Does Education Confer a Culture of Healthy Behavior? Smoking and Drinking Patterns in Danish Twins

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More education is associated with healthier smoking and drinking behaviors. Most analyses of effects of education focus on mean levels. Few studies have compared variance in health-related behaviors at different levels of education or analyzed how education impacts underlying genetic and environmental sources of health-related behaviors. This study explored these influences. In a 2002 postal questionnaire, 21,522 members of the Danish Twin Registry, born during 1931–1982, reported smoking and drinking habits. The authors used quantitative genetic models to examine how these behaviors' genetic and environmental variances differed with level of education, adjusting for birth-year effects. As expected, more education was associated with less smoking, and average drinking levels were highest among the most educated. At 2 standard deviations above the mean educational level, variance in smoking and drinking was about one-third that among those at 2 standard deviations below, because fewer highly educated people reported high levels of smoking or drinking. Because shared environmental variance was particularly restricted, one explanation is that education created a culture that discouraged smoking and heavy drinking. Correlations between shared environmental influences on education and the health behaviors were substantial among the well-educated for smoking in both sexes and drinking in males, reinforcing this notion.

Higher socioeconomic status tends to be associated with better physical health. The association is not dependent on means of measuring either socioeconomic status or physical health: Socioeconomic status can be measured by using education, income, social class, or material resources. Health can be measured by using mortality and/or longevity, general morbidity, prevalence of most specific chronic diseases, and even disease biomarkers such as blood pressure and inflammatory cytokines (1–10). The association is not confined to the most disadvantaged but is apparent across the full range of educational and economic circumstances (11). Many factors likely contribute, including access to medical care and reporting of disease incidence. The manner in which these contribute, however, is not straightforward, as the effect persists even in countries with centralized medical care (3, 4, 7, 8, 11). Health-related behaviors such as smoking and drinking are also likely contributors (2, 4, 6, 8, 11).

Well-documented declines in smoking prevalence over time have not occurred evenly throughout society (12, 13). They have been most substantial among the most educated. Thus, the least educated form increasing proportions of those who remain smokers. A differential pattern of smoking initiation and cessation (13) could be due directly to greater knowledge and/or intelligence associated with higher education at the individual level, but the pattern could also involve environmental circumstances, both social and physical, that higher education tends to foster. To the extent that different smoking norms develop within educational strata, more educated people may receive social and emotional support for not smoking, while those with less education may risk loss of social and emotional support for the same behavior (14).

Alcohol use is another health-related behavior that could have individual or cultural ties with education, although pathways may be very different from those of smoking.
Broad social trends in alcohol use have been less consistent over time than trends in smoking: Some regions and/or nations have seen dramatic increases in use in recent years, while others have seen decreases; still others have experienced little trend (15, 16).

Both smoking and alcohol use show substantial genetic as well as familial environmental influences (17), as does education (18, 19). The presence of these influences introduces the possibility that associations between education and health behaviors are also confounded by selection. Such selection is the tendency for genetic and/or family cultural influences on one outcome also to influence another, so that their association has some origin within the individual rather than being a direct effect of one factor on the other (20). This kind of selection is likely to be common but also unlikely to explain completely the links between outcomes on either genetic or family cultural levels (21). That is, people seek and/or remain in environmental situations probably because of both genetic and environmental influences, and those influences reinforce preexisting individual differences in behavioral tendencies (22).

Quantitative genetic studies can help to describe such situations and pinpoint the processes involved because they directly assess variance. Traditionally, they have been used to decompose variance into components that can be attributed to presumably independent genetic and environmental influences, but newer techniques for modeling associations between 2 traits can be used to assess how differences in one trait are related to variance in another. These newer techniques can also be used to assess how the extent of association between 2 traits may vary, making it possible to develop hypotheses about relative strengths of main environmental and selective effects, as well as about sources of any selection effects (21, 22). For example, application of these models has provided evidence that variance in physical health is lower at higher levels of education, primarily because of smaller amounts of genetic variance (23, 24). This could be because education makes it possible for people to control their lives in a manner that reduces expression of genetic vulnerabilities to disease. There is evidence that total variance in body mass index, an indicator of obesity, is also lower at higher levels of education but, unlike physical health, this appears to arise from family-related cultural processes (25).

