IBS
POPULATION PROGRAM ■
INSTITUTE OF BEHAVIORAL SCIENCE ■
UNIVERSITY OF COLORADO AT BOULDER ■

WORKING PAPER

Schools and the Heritability of Smoking Behaviors: Theoretical and Methodological Considerations

Jason D. Boardman
Jarron M. Saint Onge
Brett C. Haberstick
David S. Timberlake
John K. Hewitt

September 2006

Population Program POP2006-07

SCHOOLS AND THE HERITABILITY OF SMOKING BEHAVIORS: THEORETICAL AND METHODOLOGICAL CONSIDERATIONS*

Jason D. Boardman [a, b]
Jarron M. Saint Onge [a, b]
Brett C. Haberstick [c]
David S. Timberlake [c]
John K. Hewitt [c, d]

* This paper is part of a larger study funded by the National Institute of Child Health and Human Development (K01 HD 50336). This research uses data from Add Health, a program project designed by J. Richard Udry, Peter S. Bearman, and Kathleen Mullan Harris, and funded by a grant P01-HD31921 from the National Institute of Child Health and Human Development, with cooperative funding from 17 other agencies. Special acknowledgment is due Ronald R. Rindfuss and Barbara Entwisle for assistance in the original design. Persons interested in obtaining data files from Add Health should contact Add Health, Carolina Population Center, 123 W. Franklin Street, Chapel Hill, NC 27516-2524. Direct all correspondence to: Jason D. Boardman, Department of Sociology and Institute of Behavioral Science, Univesity of Colorado at Boulder, 327 UCB, Boulder, CO 80309-0327; phone: 303-735-0172; email: boardman@colorado.edu.

a. Department of Sociology, University of Colorado at Boulder
b. Institute of Behavioral Science, University of Colorado at Boulder
c. Institute for Behavioral Genetics, University of Colorado at Boulder
d. Department of Psychology, University of Colorado at Boulder

SCHOOLS AND THE HERITABILITY OF SMOKING BEHAVIORS: THEORETICAL AND METHODOLOGICAL CONSIDERATIONS*

Abstract

In this paper we develop and test theoretical models that position the social environment (both normative and institutional) as a critical component of sociological inquiry into behavioral genetics. We focus on schools as important social institutions that condition the magnitude of genetic influences on smoking behaviors. Specifically, we develop and test five hypotheses that examine gene-environment interactions using the sibling-pair data from the National Longitudinal Study of Adolescent Health. Based on estimates using traditional quantitative genetic techniques we find that 59 percent of the variation in smoking behaviors is heritable in nature. However, when this estimate is obtained using a multi-level modeling framework with adolescent pairs nested within schools, we observe a significant reduction in the overall estimate ($h^2 = .46$). Most importantly we also demonstrate significant variation in heritability across schools. We find that heritability is significantly reduced within schools characterized as having strong norms regarding smoking behaviors and within schools with limited resources.

Introduction

Although some have characterized *outside the body* and *inside the body* explanations as fundamentally at odds with one another (Duster 2005), there are strong theoretical reasons (Shannahan and Hofer 2005; Deater-Deckard and Mayr 2005) and corresponding empirical evidence (Turkheimer et al. 2003; Purcell 2002; Purcell and Sham 2002) to suggest that a full understanding of complex health-related behaviors such as smoking requires information on *both* inside and outside of the body processes. That is, smoking-related behaviors are in part genetically oriented but the expression of a gene that may impact tobacco use is only properly understood when situated in a particular social or environmental context. Therefore, if the degree to which behaviors such as smoking are heritable is conditional upon the social contexts that frame the normative and institutional contours of people's lives, then it is important to develop theory, hypotheses, and methods specifically designed to address the interaction between genes and environments.

This paper makes three important contributions. First, previous research suggests that roughly twenty-percent of the variation in current smoking behaviors among adolescents is due to differences across schools (Ennet et al. 1997). Therefore, we examine the role of adolescents' schools in shaping exposure to differential smoking norms and the role of norms in limiting or enabling genetic characteristics that may influence adolescent cigarette usage. In doing so, we build upon other substantive work examining gene-by-environment (GE) interactions for smoking outcomes (Timberlake, Rhee, Haberstick et al. 2006; Slombowski et al. 2005). Second, there is a mismatch between the level at which hypothesized GE interactions are believed to occur and the level at which they are measured. As Shanahan and Hofer (2005) state, "[GE interactions] are likely to reflect manifold aspects of context that exert their influence on the person as a set of variables, not individually." (71) As contexts house multiple social domains, it is important to consider well-defined social and institutional contexts such as schools as relevant sites for not only for GE interactions but also EE (Environment-Environment) interactions. Third, we provide a methodological contribution to this field by extending the DeFries-Fulker regression model (DeFries and

Fulker 1985) to a multilevel framework in order to quantify the extent to which the social environment moderates the heritability of smoking behaviors and to address some remaining methodological shortcomings and critiques (Eaves 2006). This contribution is particularly relevant to social scientists who are recently entering into these discussions.

Smoking among adolescents

According to estimates from the Monitoring the Future study, roughly fifty percent of twelfth-graders in the United States have experimented with cigarettes, fourteen percent report daily usage, and nearly seven percent report smoking at least a half of a pack of cigarettes every day (Johnston et al. 2005). Over the past 30 years, the rate of cigarette experimentation among twelfth graders fell from three in every four students to just one in every two. Despite these declines, cigarette usage among adolescents continues to be an important public-health concern because the bulk of cigarette initiation occurs at or around the age of adolescence (Breslau et al. 2001) with roughly 90% of current smokers beginning during this period (USDHHS 1989). Despite these improvements, there is evidence to suggest the declines in adolescent smoking have leveled-off (Johnston et al. 2005) and cigarette smoking remains the single most important behavioral risk factor associated with U.S. adult mortality (US Department of Health and Human Services [USDHHS] 2000). Although there is some dispute regarding the best way to characterize excess deaths due to smoking (Rogers et al. 2005), a recent report suggests that cigarette smoking and smoke exposure accounted for 438,000 premature deaths in the U.S. between 1997 and 2001 totaling roughly 5.5 million years of potential life lost (Centers for Disease Control and Prevention [CDC] 2005). Therefore, understanding the factors that increase the likelihood regular tobacco use, especially among adolescents and young adults, continues to be a research priority.

[Table 1 about here]

Within medical and behavioral sciences, the etiology of cigarette usage is believed to have a strong genetic component (Todd and Mason 1954). By comparing the smoking concordance among sibling and twin pairs, roughly 50-60% of the variance in smoking behaviors is believed to be heritable in nature (Batra

et al. 2003; Heath and Madden 1995; Sullivan et al. 1999) and the remaining 40-50% is due to environmental factors. Although different in magnitude, there is strong evidence for a genetic basis for tobacco use regardless of the degree of use or abuse. For example, in a widely cited paper, Carmelli et al. (1992) find a greater concordance in experimentation, current smoking, and cessation among monozygotic (MZ) compared to dizygotic (DZ) twin pairs. Further evidence for a genetic basis to regular tobacco use comes from studies of adult twin pairs raised in the same home compared to a comparable cohort of adult twins raised apart from one another (Kendler et al. 2000). These authors report that the overall heritability estimate for regular tobacco use among twin pairs raised apart is 61% which closely resembles the estimate of 59% obtained in a meta-analysis of quantitative genetic studies on persistent smoking behaviors (Li et al. 2003).

