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Limited common origins of multiple adult health-related behaviors: Evidence from U.S. twins☆

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ABSTRACT

Health-related behaviors are significant contributors to morbidity and mortality in the United States, yet evidence on the underlying causes of the vast within-population variation in behaviors is mixed. While many potential causes of health-related behaviors have been identified—such as schooling, genetics, and environments—little is known on how much of the variation across multiple behaviors is due to a common set of causes. We use three separate datasets on U.S. twins to investigate the degree to which multiple health-related behaviors correlate and can be explained by a common set of factors. We find that aside from smoking and drinking, most behaviors are not strongly correlated among individuals. Based on the results of both within-identical-twins regressions and multivariate behavioral genetics models, we find some evidence that schooling may be related to smoking but not to the covariation between multiple behaviors. Similarly, we find that a large fraction of the variance in each of the behaviors is consistent with genetic factors; however, we do not find strong evidence that a single common set of genes explains variation in multiple behaviors. We find, however, that a large portion of the correlation between smoking and heavy drinking is consistent with common, mostly childhood, environments. This suggests that the initiation and patterns of these two behaviors might arise from a common childhood origin. Research and policy to identify and modify this source may provide a strong way to reduce the population health burden of smoking and heavy drinking.

1. Introduction

Health-related behaviors, such as smoking and heavy drinking, are responsible for a large portion of global morbidity and mortality. For example, smoking, heavy drinking, and obesity were associated with 38% of United States mortality in 1993 and almost 50% in 2000 (McGinnis and Foege, 1993; Mokdad et al., 2004). Health-related behaviors have also been implicated as reasons for international differences in life expectancy: smoking and obesity may explain why the United States has lower life expectancy compared to other Western countries and why life expectancy in the former Soviet Union countries has stagnated relative to other European countries (Preston et al., 2011; Rehm et al., 2007).

An important question for understanding trends and variation in health outcomes is whether multiple health-related behaviors are determined by a common cause or if behaviors each have unique underlying determinants. In many studies, socioeconomic status, usually measured as either schooling or household income, is posited as a cause of health-related behaviors. On first glance, the evidence is compelling: higher levels of schooling are overwhelmingly associated with healthier behaviors across many domains and may potentially explain why more-schooled people tend to be in better health (Cawley and Ruhm, 2011). Despite these associations, a more recent literature using data on identical twins has tried to determine if these associations are causal, or if schooling is determined by unobserved characteristics that also determine health-related behaviors. The findings from these

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studies suggest that while schooling is associated with better health-related behaviors, schooling may not be a cause of these behaviors (Amin et al., 2015; Behrman et al., 2011; Behrman et al., 2015).

Genetics are also commonly cited as causes for health-related behaviors. Studies have found that a substantial part of the variation in smoking, physical exercise, and body mass index (BMI) can be attributed to genetic differences within populations (Bauman et al., 2012; Kaprio et al., 1982; Vink et al., 2005; Walters, 2002). Also, many aspects of the childhood environment have been associated with physical activity patterns (Bauman et al., 2012), smoking behavior (Gilman et al., 2003), and obesity across a wide range of adult ages (Parsons et al., 1999). While these studies have provided substantial evidence to suggest that genetics and childhood environments play an important role in the development of health-related behaviors in adulthood, the relationship between a common set of genetic endowments, childhood environments, and variation across multiple behaviors remains unclear.

In this paper we use data on U.S. twins to investigate the degree to which multiple health-related behaviors can be explained by a single set of characteristics. Our paper combines approaches from economics and behavioral genetics to determine the contribution of schooling, genetic endowments, and environments to unhealthy behaviors – or the outcomes of such behaviors such as BMI – among U.S. adults. As the health and mortality profile of high- and increasingly also low-to middle-income countries shifts further towards chronic, behavior-related, conditions, understanding the origins of health-related behaviors can help to formulate effective policies and interventions to improve population health.

1.1. Background

Given the substantial associations between health-related behaviors, morbidity, and mortality, a large literature has focused on why people engage in behaviors that are widely known to negatively affect health. Underlying much of this literature is the belief that specific factors, such as genetics, personality, or schooling, are common underlying determinants of a broad range of individual health-related behaviors. In the following sections, we briefly review evidence from health, economics, and behavioral genetic studies on the causes of health-related behaviors.

Economic studies of the underlying behavioral causes of health are heavily influenced by Grossman's model of health capital. In this model, more-educated people are more likely to make better choices regarding health inputs, including health-related behaviors, given available resources (allocative efficiency), and are better at producing health from a given set of inputs (productive efficiency) (Grossman, 1972). Similar theories suggest that more educated people may also have more available resources to invest in health (Link and Phelan, 1995). Descriptive studies of health behaviors are very consistent with these theories, since higher levels of schooling are strongly associated with healthier behaviors across many domains. For example, college graduates are less likely to smoke, less likely to be obese, less likely to drink heavily, and less likely to be physically inactive compared to high school dropouts. They are also more likely to receive mammograms, colorectal screenings, and use sunscreen (Cawley and Ruhm, 2011). Cutler and Lleras-Muney attempt to unpack these strong associations by examining the potential mechanisms behind the large education gradient in health-related behaviors. They find that around 30% of the educational gradient in health-related behaviors is explained by income, health insurance, and family background, and around 30% from knowledge and cognitive ability (Cutler and Lleras-Muney, 2010). While this study made a substantial contribution towards understanding the sources of educational differences in health-related behaviors, the study design was limited by an inability to identify whether the education health relationship is causal. In a recent paper, Heckman, Humphries, and Veramendi use a dynamic structural model of educational choice and find evidence that education may have a causal effect on health (Heckman et al., 2016). An emerging literature using data on identical twins has also tried to determine if these associations are causal, or if schooling is determined by unobserved characteristics that also determine health-related behaviors. These studies essentially assume that identical twins share the unobserved characteristics (such as parental background, genetic dispositions, the shared mostly childhood environment) that simultaneously influence schooling and health outcomes and bias estimates of the education health relationship in conventional analyses (Kohler et al., 2011). By using within-MZ-twins estimates, the cross-sectional associations between schooling and health are purged of bias from these unobserved factors. The findings from these studies suggest that while schooling is associated with better health-related behaviors, schooling may not be a cause of health-related behaviors (Amin et al., 2015; Behrman et al., 2011, 2015). Similarly, Cutler and Glaeser try to confirm empirically Grossman's model by arguing that if health-related behaviors are determined by individual investments in future health, different health-related behaviors should be correlated within individuals. Using data from the Behavioral Risk Factor Surveillance System, they find weak correlations between the health-related behaviors of individuals—such as obesity and smoking, and smoking and receiving mammograms for women—implying that the factors that determine health-related behaviors vary across behavioral domains (e.g. the factors that lead individuals to smoke do not necessarily lead individuals to be physically inactive) (Cutler and Glaeser, 2005).