The purpose of this study was to use these new models to explore associations of education with smoking and drinking behaviors in a large sample of Danish twins. Figure 1 shows 3 basic possibilities for the effects of education on total variance among which these models can distinguish. Although other combinations of effects could generate similar patterns, in Figure 1A, education has a continuous and direct effect on, for example, smoking across the range of education. Because the effect is constant across that range, it is only on the mean, and there is no effect on variance. Such effects are, however, rather rare. Situations such as those shown in Figure 1B, C and C, are much more common. In Figure 1B, high rates of smoking are less common in those with more education. Lack of education is, here, conceptualized as a marker of environmental stress or cultural

![Figure 1](image-url)

**Figure 1.** Hypothetical associations between smoking and education. In A, education’s effect on smoking is uniform across the range of education; in B, the variance in smoking is smaller with higher education, which would occur if greater education prevents smoking; in C, the variance in smoking is greater with higher education, which would occur if less education creates pressure to smoke.

pressure that triggers greater expression of smoking behavior, but there are large individual differences in environmental pressures and/or genetic and environmental susceptibilities to responding to them. Higher education may be associated with individual or cultural characteristics strong enough to suppress the variability that occurs among the less educated. In Figure 1C, lack of education is the primary driver through pressure to engage in smoking, and it is left more to individual choice among those with higher levels of education. Individual or cultural characteristics associated with lack of education are strong enough to suppress individual differences in smoking behavior. Everyone responds to lack of education relatively similarly in this scenario. Genetic and cultural heterogeneity are much less likely involved here than in Figure 1B.

MATERIALS AND METHODS

Source data

Our data came from the Danish Twin Registry (26), which was established in 1954. The first such registry established in the world, it includes more than 75,000 twin pairs born from 1870 to 2004 who are representative of the Danish population (26). For this study, we made use of data from a 2002 questionnaire mailed to 46,333 Registry participants born during 1931–1982. The questionnaire, approved by the Scientific Ethical Committee of the Danish counties of Funen and Vejle, included questions on smoking and drinking habits and on educational attainment. The response was 75.3% (34,944 individuals) and was higher in females than in males (78.7% vs. 75.1%) and among those born during 1931–1952 than among those born during 1953–1982 (76.2% vs. 74.7%). Response rates were very similar for monozygotic and dizygotic twins (75.9% vs. 74.9%). Twin zygosity was established by using standard questions on physical likeness and mistaken identity (27). This form of zygosity assessment is valid in this sample, misclassifying only 4% (27). Among the 34,944 respondents, there were 5,024 female monozygotic twins, 6,785 female same-sex dizygotic twins, 6,652 female opposite-sex dizygotic twins, 3,976 male monozygotic twins, 6,092 male same-sex dizygotic twins, and 5,265 male opposite-sex dizygotic twins, with the remainder missing zygosity information. We used the 21,522 members of same-sex pairs who provided zygosity information and usable data for either smoking and drinking habits or education. There were no significant differences in these variables between excluded opposite-sex twins and included same-sex dizygotic twins.

The questionnaire addressed education with 2 items. The first concerned secondary education in 7 steps ranging from completion of the seventh grade (step 0) to completion of the upper secondary school-leaving examination (step 6). The second concerned vocational and academic education beyond secondary school in 6 response categories ranging from none (category 0) through more than 4 years of academic education (category 5). We consolidated information in these 2 questions by summing their numeric values to form a scale ranging from 0 to 12. Participants were asked about smoking status and history, including present smoking status, number of years smoked, when they had quit (if relevant), and quantities smoked. We used this information to calculate pack-years of smoking exposure. They were also asked to indicate numbers of alcoholic beverages currently consumed each week in categories of beer, strong spirits, red wine, white wine, and dessert wine. We combined responses across beverage categories.

Table 1 provides descriptive statistics for these variables. Sex differences were substantial, making it likely that results would differ by sex. We thus analyzed women and men separately throughout. The participants were relatively uniformly distributed throughout the 1931–1982 birth cohorts, with women born on average in 1958 and men born on average in 1957. The average level of education corresponded to completion of the secondary school examination or some combination of secondary education without examination and supplementary vocational training. Women had slightly more education than did men (standard deviation, 0.17). Women smoked and drank considerably less than men and showed less variance in these health-related behaviors as well. Men smoked 0.20 female standard deviations more than women and drank 0.95 female standard deviations more. All variables showed associations with participant year of birth; those with earlier years of birth were less educated and smoked and drank more. Birth-year effects can inflate the similarity between co-twins because twins are the same age (28). To avoid this, we regressed the effects of age and age squared from both variables separately by sex. The smoking and drinking variables were positively skewed, so we log-transformed them prior to further analysis. Our basic

<table>
<thead>
<tr>
<th>Variable, Mean (SD)</th>
<th>Year Born</th>
<th>Education, Score*</th>
<th>Smoking, Pack-Years</th>
<th>Alcohol Consumption, Units/Week</th>
</tr>
</thead>
<tbody>
<tr>
<td>Females (n = 19,037)</td>
<td>1958.02 (13.66)</td>
<td>6.79 (3.34)</td>
<td>11.32 (14.91)</td>
<td>5.27 (5.80)</td>
</tr>
<tr>
<td>Males (n = 15,907)</td>
<td>1957.04 (13.69)</td>
<td>6.23 (3.40)</td>
<td>14.35 (18.08)</td>
<td>10.76 (10.68)</td>
</tr>
</tbody>
</table>

Abbreviation: SD, standard deviation.