In addition to genetic factors, both social and environmental factors have been found to increase the risk of regular cigarette smoking. Using data from Wave III of Add Health, Hu et al. (2006) present evidence describing the association between cigarette usage and a number of factors describing the contours of young adult's lives. For example, they find strong associations with social and economic (e.g., race and education), family (e.g., marital and family status), psychological characteristics (e.g., self-esteem, depression, and novelty seeking behaviors), and social-interactive (e.g., smoking status of friends) to be strong predictors of smoking behaviors. Their work builds upon a paper by Alexander et al. (2001) who show that the prevalence of cigarette usage across three important social domains (family, friends, and school-mates) strongly predicts the likelihood that adolescents will smoke. For example, adolescents involved with peer-networks in which at least half of the network smokes and those with best friends that smoke are twice as likely to smoke compared to those adolescents with peer groups and close friends that do not smoke (Slombowski et al. 2005).

Some of the strongest evidence describing the social antecedents to regular tobacco use comes from studies linking school-level smoking prevalence to adolescent smoking behaviors. For example, Ellickson et al. (2003) show that the proportion of students who smoked in an adolescent's school impacted his or her risk of smoking one year after school attendance and, more importantly, these effects were independent of

the smoking behaviors of their peers. Likewise, Eitle and Eitle (2004) show that school effects also operate independently of larger levels of aggregation. In their study, they find that school-level variables regarding the normative environment of smoking and institutional capacity of schools strongly predicted smoking incidence at the schools but that these effects operated independently of county-level characteristics such as population density and socio-demographic composition. In part, these results assuage concerns that school effects are simply picking up social processes that are situated in the more broadly defined social-geography rather than school dynamics, per se.

Less work has jointly considered both genetic and social factors and the interaction between these characteristics as potential determinants of cigarette usage among adolescents and young adults. Moreover, studies that explore these issues tend to focus on social and demographic characteristics of individuals or families (Heath et al. 1999; Lerman et al. 1999; Slombowski et al. 2005; Timberlake, Rhee, Haberstick et al. 2006) rather than contextual or compositional differences of larger social contexts (e.g., neighborhoods or schools) as related to divergent heritability estimates. Because larger social contexts impact smoking initiation and persistence (Ennet et al. 1997; Chuang et al. 2005; Eitle and Eitle 2004; Ellickson et al. 2003), work situated at this level of aggregation speaks more directly to ongoing discussions regarding the ways in which GE interactions are theoretically understood and modeled empirically (Johnston and Edwards 2002; Shannahan and Hofef 2005).

If the theory describing the anticipated relationship between genes, the environment, and a particular outcome characterizes the environment as a construct beyond that of an individual (Ryff and Singer 2005), then efforts should be made to properly position individuals within these environments when engaging in standard behavioral genetic analyses. Given the critical timing of cigarette usage during late adolescence (Breslau et al. 2001; Hu et al. 2006), the normative and institutional influences of adolescents' schools make a particularly useful level at which to examine GE interactions for smoking behaviors. Our study takes advantage of the fact that the National Longitudinal Survey of Adolescent Health (Add Health) is both a

school-based design with an oversample of twin-pairs to develop a statistical method that properly specifies the moderating role of the environment.

School context and heritability

Shanahan and Hofer (2005) provide a useful typology of GE interactions that anticipates differential heritability as a function of the social environment. In our paper, we focus on the models that they call social control. According to these authors, social control refers to both social norms and structural capacity (both constraints and resources) that may limit or facilitate an individual's efforts to meet his or her needs. Because most persons within highly controlled social contexts will exhibit similar outcomes regardless of their genetic characteristics, the relative contribution of heritability to overall trait variance is expected to be reduced in these environments. Likewise, in social areas characterized by relatively low levels of social control, individual's behaviors may be influenced more by genetic characteristics and heritability estimates for a particular trait will be significantly higher. Kendler et al. (2000) find strong evidence for social control models of heritability in their study of tobacco use among twins raised together and separately. They find that genetic factors became more influential for women's tobacco use over successive cohorts because, compared to men, social restrictions on women's tobacco use decreased substantially over this same period. This has been found for a variety of outcomes. For example, Dunne et al. (1997) compare the heritability estimates for age of first intercourse among an early (1922-1952) and a later (1952-1965) cohort in Australia and find a much higher heritability estimate in the later cohort. Koopmans et al. (1999) find heritability estimates for the initiation of alcohol use to be near zero among girls raised in religiously oriented homes but roughly 40% among girls residing in homes that did not currently practice a religion. In all cases, the more restrictive social environment –strong norms against out of wedlock sexual activity or norms against alcohol use- leads to a reduction in overall heritability.

Although normative control models explicitly point to cultural differences as the primary mechanism responsible for different heritability estimates, very few studies have made efforts to measure beliefs and values regarding particular behaviors. Rather, these environments are assessed indirectly as a function of

group membership (e.g., cohort or religion). An alternate method to assess the normative environment is to differentiate among groups of persons based on the prevalence of particular behaviors. In this case, the prevalence of smoking within an adolescent's school may serve as an important backdrop to assess the heritability of smoking behaviors where social control manifests at two ends of a continuum. That is, schools with a smoking prevalence at or near the average may not have established strong norms regarding smoking or non-smoking but schools with very low smoking rates may impose very strict social sanctions on those who transgress this norm (the smokers). Likewise, students enrolled in schools with very high rates of smoking may feel social pressures to engage in these activities regardless of genetic tendencies one way or the other. According to the social control model, heritability estimates will be the highest among students attending schools with average rates of smoking and the lowest among students from both high and low smoking prevalence schools. In other words, if this social control model adequately described this process, then we would expect to see a curvilinear association between school-level smoking prevalence and the heritability of smoking behaviors.

Social control may also operate through the structural and institutional environment. Relatively affluent social environments characterized by greater resources, more stability, and less social disorganization, may enable a priori genetically oriented characteristics to manifest. This might explain why Turkheimer et al. (2003) find that the heritability of IQ is significantly higher among siblings from more affluent families compared to those from families with limited access to socioeconomic resources. Similarly, Rowe et al. (1999) report a heritability estimate for IQ of 74% among adolescents whose parents have at least a high-school education but only 26% among those whose parents did not complete high school. Although little work has focused on the structural-control models of heritability for complex health-related behaviors, it is also possible that we would find greater heritability for smoking behaviors in the social context of more affluent compared to less affluent schools, regardless of the normative environment of smoking in the school.

Although these models are important, they do not address the important discussion of environment-by-environment (EE) interactions. That is, social control models differ in their relative emphasis of normative or institutional forms of control but there are reasons to believe that these effects are also contingent upon their respective levels. We see two possible models. First, institutional deprivation may outweigh normative pressures. Said differently, normative social forces may also require material or institutional resources to enforce their proscribed sanctions. In this *resource application model*, evidence for social-normative control will only be found in more advantaged school settings.

An argument can also be made that social-normative forces will be more effective in shaping smoking behaviors in environments characterized by fewer institutional resources. According to this *relative capacity model*, student norms regarding smoking behaviors will strongly impact smoking behaviors within disadvantaged schools because of the strength of social norms vis-à-vis the institutional capacity of schools to enforce rules and regulations regarding smoking behaviors. As others have pointed out, micro-environments may be particularly salient in the enforcement and regulation of day-to-day life activities compared to more broadly encompassing institutions, especially in areas characterized by relatively low resource availability (Anderson 1989). Absent requisite resources -despite efforts to dissuade students from smoking- the normative environment established by students within relatively disadvantage schools will have a particularly strong impact on specific behaviors.