Variation in health-related behaviors has also been examined from a behavioral genetics perspective. Under this paradigm, health-related behaviors are additively determined by genetic endowments, common (shared by sibling) environments, and individual idiosyncratic environments. Many behavioral genetic studies of health find that a large fraction of the within-population variance in health-related behaviors is consistent with variation in genetic factors. For example, a study using Dutch twins pairs reports that smoking initiation has a heritability of 44%–implying that, subject to the assumptions of the behavioral genetics model, 44% of the variation in smoking initiation is associated with genetic differences within the population (Vink et al., 2005). This same study finds that 51% of the variation in the initiation of smoking is associated with the shared, mostly childhood, environment between twins. This approach has been applied to a range of behaviors: in a meta-analysis of the heritability of alcohol abuse and dependence, Walters reports that around 12% of the variation in alcohol abuse is associated with genetic variation in the population (Walters, 2002). Genetics are also thought to play an important role in unhealthy weight–a literature review of many behavioral genetic studies finds that genetic factors are associated with between 50% and 90% of the variation in BMI (Min et al., 2013). These studies thus suggest that genetic and childhood environmental heterogeneity is an important correlate of health-related behaviors. Importantly, the size of the association between genetic factors and health-related behaviors may also interact with other behaviors. For example, Mustelin et al. find that higher levels of physical activity reduce the association between genetic factors and BMI (Mustelin et al., 2009). Boardman et al., find that the composition of the smoker population in the United States became increasingly genetically “vulnerable” to smoking as the overall population of smokers decreased (Boardman et al., 2011). The results from these studies suggest that genetics may become more correlated with health-related behaviors as the populations of individuals that engage in those

behaviors becomes more select.

Many studies in behavioral genetics have also used data on twins to explore the covariation between multiple health-related behaviors (Eisen et al., 1993; Han et al., 1999; Kaprio et al., 1982; Liao et al., 2016; True et al., 1999). For example, Eisen et al. examine the relationships between smoking and weight and alcohol and weight, by comparing the within-twins differences in smoking and drinking to within-twins differences in weight. They find that current smokers tend to weigh less compared to former and never smokers but find no relationship between alcohol consumption and weight (Eisen et al., 1993). Other twins studies have also found similar results (Liao et al., 2016). The behavioral genetics literature on the covariation between tobacco and alcohol use is less consistent, with some studies finding a large genetic correlation between the two behaviors (True et al., 1999), while other studies find negligible genetic correlation (Kaprio et al., 1982), and others significant shared environmental correlations (Han et al., 1999). The variation in the results of these studies suggests that greater investigation is needed into the covariation between health-related behaviors, especially the genetic and environmental contributions to multiple behaviors.

A more recent field in genetic research uses data from the DNA of individuals with and without a certain phenotype, such as high blood pressure, to try and identify genetic variants that are correlated with phenotypes. These genome wide association studies (GWAS) can also estimate how much of the observed heritability of traits is explained by common sets of genes. Although this field is still growing, genetic variants responsible for a significant fraction of the variance of many health-related behaviors have already been identified. For example, identified genetic variants explain 18.6% of the variation in BMI, 5.6% of the variation in cigarettes smoked per day, and 15.1% of the variation total cholesterol (Zheng et al., 2016). Based on these variances, GWAS also allows for estimates of genetic correlation between traits. The results from these analyses suggest the presence of genetic correlation between some health-related behaviors and outcomes, such as BMI and cigarettes smoked per day ($r = 0.287$) (Bulik-Sullivan et al., 2015).

Finally, a mostly descriptive literature in the health sciences has found that many aspects of the childhood environment are correlated with health-related behaviors in adulthood. A common correlate of many health-related behaviors is childhood socioeconomic status, usually measured through parental education. For example, Gilman et al. find that higher childhood socioeconomic status is negatively correlated with the risk of becoming a regular smoker and the likelihood of smoking cessation (Gilman et al., 2003). In a review of studies, Parsons et al. report similar correlates of adult obesity, identifying higher parental weight, lower childhood SES, and certain household structures as common predictors of obesity in adulthood (Parsons et al., 1999). These correlations may be the result of many mechanisms. Some studies suggest that behaviors established in childhood are more likely to persist into adulthood. For example, a cohort study of individuals from Finland finds that being physically active in childhood is a strong predictor of physical activity in adulthood (Telama et al., 2005). The effects of childhood SES on adult behaviors may also operate through parental knowledge and resources, although some studies find a persistent relationship between childhood and adulthood behaviors even after adjusting for parental income or SES (Poulton et al., 2002). One prominent potential mechanism is known as the “fetal origins” hypothesis and posits that children exposed to poor in utero environments are more likely to have high blood pressure, obesity, and develop a range of cardiovascular diseases as adults (Barker, 1990, 1995). Therefore, poor childhood SES may impact adult health outcomes by negatively affecting fetal health through pathways such as poor neonatal nutrition.

Research in multiple disciplines has identified many potential causes of health-related behaviors in adulthood. While studies have shown relationships between schooling, genetics, environments, and various health-related behaviors, the extent to which these factors determine multiple behaviors remains an open question. We use three datasets on U.S. twins to provide new evidence on the degree to which multiple health-related behaviors can be explained by an underlying common set of determinants. Our focus is limited to smoking, drinking, unhealthy weight, and physical activity, since these health-related behaviors are associated with the greatest burden of adult morbidity and mortality (McGinnis and Foege, 1993; Mokdad et al., 2004). We find that aside from smoking and drinking, most behaviors are not strongly correlated among individuals. However, smoking and drinking are among the two largest behavioral risk factors for poor health, so a correlation between these two important health-related behaviors may have large implications for population health. While we find some evidence that schooling may be related to smoking, schooling is not a strong candidate explanation for the covariation between multiple behaviors. Similarly, we find that a large fraction of the variance in each of the behaviors is consistent with genetic factors; however, we do not find strong evidence that a single common set of genes explains variation in multiple behaviors. We find, however, that a large portion of the correlation between smoking and heavy drinking is consistent with common, likely mostly in childhood, environments–suggesting that the initiation and patterns of these two behaviors might arise from a common childhood origin.