*Education was scaled so that completion of grade 7 with no further training was scored 0, and completion of education beyond a university degree was scored 12. Intermediate scores reflected both greater formal schooling and apprenticeship training.
results were robust to several alternative combinations of variable definitions and transformations.

**Analytical approach**

Our analyses relied on the standard assumption that variance in smoking and drinking could be attributed to 3 sources: additive genetic influences (A), shared environmental influences that made twins in the same pair similar but distinguished among twins from different pairs (C), and nonshared environmental influences including measurement error that made twins different from each other (E). Estimates of these variance components are derived by comparing the similarity of members of monozygotic twin pairs relative to that of dizygotic twin pairs (29). Because monozygotic twins share all their genes and dizygotic twins share on average 50% of their segregating genes, smoking or drinking correlations between monozygotic twins that are twice the correlations for dizygotic twins indicated that twin similarities in the relevant behaviors were due primarily to additive genetic influences. Shared environmental influences were indicated by dizygotic correlations greater than one-half the monozygotic correlations. Monozygotic twin correlations less than 1.0 indicated nonshared environmental influences.

This model can be extended to estimate genetic and environmental contributions to the covariance between education and health-related behaviors. The extended model includes A, C, and E influences on education that also contribute to the behavior and A, C, and E influences that are unique to the behavior. Genetic correlation (rA) is the standardized genetic covariance. Like ordinary correlations, it varies from 1.0 to −1.0. It provides an index of the degree to which the same genetic influences contribute to education and the behavior. Similar calculations produce shared (rC) and nonshared (rE) environmental correlations. Unlike ordinary correlations, these correlations can be present even when 1 of the 2 variables is constant at the observed level, because different individuals with that value of the observed variable can attain that value through many different pathways of genetic and environmental influences.

These models provide average population estimates of A, C, and E influences, assuming that these influences are constant throughout the population with no interactions among them and independent of each other with no correlations among them. It is increasingly acknowledged, however, that these assumptions are probably often violated (30). More sophisticated quantitative genetic models relax these assumptions so the possibility that education moderates the influences on smoking can be considered. For example, the rC between education and smoking could be greater at higher levels of education. In contrast to most behavior genetic studies, our purpose in fitting these more sophisticated models was not to indicate the importance of genetic relative to environmental influences but to reveal something about the transaction processes between them.

We carried out our analysis using the Purcell (31) genetic-environmental moderator model as implemented in Mx software (32) with maximum likelihood estimation, so that all twin data could be included, regardless of co-twin data availability. The model enabled us to measure the following: 1) differences in variance in smoking and drinking with level of education; 2) the extent to which variance in smoking and drinking could be attributed to genetic and environmental influences; and 3) the extent to which these same influences contributed to both education and smoking or drinking, as reflected by genetic and shared and nonshared environmental correlations. Because we were interested in differences in overall variance with education as well as differences in means, we estimated absolute genetic and environmental variance components rather than the more commonly estimated proportions of variance. In some situations, this model can produce spurious or uninterpretable results (33), but those situations did not apply here: The positively skewed smoking and drinking variables were reasonably normally distributed when log-transformed, moderation was primarily on variance unique to smoking and drinking, and variances in smoking and drinking were not dependent on their levels, leaving the results robust to transformation of scale. Other combinations of transformation and definition of variables produced similar results.

Model parsimony dictated the results presented. We dropped terms indicating the moderating effects of education on smoking and drinking when they did not create significant change in model −2 × log-likelihood. We evaluated the appropriateness of doing this using the information theoretic fit statistics Akaike’s Information Criterion (34) and Bayesian Information Criterion (35). Relevant statistics are available from the first author on request. We dropped nonsignificant moderating terms not to deny the potential existence of smaller moderating effects on paths that happened not to be significant in this particular sample but to focus attention on the effects of education most important in these data. Given our large sample, the effects we dropped were not of substantive importance.

**RESULTS**

**Moderating effects**

Figure 2 shows how mean levels and variance in smoking and drinking differed with level of education, separately for women and men. The scales were standardized on the full sample, so sex differences in means and overall variances are clearly visible. The thicknesses of the bands show total smoking and drinking variances at different levels of education (x-axes), and the overall levels of the bands on the y-axes show mean effects. In both smoking and drinking, there was much more variation among people with low levels of education. There was also greater variance in men than...
women, particularly in drinking. In general, variance at 2 standard deviations above the mean level of education was about one-third that at 2 standard deviations below.