Methodological Considerations: Another Mixed DeFries-Fulker Regression Model

(1) $$y_2 = a + b_1 p_1 + b_2 g + b_3 (y_1 g) + e_i$$

The DeFries-Fulker (DF) model is an efficient and robust method to decompose the variation of some outcome into genetic and environmental components (DeFries and Fulker 1985). This model relies on data obtained from sibling pairs and contains two primary parameter estimates (see equation 1). The DF model predicts the outcome of the 2nd sibling of a pair (y_2) as a function of the sibling's score on the same outcome (y_1), a measure of genetic similarity –proportion of genes shared by the pair- (g), and an interaction between genetic similarity and the siblings score ($y_1 g$). Two of the parameter estimates obtained from this model (b_1

and b_3) describe the relative contribution of shared environment (c^2) and heritability (h^2), respectively and the remaining proportion is due to nonshared environmental characteristics (e^2). Although this most basic DF model has undergone considerable modifications (Purcell 2003), it remains a widely used technique to assess the genetic contribution to a trait's overall variation (Cherny et al. 1992; Rende 1993; Rodgers and McGue 1994). Because this model also allows for the inclusion of covariates, it is possible to examine the interaction between the heritability estimate (b_3) and some individual or contextual variable such as family-level socioeconomic status (Turkheimer et al. 2003).

(2) $$y_{2ij} = a + b_1 y_{1ij} + b_2 g_{ij} + b_3 (y_{1ij} g_{ij}) + e_{ij} + u_{0j} + u_{1j} (y_{1ij} g_{ij})$$

We propose a multilevel model variant of the DF model that specifically addresses the potential moderating role of the social environment on heritability estimates. Building on similar work (Guo and Stearns 2002), our modification involves the inclusion of parameters capturing level-2 variance in the slope coefficients for both shared environmental variance and heritability. Toward this end, equation 2 extends the DF model (equation 1) in two ways. First, the estimate for u_{1j} value specifies an offset to the heritability estimate for pairs who currently attend the *j*th school. This allows for a test of level-2 moderation without relying on researchers to properly specify a particular level-2 characteristic. In the context of the current study, significant variance in h^2 across schools (σ^2_{u1}) provides evidence that schools moderate the magnitude of heritability. Second, with this information, researchers can then identify the source of this variance using a variety of different techniques (Raudenbush and Bryk 2002). This same approach can be extended to include school-specific offsets to the shared environment estimate.

Building on the methodological framework, we test six hypotheses regarding the social moderation of smoking behavior heritability.

(1) *Direct genetic effect*: the relative contribution of heritability to variation in smoking behaviors will be consistent across schools ($\sigma^2_{u1} = 0$; there will be no variation in the magnitude of the slope coefficient for heritability (b_3)).

(2) *Social moderation effect*: the relative contribution of heritable influences to overall variation in smoking behaviors will vary significantly across schools ($\sigma^2_{u1} > 0$).

a. Main Effects

   i. *Institutional control*: heritability estimates will be higher within schools with greater access to material and social resources.

   ii. *Normative control*: heritability estimates will be significantly reduced within schools with relatively strong norms about smoking behaviors; heritability estimates will be the highest in schools with average smoking rates and the lowest in schools with a high or low prevalence of smoking.

b. Environment-Environment Interaction Effects

   i. *Resource application*: normative control (as evident by a curvilinear association between smoking prevalence and heritability) will be higher in more affluent school settings.

   ii. *Relative capacity*: normative control will be evident across most schools but it will be particularly strong in disadvantaged school settings.

Data

All data in these analyses are drawn from the National Longitudinal Study of Adolescent Health (Add Health). This study examines health and health-related behaviors among a nationally representative sample of adolescents in 7^{th} through 12^{th} grades. In 1994, roughly 90,000 adolescents from 134 schools completed questionnaires about their daily activities, health-related behaviors, and basic social and demographic characteristics. Respondents were then followed-up in three in-home studies (Waves I-III) with more detailed questions across a number of important domains. Because six schools had relatively poor reporting on smoking prevalence, we only use data from 128 schools.

This paper takes advantage of the initial data collection within schools. Because nearly all students in the schools responded to the initial survey, it is possible to measure aspects of schools that are otherwise difficult to assess. The two school-level measures used in these analyses were calculated by aggregating responses from the survey across schools. *Smoking prevalence* measures the percent of students who reported that they have tried smoking cigarettes in the past 12 months. Smoking prevalence for schools ranged from 5-55%. *Percent Minority* measures the number of students who are not non-Hispanic and white. Minority percent ranged from 4-100%. Although percent minority and smoking prevalence are negatively associated with one another ($r = -.28$, $p < .01$), the distribution of schools is such that there are representative schools from nearly all combinations of smoking and minority prevalence (see Appendix 1).

The primary dependent variable, smoking behavior, is obtained from the Wave II of the study. Respondents were asked if they had ever smoked cigarettes, if they had ever smoked cigarettes regularly ("that is, at least 1 cigarette a day for 30 days"), and then how days per month they smoked and how many cigarettes that they typically smoked on each occasion. Similar to other work in this area (Timberlake, Haberstick, Lessem et al. 2006) we multiply frequency and quantity to assess overall smoking levels. The standardized value of this product represents the main dependent variable used in these analyses. Our analysis of smoking behavior is limited to the sibling data including 245 MZ twin, 355 DZ twin, 900 full-sibling, and 253 half-sibling pairs.

Findings

[Table 1 about here]

[Table 2 about here]

Table 1 presents tetrachoric and Pearson correlation coefficients for smoking behaviors among a nationally representative sample of sibling and twin pairs. Using a traditional estimate of twice the difference of the correlations among indentical (r_{mz} = .63) and fraternal (r_{dz} = .38) twin pairs (Bouchard and Propping 1993), roughly 50% of the variance of total smoking is considered heritable in nature which is in line with other heritability estimates for smoking (Rende et al. 2005; Sullivan et al. 2005 Heath et al. 1999; Heath and Madden 1995).

The Add Health study contains full and half-sibling pairs which are used in the multilevel DeFries-Fulker regression estimates presented in table 2. These estimates address the degree to which the social environment moderates the heritability of smoking behaviors among adolescents. Model 2 provides DF regression estimates for heritability and shared environment of .59 and .27, respectively. That is, according to these values, 59 percent of the variation in smoking behaviors is considered heritable in nature whereas 27 is due to environmental characteristics shared by siblings, and the remaining 14% due to environmental characteristics that the siblings do not share with one another (DeFries and Fulker 1985).

The remaining models in Table 2 denote one of the primary contributions of this paper. Namely, using the multilevel model framework one can allow the slopes associated with both heritability and shared environment to vary across schools. The significant value for the value in Model 3 ($\sigma^2_{u1} = .85$) suggests that, on average, the predicted value of heritability differs for sibling pairs depending on what school that they attend. The magnitude of this coefficient is quite large considering the scale of the variable and its comparison to the level-1 residual variance. Furthermore, once this variation is taken into account, the heritability estimate declines appreciably from .59 to .48. Overall, these estimates do not support the direct genetic hypothesis. That is, although a significant amount of smoking behaviors may be heritable in nature the relative contribution of heritability also depends importantly on the school-environment.

Model 5 denotes the final model and provides between school variance estimates for both heritability and shared environment. This model is particularly useful because school-specific point estimates can be calculated that describe the offset to the average effect for a particular school; schools with greater than average levels of heritability receive positive values, those with average levels of heritability receive values at or near zero, and those with reduced heritability receive negative values. To better conceptualize these school-level differences, school-specific estimates for h^2, c^2, and e^2 were calculated for all 128 schools. Figure 1 visually presents the variability in the estimates. As is evident in multilevel model estimates, the overall level of heritability is estimated at .46, but there is considerable variability in this estimate across schools. Similarly, there is variability in the c^2 estimate but this variance is considerably smaller than variance in the h^2 estimate.