1.2. Data

Our analyses use three separate sources of data on American twins: the National Longitudinal Study of Adolescent to Adult Health (Add Health), the National Survey of Midlife Development in the United States (MIDUS), and the Socioeconomic Survey of Twins of the Minnesota Twin Registry (MTR).

1.3. Description of the data sources

Add Health is a nationally representative longitudinal survey that first surveyed children in grades 7 through 12 in 1994 and 1995, with follow-up surveys in 1996, 2001, and 2008. Beginning in the first wave, the Add Health followed a sibling subsample that included both identical (MZ) and fraternal (DZ) twins. Since the focus of this paper is on adults, we use data on the twin sample from the fourth wave of data collection, when the individuals in the cohort were between the ages of 25 and 32.

MIDUS is a longitudinal survey of the non-institutionalized population of the United States between the ages of 25 and 74. The first wave of data collection was in 1995 with a follow-up survey between 2006 and 2009. For this paper, we focus specifically on the twin subsample, pooling data from both survey years.

Finally, we use data from the Socioeconomic Survey of Twins of the Minnesota Twin Registry (MTR). The MTR is a registry of all twins born between 1936 and 1955 in Minnesota. Our data are from the Socioeconomic Survey of Twins, a mail-based survey of same-sex MZ and DZ twins conducted in 1994.

Different procedures were used to identify zygosity across the three datasets. Zygosity in the Add Health data was initially self-reported by the twins but was later confirmed by DNA testing. In the MIDUS data, twins were given a separate survey and asked to self-report their zygosity as either monozygotic or dizygotic. Finally, the zygosity of individuals in the MTR sample was based on analysis of blood enzymes, serum proteins, fingerprint ridgecount, and other biological comparisons. For all three surveys we only consider MZ and same-sex DZ twins, since opposite-sex DZ twins

reduce the tenability of the "shared environments" assumption of behavioral genetics models (many behavioral genetic studies also drop opposite sex pairs (Han et al., 1999; Kaprio et al., 1982)).

1.4. Schooling

While socioeconomic status is reflected over multiple measures, such as income, occupation, and schooling, we limit our focus to schooling for the following reasons. First, measures such as income have been shown to fluctuate over the life course. Income and occupation may also be inversely related with health, where individuals with poor adult health and health-related behaviors earn less money and are less likely to be employed (Stronks et al., 1997). For both these reasons, income and occupation may not be stable measures of socioeconomic status. In contrast, schooling is preferred as a measure of socioeconomic status in many studies since it is established relatively early in life, and for most people, remains unchanged over the life course (Elo, 2009).

For all three datasets individuals categorically reported their highest level of completed schooling. Based on these responses, we created a continuous measure of grades of schooling by assigning grades of schooling to each of the completed categories. The categories were assigned as follows.

Add Health: Eighth grade or less (8 grades), some high school (10 grades), high school graduate (12 grades), some vocational/technical training (12.5 grades), completed vocational/technical training (13 grades), some college (14 grades), completed college (16 grades), some graduate school (17 grades), completed master's degree (18 grades), some graduate training beyond a master's degree (20 grades), completed a doctoral degree (22 grades), some post baccalaureate professional education (18 grades), completed post baccalaureate professional education (20 grades).

MIDUS: No school/some grade school (3 grades), eighth grade/junior high school (7 grades), some high school (10 grades), GED (10 grades), graduated from high school (12 grades), 1–2 years of college (13 grades), graduated from a 2-year college (14 grades), 3 or more years of college (15 grades), graduated from a 4- or 5-year college (16 grades), some graduate school (17 grades), master's degree (18 grades), doctoral degree (21 grades).

MTR: No schooling or completed grades up through secondary school graduation (actual grades as reported), GED (11 grades), vocational degree (13 grades), associate degree or some college (14 grades), bachelor degree (16 grades), masters degree (18 grades), doctoral degree (21 grades).

1.5. Health-related behaviors

We created two binary variables for smoking and drinking to capture both initiation and quantity consumed. For smoking, we created a variable for ever smoker if an individual reported ever regularly smoking and variable for heavy smoker if an individual reported currently smoking a pack per day or more. Similarly, we created a variable for ever drinker if an individual ever reported consuming alcohol and a variable for heavy drinker if an individual reported currently drinking four or more drinks per sitting on average (unfortunately, the MTR did not ask about drinks per day, rather they asked the number of days an individual drank per week so for heavy drinking is defined in terms of drinking more on more than four days per week). We preferred drinks per day rather than the number of days an individual drank, since this measure may better capture harmful binge drinking patterns (Viner and Taylor, 2007).

Measurements of physical activity varied slightly across datasets. For Add Health, we measured physical activity by the number of times per week an individual reported engaging in vigorous physical activity. This was constructed based on a series of questions on different types of physical activity: we first categorized these questions as light, moderate, and vigorous activity based on their MET score (Ainsworth et al., 2011), then translated the number of times an individual performed each type of activity into the total number of times they engaged in vigorous activity. In the MIDUS, we used a continuous variable of the average number of days per month that an individual reported engaging in vigorous activity (this variable was top coded at 14 days in the MIDUS data). Finally, we do not have measurements of physical activity in the MTR since individuals were not asked about their activity patterns. Due to the difficulty in measuring diet, we proxied the combined effects of diet and physical activity as unhealthy weight–measured by BMI for all three datasets.

1.6. Validity and reliability of the outcome measures

Although we were not able to directly assess the reliability or validity of our outcomes, we use standard measurements with extensively documented reliability and validity. Based on a meta-analysis of the validity of self-reported smoking, Patrick et al. find that across studies, self-reported smoking tracks closely with biomarker measures of tobacco use (Patrick et al., 1994). Self-reported smoking has also been shown to be reliable, with a greater reliability for ever-smoking (kappa = 0.82) compared to categories such as light or heavy smoker (kappa = 0.6) (Brigham et al., 2008; Kenkel et al., 2003). Retrospective quantity smoked has also been found to agree with cigarette sales (Hatziandreu et al., 1989). Retrospective alcohol information has shown moderate to high reliability: one study estimates a kappa between 0.26 and 0.54 while another finds that retrospective alcohol accounts for 86% of the variability in current alcohol consumption (Czarnecki et al., 1990; Harris et al., 1994). Although the validity of self-reported alcohol is harder to assess, a large meta-analysis concludes that self-reported alcohol is a generally valid measure (Midanik, 1988). For self-reported physical activity, studies of the test-retest reliability find that reliability and validity is generally high, but more so for vigorous than moderate activity (Sallis and Saelens, 2000). For example, a study of Latinos finds a correlation of $r > 0.4$ between self-reported vigorous activity and measured activity (Rauh et al., 1992). Finally, BMI was directly measured for two of the three datasets; in the MTR data, BMI was calculated based on self-reported height and weight. For this dataset, BMI might be underestimated due to height underreporting for men and weight underreporting for women (Merrill and Richardson, 2009). There is a general question on whether BMI is a valid measure of body fat; studies find that the validity of BMI as a measure of fat is moderate in the middle ranges and high at higher levels of BMI (Deurenberg et al., 1991; Romero-Corral et al., 2008). Overall, our measures are generally regarded as valid and reliable but it is still important to note potential errors introduced by self-reports, especially for physical activity and alcohol behavior (for the within-MZ twins models, reporting error would only bias the estimates if one twin misreports differently than the other).

