groups, cohabitation effects were small and nonsignificant for pairs with more than 12 years difference in age.

Fourth, the effect of living in the same small residential area or same municipality on resemblance for drug abuse in sibling pairs was similar to that seen for living in the same household. These results suggest that much of the shared environmental effect on drug abuse comes from community-wide influences such as drug availability, school environment, or peer group effects rather than arising largely from influences specific to individual households, such as parental monitoring or the quality of parent-child relationships. To formally test this hypothesis would require us to model the three contexts (household, small residential area, and municipality) independently; however, we could not formally do this because they were nested

(e.g., living in the same household always means living in the same small residential area).

Fifth, consistent with the results of our twin sibling modeling, male-male sibling pairs were more sensitive to the effects of living in the same home or community than female-female pairs. These findings are consistent with several lines of evidence. Previous research suggests that, compared with females, males are more motivated to use psychoactive substances to conform to subgroup values (e.g., fitting in with a peer group) and more influenced by peers in their intake of drugs because they regard peer consumption as a challenge (19, 20). In a Swedish survey of high school students, Svensson (21) found that males had consistently higher levels of exposure to deviant peers than females and concluded that this arose largely because parents of girls monitored their offspring's behavior and friends more closely than did parents of boys. Particularly relevant, he found the probability of drug use to be more strongly predicted by exposure to peer deviance in males than in females (21). Both more frequent exposure to peer deviance and a greater impact of that exposure on drug use in males compared with females would likely translate into stronger peer influences in young Swedish men than Swedish women. Furthermore, in nationwide epidemiological analyses using the same definition of drug abuse employed here, we found that the risk for drug abuse in men was considerably more sensitive to neighborhood-level deprivation than it was in women (J. Sundquist et al., unpublished 2012 manuscript). Follow-up analyses in our sample pointed to two further mechanisms that might contribute to greater shared environmental influences on drug abuse in males than females. Across all the birth decades of our sample, males left home at a later age than females, with mean differences ranging from 0.6 to 3.5 years. Furthermore, in our sample, men had an earlier age at first registration for drug abuse (mean age, 27.4 years [SD=9.8]) than did women (mean age, 29.4 years [SD=11.4]) (t test for difference, $p<0.0001$). Our data suggest that the greater environmental sensitivity to drug abuse of males compared with females may result from effects at several levels including peer relationships, community-wide effects, and the ages at leaving home and starting to abuse drugs.

We are unaware of previous studies using similar methods to which our findings might be compared. Most relevant is a study of adolescent Finnish twins that included school classmates and so could estimate the percentage of school-based environmental variance (22). The strongest effects were seen for alcohol and cigarette use where school-based effects accounted for 25%−30% of the variance. Our findings are also consistent with previous analyses of full sibling pairs in Sweden, which demonstrated that concordance for drug abuse was inversely related to age differences, and transmission of drug abuse was more potent from older to younger siblings than from younger to older siblings (10).

Impact of Family Socioeconomic Status and Neighborhood Social Deprivation

Our final results examined features of the family and neighborhood environments (socioeconomic status and social deprivation, respectively) suggested by previous research to affect the risk for drug abuse and form part of the shared environment detected by our twin sibling and cohabitation analyses. Consistent with classical studies in adult populations (11, 12) and developmental studies showing that childhood poverty predisposes to externalizing outcomes such as drug abuse (23, 24), we found that risk for drug abuse was independently predicted by family socioeconomic status and neighborhood social deprivation.

Three features of these findings are noteworthy. First, a major problem in the interpretation of individual socioeconomic status effects is that of drift compared with selection. To what extent might low socioeconomic status and drug abuse be associated because drug abuse (or associated traits) predisposes to poverty or poverty predisposes to drug abuse? We have reduced this interpretational problem by examining adolescents living with parental figures because the association between neighborhood social deprivation and future risk for drug abuse could not plausibly result from adolescents selecting themselves into high social deprivation communities. However, our design cannot explicitly rule out family-level effects that might arise if genetic risk for drug abuse in children were correlated with poor occupation success in parents. Second, our results show that neighborhood level social deprivation affects the risk for drug abuse above and beyond family socioeconomic status effects. This finding substantially reduces the likelihood that our results arise from family-level drift and provides one potential mechanism for our cohabitation findings. Our results predict that individuals living in the same small residential area, but not the same household, would be correlated in their drug abuse risk, with the magnitude of the correlation increasing with years shared in that neighborhood. This is just what we observed. Third, socioeconomic status and social deprivation are surely not the only family- and community-level environmental factors affecting drug abuse risk in adolescence. Indeed, previous Swedish results showing greater risk for drug abuse when an older sibling has drug abuse than a younger sibling (25) and a large body of research on peer deviance (26–28) both suggest the importance of direct social transmission of drug use and abuse in adolescence.

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Family and Community Effects on Drug Abuse

Environmental factors, as well as genetics, influence the likelihood of drug abuse, especially in males. Nationwide Swedish registries examined by Kendler et al. revealed that sharing a household with a drug-abusing sibling increased the probability of drug abuse in another sibling. Living in the same area or city had a similar effect. Drug abuse was also associated with low family socioeconomic status and neighborhood social deprivation. These varied environmental influences, notes editorialist Hopfer (p. 140), suggest both individual and public approaches to preventing drug abuse.