[Figure 1 about here]

As described above, there are theoretical reasons to expect that school-level differences in social control will account for school-level variation in heritability. To address this, we examined two measures of social control: (1) normative control (the prevalence of smoking); and (2) institutional control (the percent minority students). Table 3 presents the results of regressing level-2 residuals on both school-level smoking and percentage of minority students. The positive and significant coefficient for smoking prevalence in conjunction with the negative and significant quadratic term suggests a curvilinear association with heritability that supports the normative control model. Likewise, the negative coefficient for percent minority suggests that heritability is reduced as the proportion of minorities in the school increases and provides support for the institutional control model.

[Table 3 about here]

[Figure 2 about here]

The two interaction terms test the competing hypotheses termed resource application and relative capacity. In general, the direction and magnitude of these coefficients provide support for the *relative capacity* explanation. To better understand these parameter estimates, Figure 2 plots a three-dimensional

graph of the fitted values from first model in Table 3. The range of smoking prevalence values is limited (10-40%) because the range of smoking prevalence is constrained within schools with a high proportion of minority students (see Appendix 1). Several findings are worth noting. First, heritability estimates are the lowest in schools with the highest rates of smoking. Moreover, the strong effect of smoking is consistent regardless of the socio-demographic composition of the school. In other words, there is no evidence for institutional advantages within schools with relatively high rates of smoking. Second institutional constraints limit heritability but only in low smoking schools. Third, the normative control model of heritability is only meaningful in schools with greater proportions of minority students. Evidence for the normative control model exists when there is a curvilinear association between smoking prevalence and heritability estimates. This functional form is only evident within those schools with at least 40% minority students and is strongly evident in schools with few non-Hispanic white students. These estimates support the relative capacity hypothesis with respect to the role of schools in shaping the heritability of smoking behaviors.

Discussion

In this study, we use a nationally representative sample of US adolescents to examine the extent to which the heritability of smoking is conditional on the school environment. There were several important findings. First, we find that roughly 50% of the variation in smoking behaviors is genetically influenced however there is significant variability in this estimate across schools. That is, while 50% appears to be an average measure of genetic contribution to smoking behaviors there are contexts in which the overall heritability is significantly limited. Second, heritability of smoking is reduced in school-environments characterized by high levels of normative control evidenced by both relatively high and relatively low rates of smoking. Third, the moderating effect of the normative environment is particularly strong in relatively disadvantaged school-settings.

These findings are important because they address concerns of regarding the most appropriate theoretical and methodological approaches for studying GE and EE interactions in health-related research (Ryff and Singer 2005). Our results suggest that researchers may need to define the contours of the social environment

more explicitly and to consider that these effects may operate both additively and multiplicatively. They also highlight an important (but rarely used) aspect of the Add Health data. That is, the in-school assessment affords researchers the opportunity to build rich school-level data sets capturing a behavioral domain of adolescent's lives that are otherwise difficult to assess. Moreover the longitudinal nature of the study makes it possible to assess long term consequences of attending schools with particular characteristics.

The focus on school-context also helps to rule out methodological issues concerning both selection and level of aggregation that often hinder GE research. For example, the possibility that students with a tendency to smoke may select into smoking networks is somewhat tempered by the finding that an adolescent's risk of smoking significantly increases with the smoking prevalence of the school. That is, adolescents may select into peer groups with relatively high rates of smoking, but it is less likely that parents select schools based on smoking prevalence. Therefore, although placement in schools with respect to smoking may not be a random process, it is less likely than peer affiliation to represent an active selection process. This helps to mitigate the possible effect of a gene-environment correlation (r_{GE}), which in this context can arise when school selection is a function of an individual's genetic predisposition to smoking, not a moderator of it. The confounding effect of r_{GE} is often problematic in epidemiologic studies of children of alcoholics, for example, who passively inherit genetic and environmental influences from their parents (Cleveland and Wiebe 2003). Determining the extent to which upbringing moderates genetic influences on drinking behavior, in the presence of significant r_{GE}, is limited in twin studies whose participants are reared together. Given the uncertain relation between school selection and smoking prevalence in the current study, it is anticipated that a gene-environment correlation would neither be significant nor confound the interaction between social environment and genetic influence on smoking behavior.

Limitations

There are also several important aspects of this paper that could be improved upon with additional research. First, the methods used in these analyses rely upon similarities among siblings to infer heritability,

but we do not identify specific genotypes that may account for these implied associations. There is evidence from a number of studies pointing to candidate genes associated with regular tobacco use (see Batra et al. 2003 for a useful review). The genes identified have been associated with the regulation of three systems involved in nicotine use and dependence: a) nicotine metabolism (Tyndale and Sellers 2001); b) the dopaminerigic system (Timberlake, Haberstick, Lessem et al. 2006; Sabol et al. 1999; Lerman et al. 1999); and c) serotonergic neurotransmission (Ishikawa et al. 1999). The Add Health study has collected biological specimens and has genotyped respondents for loci associated with nicotine metabolism (CYP2A6), dopaminergic regulation (SLC6A3, DRD2, DRD4), and serotonegic transmission (5HTT). With few exceptions (Lerman et al. 1999; Timberlake, Rhee, Haberstick, et al. 2006) the bulk of the observed associations between genotype and smoking phenotypes have not carefully considered the social environment as a potential moderator. Our results, suggest that these same associations may vary in magnitude across schools. Thus, our initial biometrical analyses support future molecular genetic studies that more fully describe the genetic aspects of smoking in relation to the social environment.

Second, our statistical models were quite limited in terms of control variables. For example, we did not include any information about local or state laws concerning tobacco sales. This is an important aspect of social control at a broad social level that we may have incorrectly associated with school-level phenomena (CDC 2000) especially if advertising for smoking is concentrated in areas containing relatively vulnerable populations (Luke et al. 2000). Nor did we include information about cigarette use in the household or smoking behaviors of peers which remain the strongest predictors of tobacco use among adolescents (Alexander et al. 2001; Vink et al. 2003; Chuang et al. 2005; Menning 2006). We also ignored potential differences between males and females. Although somewhat mixed, there is evidence suggesting that the genetic influence on smoking behaviors is different for men and women at different stages of the lifecourse (Kendler et al. 2000; Heath et al. 1999; Li et al. 2003). Finally, the multilevel analysis of only one dependent variable limited our assessment of the heritable contributions to other forms of smoking behavior (e.g.

smoking initiation). Twin studies have suggested that while the genetic determinants of smoking initiation and persistence are highly correlated, they do not perfectly overlap (Maes et al. 2004; Kendler et al. 1999).

Conclusion

Social demographers and sociologists concerned with the health-related behaviors of adolescents and young adults have made great efforts to operationalize and measure the normative environment of adolescent's schools, neighborhoods, and families; the social contexts in which youth primarily interact with one another (Berkman and Kawachi 2003). Given this interest, it is important for social scientists to be active participants in the field of behavioral genetics. Although social demographers have contributed to discussions regarding the role of genetic characteristics as potential determinants of health and well-being (Van den Oord & Rowe 2000; Guo and Stearns 2002; Cleveland and Crosnoe 2004; Cleveland 2003), the number of researchers in this engaged in these discussion is relatively small. With an increasing number of data sets including genetic information on respondents, sociologists are poised to make important contributions to this growing and important body of work. While the issues of structure, agency, and structuration (Giddens 1979) remain the guiding principles of our work these important theoretical considerations have thus far had little influence on the work of behavioral geneticists.