1.7. Missing values and sample size

For Add Health, the total wave 4 twin sample consisted of 396 complete MZ or same-sex DZ twin pairs. 22 twin pairs (5.6%) were dropped for missing information for one or both members of the twinship for a final sample of 373 twin pairs (206 MZ twin pairs and 167 DZ twin pairs). The total MIDUS twin sample for waves 1 and 2 pooled consisted of 1085 complete twin pairs. 332 twin pairs (30.6%) were dropped for missing information on the key covariates for one or both members of the twinship for a final sample size of

753 twin pairs (416 MZ twin pairs and 337 same-sex DZ twin pairs). Finally, the MTR had an initial twin sample of 1399 complete twin pairs. 246 twin pairs (17.6%) were dropped for missing information on the key covariates for a final sample of 1153 twin pairs (647 MZ twin pairs and 506 same-sex DZ twin pairs).

2. Methods

If health-related behaviors are determined by a common set of determinants, we would expect them to correlate within individuals. Therefore, we first estimated a simple correlation table of each of the health-related behaviors for each of the datasets.

2.1. Within-MZ twins models

Our next goal was to determine if schooling is a common cause of multiple health-related behaviors. While a simple regression of health-related behaviors on schooling would quantify the association between schooling and each health-related behavior, both schooling and health-related behaviors may be determined by unobserved characteristics (such as unobserved dimensions of parental and family background, genetic dispositions, and the childhood environment). By comparing differences in schooling and health-related behaviors, within-MZ twins regressions can net out confounding from these unobserved factors, since identical twins have identical genes at birth, the same parental and family characteristics, and largely the same childhood environment. For example, for a health-related behavior y_i for individual i, the regression of y_i on schooling would be:

$$y_i = \beta_0 + \beta_1 + \beta_2 schooling_i + \beta_3 age + \beta_4 male + \gamma z_i + \varepsilon_i$$

where z_i are the unobserved parental, family, genetic, and child environmental characteristics discussed above. The β_2 is the association between schooling and behavior y, but it is not the causal effect, since both schooling and behavior y are affected by z. By comparing the within-MZ twins difference in both schooling and health-related behaviors, we can instead estimate the following regression for twinship j:

$$\left(y_{1j} - y_{2j}\right) = \beta_1\left(schooling_{1j} - schooling_{2j}\right) + \gamma(z_{1j} - z_{2j}) + (\varepsilon_{1j} - \varepsilon_{2j})$$

Since MZ twins have identical genes at birth, parental and family backgrounds, and childhood environments, $z_{1j} - z_{2j}$ cancels out, removing the confounding from these unobserved factors.

These models are subject to some potential limitations. First, we have to assume that the source of the within-MZ twins difference in schooling is unrelated to the within-MZ difference in each health-related behavior. If, for example, the same shock caused one twin to discontinue schooling before their cotwin and make them smoke, the within-MZ estimate would falsely attribute the smoking difference between twins to the schooling difference, rather than the true unobserved shock. Therefore, if this assumption is violated, the within-MZ estimates becomes a bound on the true on the true causal estimate (Kohler et al., 2011). In addition, if there is measurement error in schooling, the degree of error would be increased for the within-MZ twins regression, biasing the estimated effect towards zero (Bound and Solon, 1999). The plausibility of these estimates depends on the size of the within-twins differences in both schooling and each outcome; in Appendix Figs. 1–3 we graph the within-twins distributions and find a wide range of differences across twin pairs. While these sources of bias may be important, both produce predicable bounds on the true causal estimate (Kohler et al., 2011). Despite these limitations, the within-MZ regressions provide a robust approach for controlling for unobserved characteristics that may confound the schooling and health-related behavior relationship. We therefore estimated a regression of the form (2) for each of the health-related behaviors.

2.2. Behavioral genetics models

While the economics literature has focused on the effects of schooling on health and health-related behaviors, behavioral genetics has focused on the role of genetics and environments. In many behavioral genetics studies, observed characteristics like health-related behaviors are expressed as the result of additive genetic endowments (A), the shared environment between twins (C), and individual environmental factors (E). Each health-related behavior can be the result of its own A, C, and E, or the A, C, E factors that also determine other behaviors. The degree to which multiple health-related behaviors are determined by a common set of genetic, shared environment, and individual environmental factors can then be determined by seeing how much of the variance in multiple behaviors is due to a common subset of A, C, E factors and how much variation is due to behavior-specific factors. This is the intuition behind the multivariate ACE model, which can be represented by the path diagrams in Fig. 1 (the figure is shown for only two health-related behaviors for clarity, but this approach generalizes to any number of behaviors).

Here, x_{ij}^1 and x_{ij}^2 are two behaviors for individual i in twin pair j

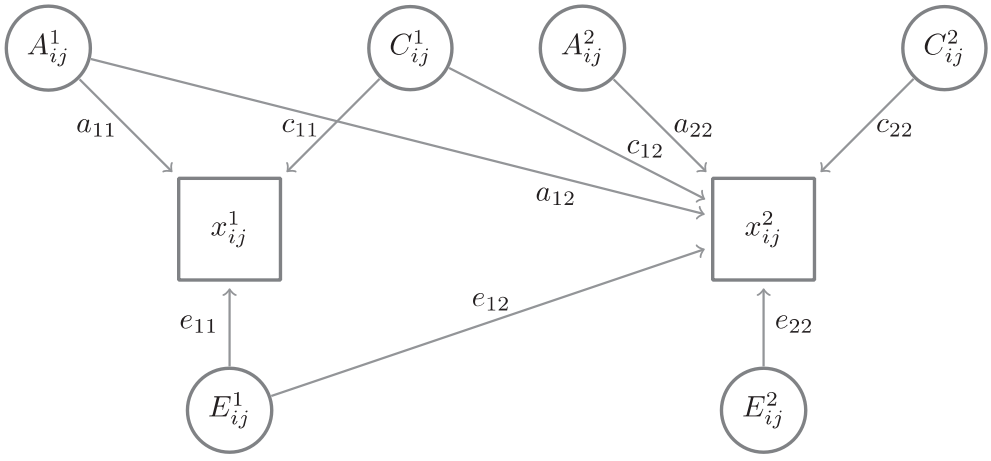


Fig. 1. Path diagrams for the multivariate ACE model.