Figure 3 shows variances separately by source, again separately for women and men. Additive genetic variance is shown with light-gray bands, shared environmental variance with medium-gray bands, and nonshared environmental variance with black bands. There was substantially less shared environmental variance (black bands) in both smoking and drinking in both women and men with higher levels of education. With the exception of smoking in men, there was almost no shared environmental variance at the highest levels of education. There was also less additive genetic variance (light-gray bands) in smoking at higher levels of education in both sexes, but the moderating effect was smaller and not significant for drinking in women. There was less nonshared environmental variance (black bands) in smoking at higher levels of education in both sexes, especially women, but no effects on drinking. Thus, variation in smoking and drinking was moderated by level of education, and the principal target of moderation was shared environmental variation, which was restricted markedly at higher levels of education.

Genetic and environmental correlations

Observed correlations between smoking and education were −0.17 in women and −0.21 in men. For drinking and education, they were 0.12 in women and 0.02 in men. Figure 4 shows how the mean levels and genetic and shared and nonshared environmental correlations varied with level of education. Solid black lines show mean levels of smoking or drinking (y-axes) in standard units at different levels of education (x-axes). As the correlations indicated, there was more smoking among people with less education, but the drinking level was higher in women with more education and essentially independent of education in men. The 3 broken lines in each panel of Figure 4 show how the extent of overlap in the relevant sources of influences on education and on smoking or drinking (y-axes) varied with level of education (x-axes). The lines representing genetic and nonshared environmental correlations showed little if any
difference with level of education, and these correlations were small.

In sharp contrast, the uneven broken lines in Figure 4 denoting shared environmental correlations showed substantial differences with level of education in both smoking and drinking in both sexes. For smoking, the correlations were small (negative) when the level of education was low but became increasingly strongly negative at higher levels of education. Negative correlations indicated that familial and local community influences making twins similar and contributing to more education were associated with less smoking. The increasing strength of these correlations with level of education indicated that cultural effects were stronger when the level of education was high. For drinking, the correlation was small in women and shifted from negative to positive with higher levels of education. In men, the correlation was small but positive when education was low and increased in strength with more education. This suggested that familial and community influences making twins similar and contributing to more education encouraged
drinking, but only in moderation, as drinking’s variance was also restricted.

**DISCUSSION**

This study supported the existence of the kind of situation depicted in Figure 1B for both smoking and drinking. With higher levels of education, there was less variance, and most of the variance that was restricted was shared environmental for both behaviors in both sexes. This suggests that more education may be associated with cultural (not individual) characteristics that are strong enough to suppress the variability in smoking and drinking occurring among the less educated. Further evidence for this was provided by the shared environmental correlations, which were small when education was low but very strong for smoking in both sexes.
when education was high. This indicated that family and cultural influences contributing to both high educational attainment and restriction of smoking behavior had substantial effects, but the corresponding family and cultural influences contributing to low educational attainment were largely independent of family and cultural influences on smoking. In other words, policies aimed at reducing smoking through education and legal and social sanctions may have been effective, at least among the well-educated.

The pattern involving family and cultural effects for alcohol use was similar in males, although restriction in variance was, if anything, associated with some increase in use. Whatever family and cultural effects prevailed appeared to be related not to quantity of use but to other factors, such as the kind of alcohol consumed or frequent versus binge use. This bears exploration in future research. In females, more education was associated with greater alcohol use through restriction of shared environmental variance, but the shared environmental variance was not closely associated with that on higher educational attainment as for males. This also bears exploration in future research. In general, there was more variance in both smoking and drinking in males than in females. Levels of smoking were higher among males with less education than among females with less education, but the reverse was true among those with more education. Levels of alcohol use were almost uniformly lower in females than males across the range of education.

This study has limitations despite its large and well-characterized population-representative nationwide sample. The smoking and drinking variables were self-reported and are subject to reporting biases and failures of recall. Moreover, education may influence how people make self-reports, people are often still in school when they begin smoking, and estimates of genetic influences could include systematic sources of measurement error. However, self-reports have the advantage that they can comprehensively and sensitively summarize people’s health behaviors, and eventual educational attainment generally reflects long-standing academic achievement. Education cannot actually be measured as continuous, although we treated it as such. The sample had a wide range of ages. Although it was population representative of those ages at the time of survey, because smoking has declined over time and is associated with mortality, the sample was differently representative of specific age for the individual age cohorts it contained. Age was associated with both health behaviors and education. Our models accounted for its direct effects, but there may have been indirect effects, such as differences in socioeconomic status at particular levels of education for which we did not account. Clearly, results apply to twins in the age and geographic group studied here. Finally and importantly, our characterization of influences as additive genetic included epigenetic and pre- and perinatal effects involving environmental control of gene expression that operated to make monozygotic twins more similar than dizygotic twins. Similarly, our characterizations of influences as shared and nonshared environmental included any effects that made members of twin pairs similar regardless of zygosity (shared) and different from each other (nonshared).

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