Likewise, the notion that genetic characteristics of individuals accounts for some of the differences among individuals, although unquestioned in other disciplines, remains hotly contested or simply ignored by social scientists (Duster 2005; Saguy and Riley 2005; Frank 2001; Zuberi 2001). A good example of this comes from a recent paper published in the Journal of Health and Social Behavior; the medical sociology journal of the American Sociological Association. In this paper, Menning (2006) provides a thorough examination of smoking behaviors among adolescents using the Add Health study. He examines the probability of previously non regular-smoking adolescents becoming regular smokers one year later as a function of non-resident father's smoking behaviors and the degree of involvement of the father controlling for sociodemographic characteristics (race, age, income, maternal education, and sex), peer smoking, cigarettes available in the home, maternal relationship. Independent of these controls, Menning (2006) shows

that the smoking behaviors of non-resident fathers --controlling for father's involvement -- predicts the smoking behavior of their children. More importantly, the effect of non-resident father smoking on their children's smoking behaviors is the same for father's who regularly interact with their children as those who have little to no contact with their children. The author tests important sociological and social psychological hypotheses focusing on control and social learning theories of adolescent development but at no point discusses the possibility that this link is simply maintained through the genetic similarity of children and their biological fathers. This is not to question the findings of the author, rather it is meant to illustrate the relatively narrow scope of inquiry among sociologists engaged in these issues. It is precisely these issues that social scientist's theories, methods, and data are particularly well-equipped to deal with.

This comment is summarized nicely by Hewitt and Turner (1995) in the introduction to *Behavior Genetic Approaches in Behavioral Medicine.* They write,

> Behavior genetics is the study of genetic *and* environmental determination of individual differences in characteristics with a behavioral component. Traditional genetics has treated environmental variation largely as "noise." Traditional epidemiology and many of the social sciences have either ignored *genetic* variation or wished it would go away. In some cases, the emphasis of these traditional disciplines on a particular component, *either* genetic *or* environmental can serve a useful scientific purpose. In other cases, it may be misleading and an obstacle to understanding. (p. 3)

In the case of smoking behaviors among adolescents, our findings clearly indicate the need to pursue not only genetic and environmental characteristics as independent factors, but also to consider the conditional nature of these important domains in understanding complex behaviors like tobacco use. Thus, this study builds on prior research that stresses the importance of social environments as critical determinants of genetic expression (i.e. Caspi et al. 2003). By doing so, this study reinforces the notion of social environments as "fundamental causes" of health and health-related behaviors (Link and Phelan 1995) by highlighting the upstream determinants of complex health outcomes. This relatively simple yet often overlooked idea brings context and social conditions into current work of behavioral geneticists and it captures an important contribution of social science researchers to these critical debates.

References

Alexander, C., Piazza, M., Mekos, D., and Valente, T. 2001. "Peers, schools, and adolescent cigarette smoking." *Journal of Adolescent Health* 29:22-30.

Anderson, Elijah. 1990. *Streetwise: Race, Class, and Change in an Urban Community*. Chicago: University of Chicago Press.

Batra, Vikas, Ashwin A. Patkar, Wade H. Berrntttini, Stephen P. Weinstein, and Frank T. Leone. 2003. "The genetic determinants of smoking." *Chest: The cardiopulmonary and critical care journal* 123:1730-9.

Berkman, Lisa F. and Kawachi, Ichiro. 2000. *Social epidemiology*. New York: Oxford University Press.

Breslau, Naomi, Eric O. Johnson, Eva Hiripi, and Ronald Kessler. 2001. "Nicotine dependence in the United States: Prevalence, trends, and smoking persistence." *Arch Gen Psychiatry* 58:810-816.

Brochard, TJ, and Propping, P., eds. 1993. *Twins as a tool of behavioral genetics*. West Sussex, England: Wiley.

Carmelli, D., Swan, G. E., Robinette, D., and Fabsitz, R. 1992. "Genetic influence on smoking: A study of male twins." *The New England Journal of Medicine* 327:829-33.

Caspi, A., Sugden, K., Moffitt, T., Taylor, A., Craig, I.W., Harrington, H.L., McClay, J., Mill, J., Martin, J., Braithwaite, A., and Poulton, R. 2003. "Influence of life stress on depression: moderation by a polymorphism in the 5-htt gene." *Science* 301:386-389.

Centers for Disease Prevention and Control [CDC] (2004). "State-Specific Prevalence of Current Cigarette Smoking Among Adults and the Proportion of Adults Who Work in a Smoke-Free Environment --- United States, 1999." MMWR. 54(44):1124-7.

Centers for Disease Prevention and Control [CDC] (2005). "Annual Smoking-Attributable Mortality, Years of Potential Life Lost, and Productivity Losses—United States, 1997-2001.: MMWR.54:625-8.

Centers for Disease Prevention and Control [CDC] (2006). "Cigarette use among high school students-united states, 1991-2005." MMWR 55:724-6.

Cherny, S.S., J.C. DeFries, and D.W. Fulker. 1992. “Multiple regression analysis of twin data: A model-fitting approach.” *Behavior Genetics* 22:489–97.

Chuang, Ying-Chih, Susan T. Ennet, Karl E. Bauman, and Vangie A. Foshee. 2005. “Neighborhood influences on adolescent cigarette and alcohol use: Mediating effects through parent and peer behaviors.” *Journal of Health and Social Behavior* 46:187-204.

Cleveland HH, Wiebe RP. 2002. The moderation of genetic and shared-environmental influences on adolescent drinking by levels of parental drinking. J Stud Alcohol. 64(2):182-94.

Cleveland, H. H. 2003. “Disadvantaged Neighborhoods and Adolescent Aggression: Behavioral Genetic Evidence of Contextual Effects.” *Journal of Research on Adolescence* 13:211-38.

Cleveland, H. H. and Robert Crosnoe. 2004. “Individual Variation and Family-Community Ties: A Behavioral Genetic Analysis of the Intergenerational Closure in the Lives of Adolescents.” *Journal of Adolescent Research* 19:174-91.

Deater-Deckard, Kirby and Ulrich Mayr. 2005. “Cognitive change in aging: Identifying Gene-environment correlation and nonshared environment mechanisms.” *Journals of Gerontology: SERIES B* 60B(Special Issue I):24-31.

DeFries, J. C. and David W. Fulker. 1985. “Multiple Regression Analysis of Twin Data.” *Behavior Genetics* 15:467-73.

Dunne, M. P., Martin, N.G., Statham, D. J., Slutske, W. S., Dinwiddie, S. H., et al. 1997. Genetic and environmental contributions to variance in age at first sexual intercourse. *Psychological Science* 8:211-6.

Duster, Troy. 2006. “Comparative perspectives and competing explanations: Taking on the newly configured reductionist challenge to Sociology.” *American Sociological Review* 71(1):1-15.

Eaves, Lindon J. 2006. “Genotype x environment interaction in psychopathology: Fact or artifact?” *Twin research and human genetics* 9:1-8.

Eitle, David J. and Tamela M. Eitle. 2004. “School and county characteristics as predictors of school rates of drug, alcohol, and tobacco offenses.” *Journal of Health and Social Behavior* 45:408-21.

Ellickson, Phyllis L., Chloe E. Bird, Maria Orlando, David J. Klein and Daniel F. McCaffrey. 2003. “Social context and adolescent health behavior: Does school-level smoking prevalence affect students' subsequent smoking behavior*?” Journal of Health and Social Behavior* 44:525-35.