and all the A_{ij}^k, C_{ij}^k, and E_{ij}^k are the behavior-specific factors. As the diagram shows, each behavior can be the result of its own *A*, *C*, and *E* factor (paths a_{11}, c_{11}, e_{11}, a_{22}, c_{22}, e_{22}) and the *A*, *C*, *E* factors of the other behaviors (paths a_{12}, c_{12}, e_{12}).

One important conceptual issue arises in the measurement of smoking and drinking. In many behavioral genetic studies of smoking and drinking, researchers assume that every individual has an underlying latent "propensity" for smoking and drinking. Categories such as ever smoker/ever drinker and heavy smoker/heavy drinker simply classify individuals that fall above some threshold on the latent propensities. We follow this approach by combining ever and heavy use into one categorical variable, and then use this model to estimate smoking and drinking as continuous latent propensities.

Using information on both MZ and DZ twins and assuming that MZ twins share identical genetic endowments and common environments while DZ twins share identical common environments and on average 50% of their genetic endowments, we can represent the correlations between all the behaviors as a function of the *a*, *c*, and *e* path coefficients. This has the advantage of then letting us determine how much of the correlation between the behaviors is due to common genetic factors (*A*), common shared environments between twins (*C*), and common individual idiosyncratic environments (*E*) by looking at the correlations generated by just the subset of the *a*, *c*, and *e* path coefficients respectively. For more details on the estimation of these models see: (Neale and Cardon, 1992).

We determine the role of a common set of genetic, shared environment, and individual environmental factors by using the model presented in Fig. 1 to first estimate the correlation between behaviors as a function of all the path coefficients. We then decompose these correlations into the contribution of genetic endowments, shared environments, and individual environments. Large factor-specific contributions to the correlations would imply that a common set of factors is influencing multiple behaviors.

In many twins studies, researchers fit alternative models that assume some factors have no influence (AE, CE, and E models)—in Appendix Table 1 we compare the fit of these sub-models to the standard ACE model and find that the ACE provides the best statistical fit for two of the three datasets. Although the AE model provides the best statistical fit for one dataset, our theoretical question revolves around the role of shared environments, so we did not want to constrain this factor to be 0. Similarly, we do not estimate models with genetic dominance effects since they cannot be identified simultaneously with the shared environment parameters unless one is willing to assume an absence of additive genetic effects (an assumption that is generally not plausible).

The behavioral genetics models also make a number of important assumptions that have implications for the results. First, the models assume that the means and variances of each behavior are equal across MZ and DZ twins. In Appendix Table 2 we present the proportions, means, and standard deviations across all the variables and find that the levels for most variables are similar across zygosity. Still, there are differences in heavy smoking and heavy drinking across zygosity that may lead to error in the model estimation. Second, the models as presented here assume no gene-environment interactions. This is an important assumption and can potentially bias the genetic contributions if the size of the genetic contribution varies based on environmental interactions (Mustelin et al., 2009). Third, the models assume that the influence of the shared environment is equivalent for both MZ and DZ twins. If, for example, parents were more likely to treat MZ twins similarly compared to DZ twins, the size of the A contributions would be biased upward, leading to inflated estimates of the role of genetics. The models also assume that there is no assortative mating in the population. If individuals with similar health-related behaviors were more likely to have children, the estimated C contributions would be biased upward. Finally, measurement error in the outcomes can lead to inflated estimates of E while biasing the A and C estimates downward. This bias would lead to conservative estimates of the contribution of genetics and shared environments. Although these assumptions are important to consider, the behavioral genetic models still provide a strong way to assess the relationship between genetic and environmental factors and adult health-related behaviors.

3. Results

Table 1 presents a descriptive overview of the three twins samples. The MIDUS and MTR samples are on average middle aged (47.07 years old for MTR and 47.53 for MIDUS) while individuals in the Add Health are slightly younger (28.93 years). All three samples have a greater share of women compared to men–this difference is especially pronounced for the MTR sample (65.13% female). Most of our analyses focus specifically on differences within twins pairs and would not be biased by the sex composition of the samples. Across

Table 1
Descriptive characteristics of the Add Health, MIDUS, and MTR twins samples.

	Add Health Twins		MIDUS Twins		MTR Twins	
	N = 746		N = 1506		N = 2306	
	Mean or n	SD or %	Mean or n	SD or %	Mean or n	SD or %
Age	28.93	1.62	47.53	12.31	47.07	5.62
Sex						
Male	362	48.53	630	41.83	804	34.87
Female	384	51.47	876	58.17	1502	65.13
Zygosity						
MZ	412	55.23	832	55.25	1294	56.11
DZ	334	44.77	674	44.75	1012	43.89
Ever smoker	292	39.14	824	54.71	947	41.07
Heavy smoker	41	5.5	212	14.08	292	12.66
Ever drinker	577	77.35	1147	76.16	1628	70.6
Heavy drinker	147	19.17	330	21.91	136	5.9
Vigorous activity per month	–	–	6.37	5.36	–	–
Vigorous activity per week	2.44	2.56	–	–	–	–
BMI	28.07	7.29	28.59	5.10	25.82	4.64

Notes: Data are shown for the total number of people (the number of twin pairs is the total sample size divided by 2). The MTR twins did not contain questions on drinks per sitting (heavy drinking in the MTR is measured as drinking more than 3 days per week) or physical activity. Two different measures of vigorous activity are presented since the Add Health and MIDUS surveys asked physical activity over different recall periods.

all four of the identified health-related behaviors, we observe a common pattern: large fractions of individuals have ever smoked or drank with a much smaller number of individuals currently consuming heavy quantities. For example, between 30% and 40% of individuals in all three samples reported ever smoking; in contrast, the fraction that currently heavy smoke is only between 5% and 14%. Similar patterns are observed for drinking: over 70% of individuals reported ever drinking in all three samples but only around 20% currently consume four or more drinks per sitting (based on the Add Health and MIDUS samples). Although average levels of vigorous physical activity are fairly low (2.44 times per week among the Add Health sample and 6.37 times per month in the MIDUS sample), both measures have large standard deviations, implying a wide distribution in physical activity behavior. Based on the standard Centers for Disease Control and Prevention cutoffs for BMI, the samples are on average slightly overweight.

Figs. 2–4 graph the correlation matrix of the selected health-related behaviors for all three samples. The below diagonal elements are the scatterplots of the behaviors against one another while the above diagonal elements are the correlation coefficients. Across all three samples, the most striking initial result is the lack of correlation among many of the behaviors. For example, heavy smoking and physical activity has a correlation of -0.083 in the Add Health sample and a correlation of -0.077 in the MIDUS sample–implying that individuals that smoke heavily are only very slightly less likely to engage in physical activity. Similarly, the correlation of heavy drinking and BMI is -0.038 in the Add Health sample, 0.014 in the MIDUS sample and -0.032 in the MTR sample. These correlations indicate that individuals who drink heavily are not more likely to have higher levels of unhealthy weight. On first glance, these results suggest that a single factor (whether it is personality, schooling, environments, or genetics) is unlikely to be a strong cause of multiple health-related behaviors since the behaviors themselves do not correlate highly. This general lack of correlation between the health-related behaviors is consistent for almost every pairwise comparison except for one: smoking and drinking. We find a large correlation between ever smoking and heavy drinking in two datasets (0.20 in the Add Health, 0.23 in the MIDUS) and between ever smoking and ever drinking in the MTR data ($r = 0.25$). In the following section, we investigate the role of schooling, genetics, and the childhood and adolescent environment in explaining the covariation between health-related behaviors, paying special attention to smoking and drinking.

In Tables 2–4, we show the results from the OLS and within-twins fixed-effect regressions of each health-related behavior on years of schooling. Focusing on just the OLS regressions, we find the commonly reported conclusion of an association between schooling and better health-related behaviors. In the Add Health sample, a one-year increase in schooling is associated with a lower probability of ever smoking, a lower probability of heavy smoking, an increase in the times an individual engages in vigorous activity per week, and a lower BMI. This pattern of associations between schooling and health-related behaviors is largely similar in the other two samples: in the MIDUS sample schooling is associated with less smoking, less heavy drinking, more vigorous activity per week, and a lower BMI. While these results indicate an association between schooling and health-related behaviors, an important question is whether these associations are robust to unobserved characteristics.

Tables 2–4 also report the within-MZ twins regressions,

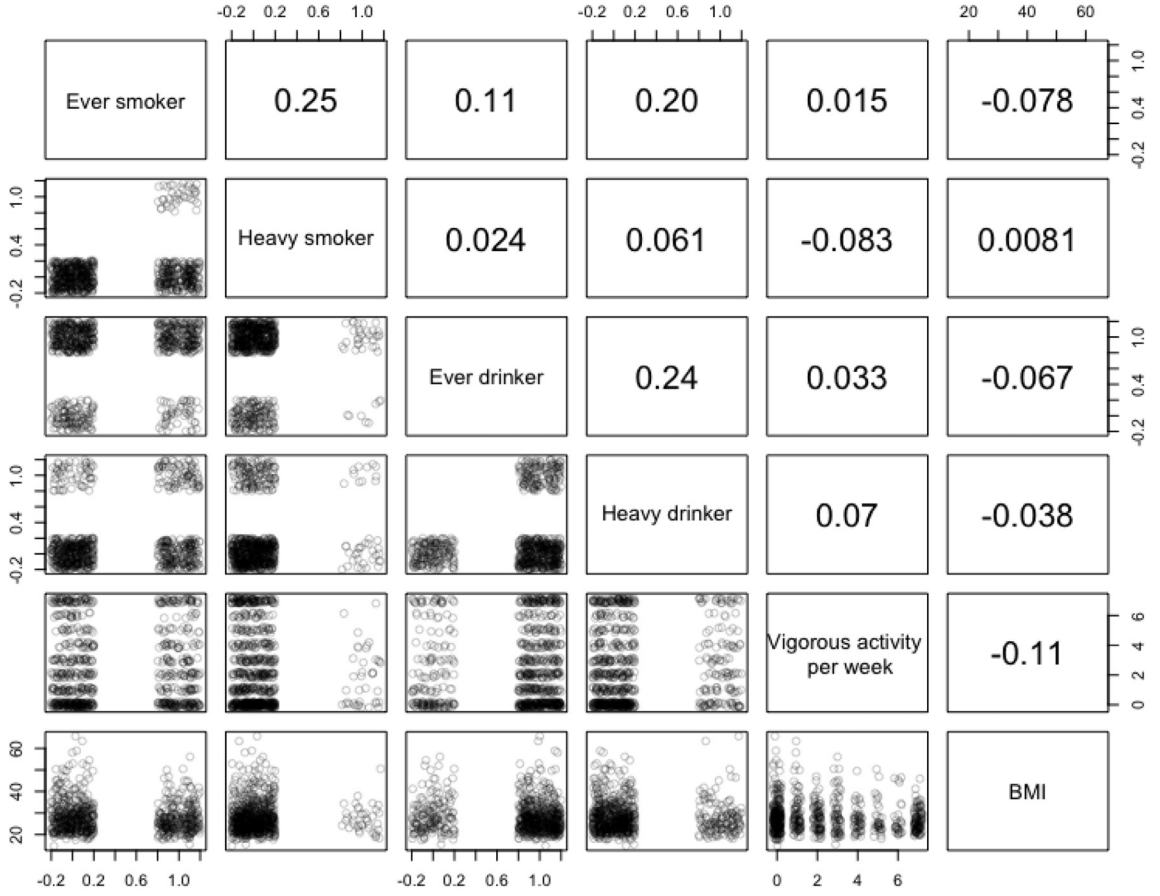


Fig. 2. Correlation matrix and scatter plots for the selected health-related behaviors, Add Health Twins, N = 746.