Ennet, Susan T., Robert L. Flewelling, Richard C. Lindrooth, and Edward C. Norton. 1997. “School and neighborhood characteristics associated with school rates of alcohol, cigarette, and marijuana use.” *Journal of Health and Social Behavior* 38:55-71.

Frank, R. 2001. The misuse of biology in demographic research on Racial/Ethnic differences: A reply to van den Oord and Rowe. *Demography* 38(4):563-7.

Giddens, A. 1979. *Central problems in social theory: action, structure, and contradiction in social analysis*. Berkeley: University of California Press.

Guo, Guang and Jianmin Wang. 2002. “The mixed or multilevel model for behavior genetic analysis.” *Behavior Genetics* 32(1):37-49.

Guo, Guang., and Stearns, E. 2002. “The social influences on the realization of genetic potential for intellectual development.” *Social Forces,* 80:881-910.

Heath, A.C., K.M. Kirk, J.M. Meyer, and N.G. Martin. 1999. “Genetic and social determinants of initiation and age at onset of smoking in Australian twins.” *Behavioral Genetics* 29(6):395-407.

Heath, Andrew C., and Pamela A.F. Madden. 1995. “Genetic influences on smoking behavior.” Pp. 45-66 in J.R. Turner and L.R. Cardon, and J.K. Hewitt (Eds.) *Behavior genetic approaches in behavioral medicine.* New York: Plenum.

Hu, Mei-Chen, Mark Davies, and Denise Kandel. 2006. “Epidemiology and correlates of daily smoking and nicotine dependence among young adults in the United States.” *American Journal of Public Health* 96(2):299-308.

Hewitt, John K., and J. Rick Turner. 1995. “Behavior genetic approaches in behavioral medicine: an introduction.” Pp. 3-13 in J.R. Turner and L.R. Cardon, and J.K. Hewitt (Eds.) *Behavior genetic approaches in behavioral medicine.* New York: Plenum.

Ishikawa, H, Ohtsuki T, Ishiguor, H, et al. 1999. “Association between serotonin transporter gene and polymorphism and smoking among Japanese males.” *Cancer Epidemiol Biomarkers Prev* 8:831-833.

Johnston, L. D., O'Malley, P. M., Bachman, J. G. and Schulenberg, J. E. 2005. “Decline in teen smoking appears to be nearing its end.” University of Michigan News and Information Services: Ann Arbor, MI. [On-line]. Available: www.monitoringthefuture.org; accessed 7/10/06.

Johnston, T. D., Edwards, L. 2002. “Genes, interactions, and the development of behavior.” *Psychological Review* 109:26-34

Kendler, Kenneth S., Laura M. Thornton, and Nancy L. Pederson. 2000. “Tobacco consumption in Swedish twins reared apart and reared together.” *Arch Gen. Psychiatry* 57:886-92.

Kendler KS, Neale MC, Sullivan P, Corey LA, Gardner CO, Prescott CA. 1999. “A population-based twin study in women of smoking initiation and nicotine dependence. *Psychol Med* 29(2):299-308.

Koopmans, JR, WS Slutske, AC Heath, MC Neale, and DI Boomsma. 1999. “The genetics of smoking intuition and quantity smoked in Dutch adolescent and young adult twins.” *Behavior Genetics* 29:383-93.

Lerman, C, NE Caporaso, J Audrain, et al. 1999. “Evidence suggesting the role of specific genetic factors in cigarette smoking.” *Health Psychology* 18:14-20.

Li, M., Cheng, R., Ma, J., and Swan, G. 2003. “A meta-analysis of estimated genetic and environmental effects on smoking behavior in male and female adult twins.” *Addiction* 98:23-31.

Link, B.G. and Phelan, J. 1995. “Social conditions as fundamental causes of disease.” *Journal of Health and Social Behavior,* extra issue:80-94.

Luke, Douglas, Emily Esmundo, and Yael Bloom. 2000. “Smoke signs: patterns of tobacco billboard advertising in a metropolitan region.” *Tobacco Control* 9:16-23.

Maes HH, Sullivan PF, Bulik CM, Neale MC, Prescott CA, Eaves LJ, Kendler KS. 2004. “A twin study of genetic and environmental influences on tobacco initiation, regular tobacco use and nicotine dependence.” *Psychol Med*. 34(7):1251-61.

Menning, Chadwick L. 2006. “Nonresident father’s involvement and adolescents' smoking.” *Journal of Health and Social Behavior* 47(1):32-46.

Purcell S. 2002. “Variance components models for gene-environment interaction in twin analysis.” *Twin Research* 5(6):554-71.

Purcell, Shaun 2003. "A model-fitting implementation of the DeFries-Fulker model for selected twin data." *Behavior Genetics* 33(3):271-8.

Purcell S. and P.C. Sham. 2002. “Variance components models for gene-environment interaction in quantitative trait locus linkage analysis.” *Twin Research* 5(6):572-6.

Raudenbush, Stephen W. and Anthony S. Bryk. 2002. *Hierarchical Linear Models: Applications and Data Analysis Methods* (2nd Edition). Thousand Oaks: Sage Publications.

Rende, R. 1993. “Genes, environment, and addictive behavior: Etiology of individual differences and extreme cases.” *Addiction* 88:1183-8.

Rodgers, J. L., and McGue, M. 1994. “A simple algebraic demonstration of the validity of DeFries-Fulker analysis in unselected samples with multiple kinship levels.” *Behavior Genetics* 24:259-62.

Rogers, Richard G., Robert A. Hummer, Patrick M. Krueger, and Fred C. Pampel. 2005. “Mortality Attributable to Cigarette Smoking in the United States.” *Population and Development Review* 31(2): 259-92.

Rowe, DC, KC Jacobson, and E van den Oord. 1999. “Genetic and environmental influences on vocabulary IQ: Parental education level as moderator.” *Child Development* 70:1151-62.

Ryff, C. D. and B. H. Singer. 2005. “Social Environments and the Genetics of Aging: Advancing Knowledge of Protective Health Mechanisms.” *The Journals of Gerontology. Series B, Psychological Sciences and Social Sciences* 60 Spec No 1:12-23.

Sabol, S.Z., Nelson, M.L., Fisher, C., Gunzearth, L., Brody, C.L., Hu, S., et al. 1999. A genetic association for cigarette smoking behavior. *Health Psychology* 18:7-13.

Saguy, Abigail C. and Kevin W. Riley. 2005. “Weighing Both Sides: Morality, Mortality and Framing Contests over Obesity.” *Journal of Health Politics, Policy, and Law*. 30:869-921.

Shanahan, Michael J. and Scott M. Hofer. 2005. “Social context in gene-environment interactions: retrospect and prospect.” *Journals of Gerontology: SERIES B* 60B(Special Issue):65-76.

Slombowski, Cheryl, Richard Rende, Scott Novak, Elizabeth Lloyd-Richardson, and Raymond Niaru. 2005. “Sibling effects on smoking in adolescence: Evidence for social influence from genetically informative design.” *Addiction* 100:430-8.

Sullivan, P. F., and Kendler, K. S. 1999. “The genetic epidemiology of smoking.” *Nicotine and Tobacco Research*, 1(Suppl. 2), S51–S57.

Timberlake, David S., Brett C. Haberstick, Jeffrey M. Lessem, Andrew Smolen, Marissa Ehringer, and John K. Hewitt. 2006. “An association between the DAT1 polymorphism and smoking behavior in young adults from the National Longitudinal Study of Adolescent Health.” *Health Psychology* 25(2):190-7.

Timberlake, David S., Rhee, Soo Hyun, Haberstick, Brett C., Hopfer, Christian, Ehringer, Marissa, Lessem, Jeffrey M., Smolen, Andrew, and Hewitt, John K. 2006. “The moderating effects of religiosity on the genetic and environmental determinants of smoking initiation.” *Nicotine and Tobacco Research* 8:123–133.

Todd, GF and Mason JI. 1959. “Concordance of smoking habits in monozygotic and dizygotic twins.” *Heredity* 13:417-44.

Turkheimer, Eric, Andreanna Haley, Mary Waldron, Brian D’Onofrio, and Irving I. Gottesman. 2003. “Socioeconomic status modifies heritability of IQ in young children.” *Psychological Science* 14(6):623-628.

Tyndale, RF, and Sellers, EM. 2002. “Genetic variation in CYP2A6-mediated nicotine metabolism alters smoking behavior.” *Therapeutic Drug Monitoring* 24(1):163-71.

US Department of Health and Human Services. 2000. Healthy people 2010: understanding and improving health. 2nd ed. Washington, DC: US Department of Health and Human Services; 2000.

US Department of Health and Human Services. 1989. Reducing the health consequences of smoking: 25 years of progress. A report to the surgeon general. USDHHS, CDC, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health.

van den Oord,Edwin J.C.G., and Rowe, D. C. 2000. “Racial differences in birth health risk: A quantitative genetic approach.” *Demography* 37(3):285-298.

Vink, J., Willemsen, G., Engels, R., and Boomsma, D. 2003. “Smoking status of parents, siblings and friends: predictors of regular smoking? Findings from a longitudinal twin-family study.” *Twin Research* 6:209-217.

Zuberi, T. 2001. “One step back in understanding racial differences in birth weight.” *Demography* 38(4):569-571.

Table 1. Tetrachoric and Pearson Correlation coefficients for smoking behaviors among adolescent sibling pairs: National Longitudinal Study of Adolescent Health (Wave II).

Pair type	Ever smoked	Regular Smoker	Total Smoking	N
Identical twins	0.63	0.84	.63	245
Fraternal twins	0.45	0.69	.38	355
Full siblings	0.41	0.56	.26	900
Half siblings	0.31	0.48	.30	253

Note: estimates obtained from wave II of the Add-Health study using the sibling-pair sample (n = 1753 sibling pairs).

Table 2. The Moderating Role of the Social Context in the Heritability of Smoking Behaviors: Multilevel DeFries-Fulker Regression Estimates

	Model 1	Model 2	Model 3	Model 4	Model 5
Intercept	-.05	-.03	-.03	-.03	-.03
Main effects					
Sibling smoking (X)		.27***	.25***	.25***	.23***
Genetic similarity (G)		-.01	-.01	-.02	-.03
Interaction effect					
XG		.59***	.48***	.55***	.46***
Residual Variance					
Level 1	.93**	.87**	.85**	.83**	.82**
Level 2					
Intercept	.05*	.02*	.02*	.02*	.02*
Slopes					
XG			.85***		.50**
X				.04*	.04*
Log likelihood	-4623.69	-4316.93	-4304.25	-4277.83	-4272.58
-2Log Likelihood	9247.38	8633.86	8608.50	8555.66	8545.16
Likelihood ratio		613.52***	25.36***	78.20***	88.70***

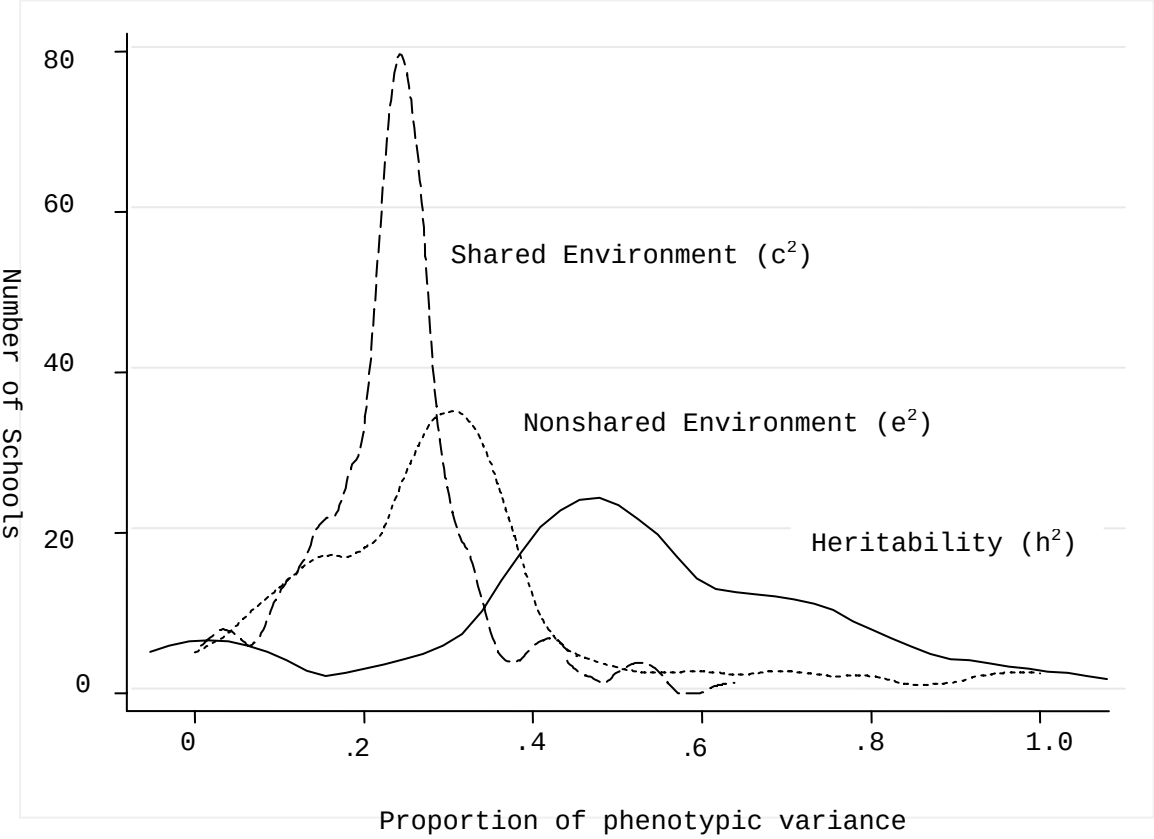
Note: Data on sibling smoking obtained from Wave II of Add Health. *** p<.001; ** p<.01; * p<.05

Table 3. Describing variation in school-level heritability estimates: level-2 residuals (Empirical Bayes Estimates) regressed on school-level smoking and minority representation (n = 128).