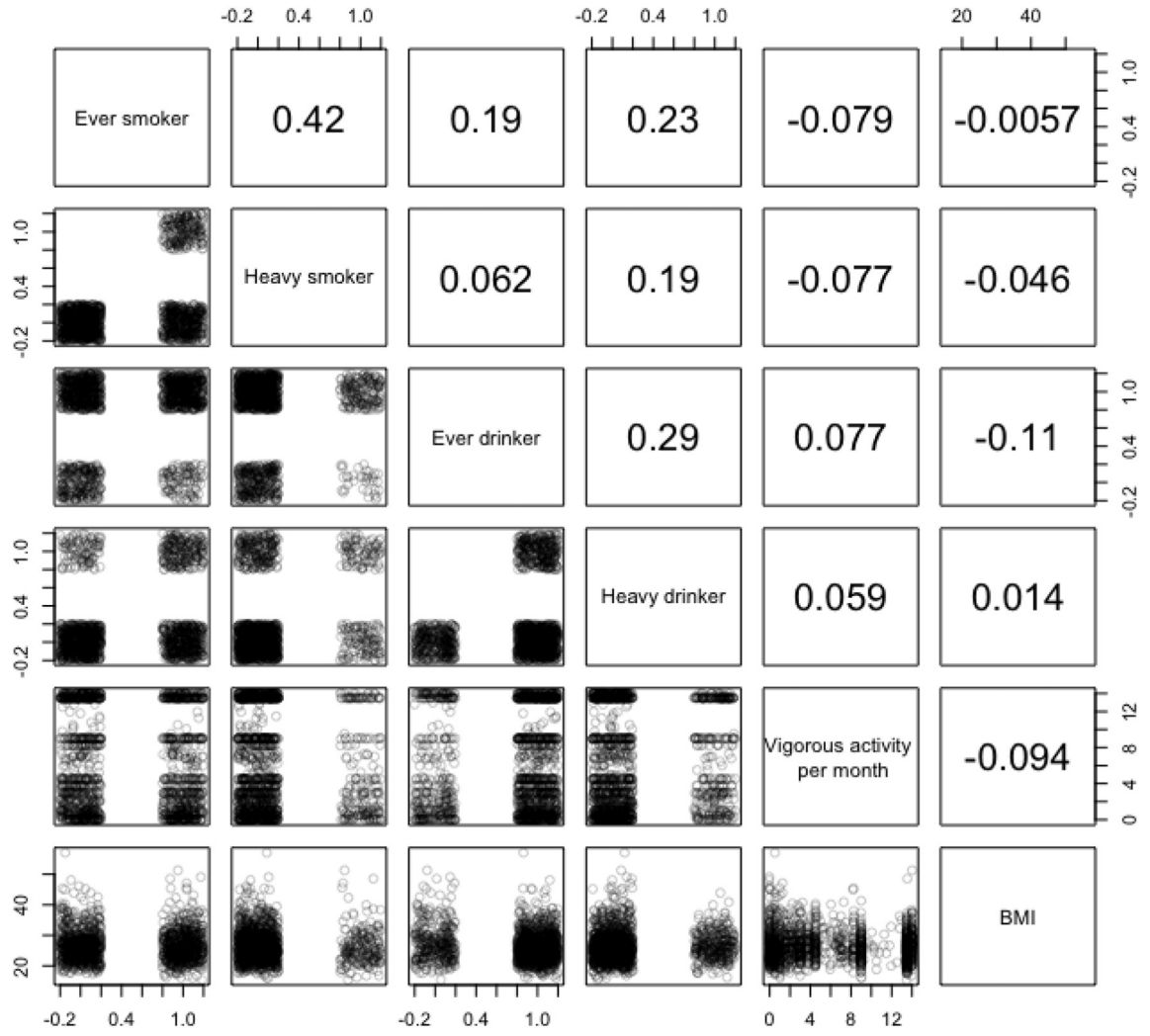


Fig. 3. Correlation matrix and scatter plots for the selected health-related behaviors, MIDUS Twins, N = 1506.

providing a more robust evaluation of the schooling-health-related-behavior relationship (for the Add Health and MIDUS samples both twins were not interviewed on the same day. This resulted in a one-year difference in age between the twins for a minority of cases, leading to an estimated coefficient for age even for the within-MZ models). The within-MZ results display a much different overall pattern compared to the standard OLS results. For most of the significant OLS associations, the within-twins estimates are substantially smaller in magnitude and most lose statistical significance. For example, the relationship between schooling and heavy smoking moves from -0.014 to -0.006 in the Add Health sample, from -0.030 to -0.020 in the MIDUS sample, and from -0.017 to -0.001 in the MTR sample (for the MIDUS sample the within-MZ effect is still significant). Similarly, the coefficient for the BMI outcomes moves from -0.502 to 0.041 in the Add Health, from -0.229 to -0.060 in the MIDUS, and from -0.160 to 0.041 in the MTR sample. Not every relationship diminishes or loses statistical significance. In the MIDUS sample, the OLS and within-MZ coefficients are significant for heavy smoking and in the Add Health sample the OLS and within-MZ estimates are both significant for ever smoking, suggesting that schooling may be related to smoking behavior.

While the results from the schooling regressions (Tables 2–4) suggest that schooling may be related to some health-related behaviors, we find almost no support for the hypothesis that schooling affects all four of the behaviors examined. Focusing specifically on smoking and drinking, the two most correlated health-related behaviors, we find that the schooling effect is much larger in magnitude for smoking than for drinking in all of the three samples (where the schooling-drinking effect is extremely close to zero). These results suggest that schooling is unlikely to be an important common cause of both behaviors.

In Tables 5–7 we move towards investigating the role of genetics and the childhood environment as potential causes of health-related behaviors. For each table, we present the implied correlation matrix calculated through the behavioral genetics model, and the genetic, shared environment, and individual environment specific contributions to the estimated correlations. These second two matrices estimate the portion of the correlation between the behaviors that arise from a common set of genes or shared environments. The diagonals of the genetic, environmental, and individual matrices represent the fraction of variance in each behavior that is consistent with genetic endowments and environmental factors.