	Heritability (h^2)	Shared Environment(c^2)
Intercept	.12 (.09, .14)	.00 (-.01, .01)
Main Effects		
Smoking Prevalence (S)	.62 (.45, .80)	-.78 (-.84, -.71)
Smoking Prevalence^2 (S^2)	-2.33 (-2.60, -2.05)	1.52 (1.42, 1.62)
Percent Minority (M)	-.51 (-.58, -.43)	.15 (.12, .18)
Interaction Effects		
SM	2.46 (1.90, 3.02)	-.38 (-.58, -.19)
S^2M	-2.92 (-3.89, -1.95)	.73 (.39, 1.07)
R-squared	.08	.13

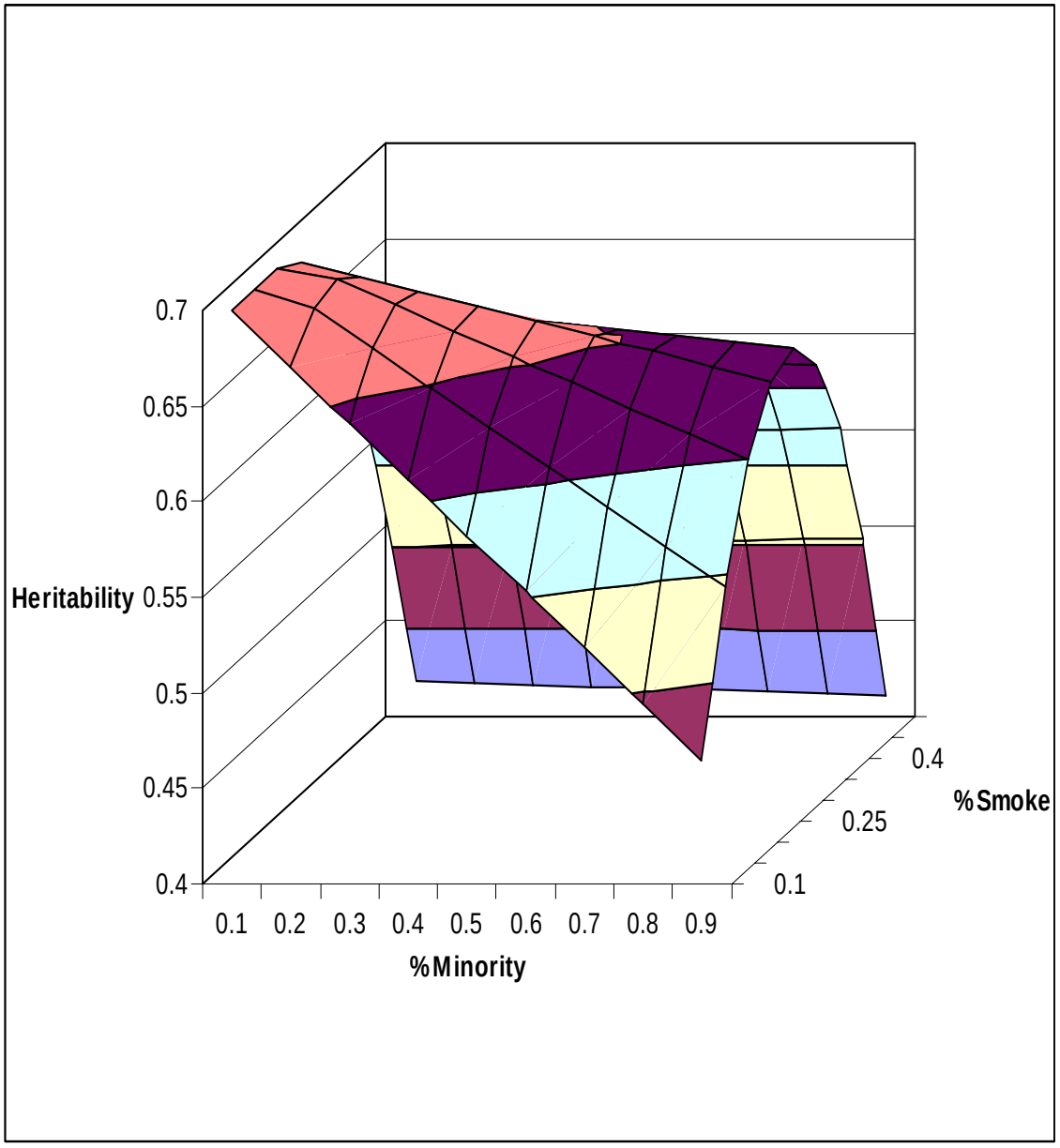
Note: data weighted using the SCHADMWT from the in-school study; cell entries represented OLS parameter estimates and 95% confidence intervals.

Figure 1. School-level residual variation in quantitative genetic parameter estimates for smoking behaviors among adolescents.



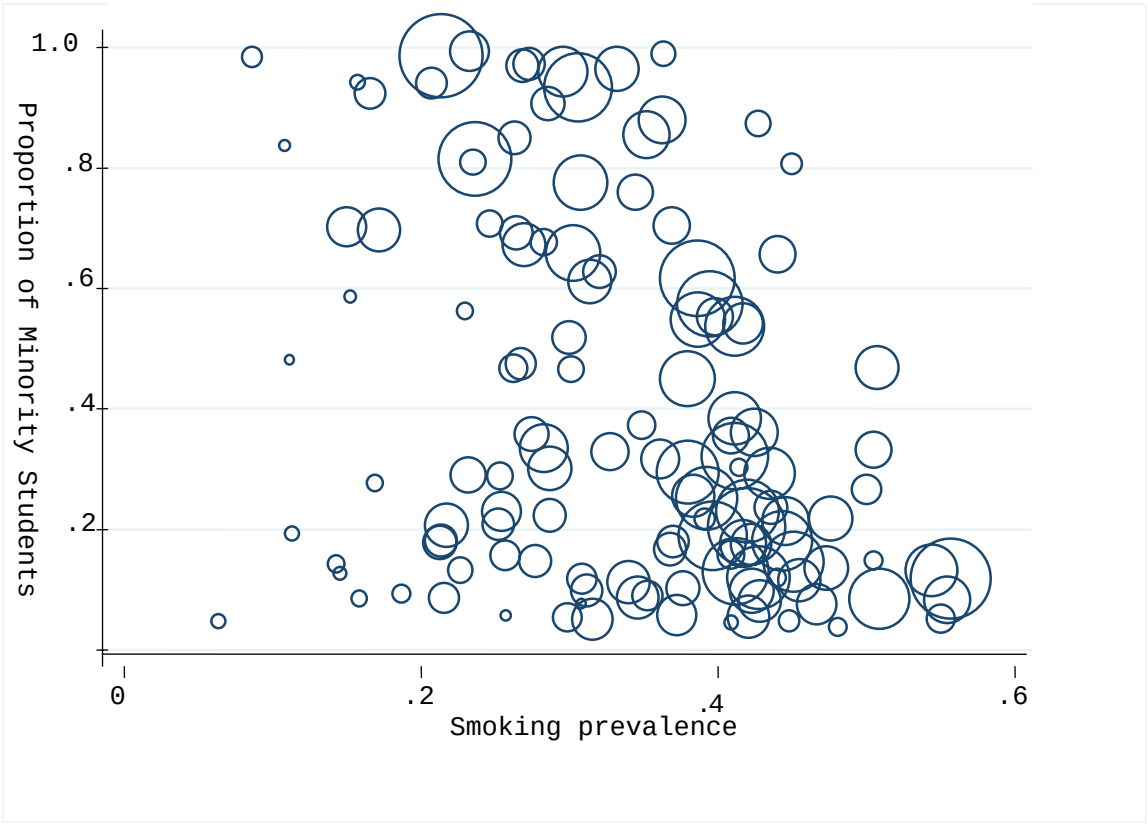
Note: estimates derived from post-hoc empirical Bayes estimates from a multilevel model presented in Model 5 of Table 2 (n = 128 schools).

Figure 2. Variation in smoking behaviors due to the heritability: interactive environmental effects of social control.



Note: Estimates derived from the first column of results from Table 3.

Appendix 1. Distribution of schools by smoking behavior and minority population: 1995 in-school sample of the National Longitudinal Study of Adolescent Health ($n_{schools}$ = 128).



Note: Number of respondents per school is captured by the size of the circles.