Across all three samples, we find that genetic endowments are consistent with a large fraction of the variance in many of the health-related behaviors. For smoking, genetic endowments are consistent with 29% of the variance among the Add Health twins, 27% among the MIDUS twins, and 15% of the variance among the MTR twins. Similarly, genetic endowments are consistent with a large fraction of the variance in BMI: 77% in Add Health, 64% in

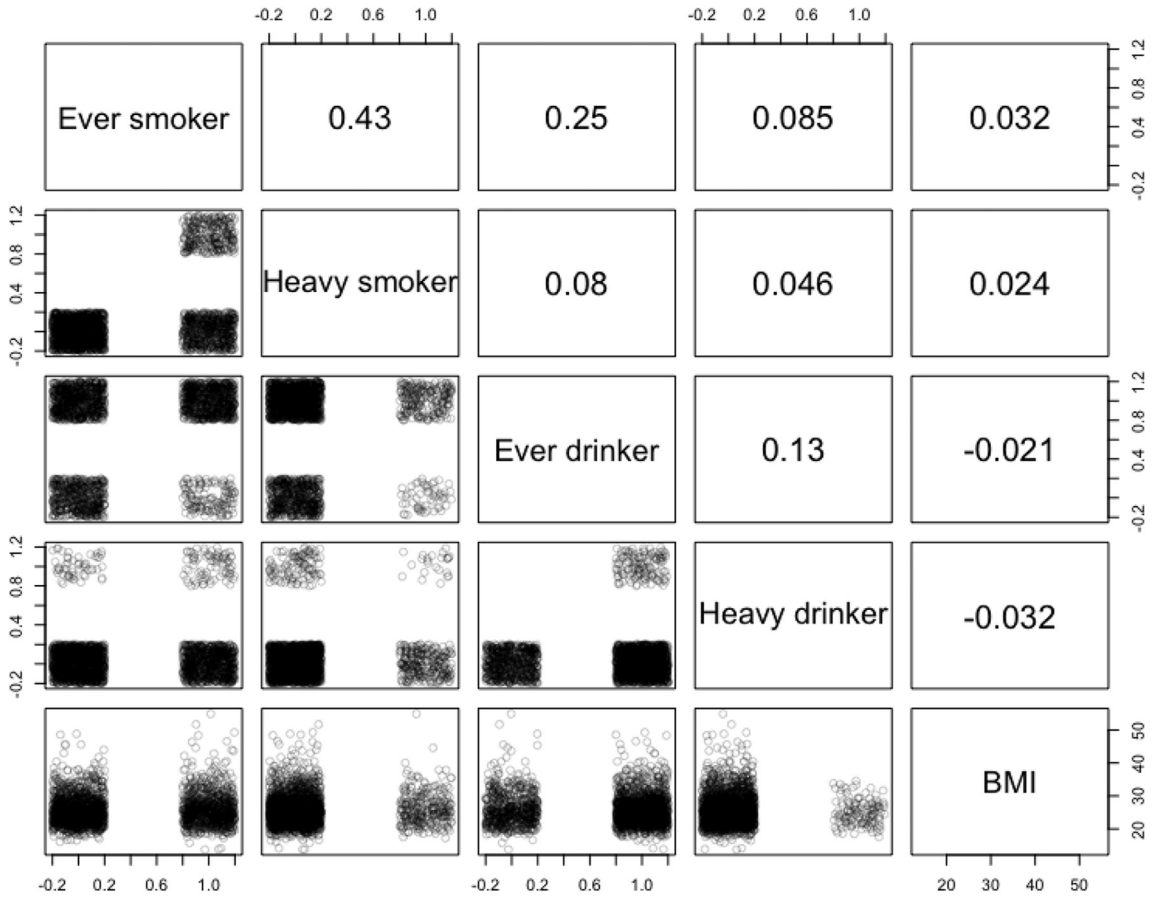


Fig. 4. Correlation matrix and scatter plots for the selected health-related behaviors, MTR Twins, N = 2306.

Table 2
Estimated OLS and within-MZ twins regressions of smoking, drinking, physical activity, and unhealthy weight on schooling, Add Health Twins, N = 412.

Variables	Ever smoker		Heavy smoker		Ever drinker		Heavy drinker		Vigorous act per week		BMI	
	OLS	FE	OLS	FE	OLS	FE	OLS	FE	OLS	FE	OLS	FE
Years of schooling	−0.053***	−0.043*	−0.014**	−0.006	0.035***	0.007	−0.005	0.008	0.134*	0.056	−0.502**	0.041
	(0.010)	(0.018)	(0.005)	(0.004)	(0.009)	(0.016)	(0.008)	(0.013)	(0.059)	(0.112)	(0.163)	(0.108)
Age	0.034+	0.062	0.022**	−0.053	−0.018	−0.051	−0.013	−0.301+	0.037	1.039	−0.191	−3.506
	(0.018)	(0.165)	(0.008)	(0.054)	(0.016)	(0.162)	(0.014)	(0.170)	(0.089)	(1.009)	(0.277)	(2.869)
Male	0.028		0.033		0.128**		0.125**		0.580*		1.560+	
	(0.055)		(0.024)		(0.046)		(0.044)		(0.286)		(0.932)	
R-squared	0.081	0.032	0.050	0.003	0.064	0.001	0.027	0.017	0.032	0.007	0.040	0.028

Standard errors are clustered by twinship. Linear probability models were estimated for dichotomous outcomes.
***p < 0.001, **p < 0.01, *p < 0.05, + p < 0.1.

Table 3
Estimated OLS and within-MZ twins regression of smoking, drinking, physical activity, and BMI on schooling, MIDUS Twins, N = 832.

Variables	Ever smoker		Heavy smoker		Ever drinker		Heavy drinker		Vigorous act per month		BMI	
	OLS	FE	OLS	FE	OLS	FE	OLS	FE	OLS	FE	OLS	FE
Years of schooling	−0.044***	−0.014	−0.030***	−0.020*	0.011+	0.007	−0.020***	−0.002	0.230**	0.160	−0.229**	−0.060
	(0.007)	(0.010)	(0.006)	(0.009)	(0.006)	(0.011)	(0.005)	(0.008)	(0.079)	(0.141)	(0.079)	(0.081)
Age	0.002	−0.028	−0.002	−0.005	−0.008***	0.002	−0.007***	−0.074	−0.096***	−0.823	0.030+	0.309
	(0.002)	(0.064)	(0.001)	(0.060)	(0.001)	(0.070)	(0.001)	(0.064)	(0.016)	(0.825)	(0.018)	(0.616)
Male	0.094*		0.021		0.071*		0.258***		1.180**		0.946*	
	(0.042)		(0.028)		(0.035)		(0.033)		(0.401)		(0.416)	
R-squared	0.063	0.004	0.050	0.012	0.064	0.001	0.145	0.004	0.075	0.004	0.029	0.002

Standard errors are clustered by twinship. Linear probability models were estimated for dichotomous outcomes.
***p < 0.001, **p < 0.01, *p < 0.05, + p < 0.1.

Table 4
Estimated OLS and within-MZ twins regression of smoking, drinking, and BMI on schooling, MTR Twins, N = 1294.

	Ever smoker		Heavy smoker		Ever drinker		Heavy drinker		BMI	
	OLS	FE	OLS	FE	OLS	FE	OLS	FE	OLS	FE
Years of schooling	−0.040*** (0.005)	−0.016 (0.010)	−0.017*** (0.003)	−0.001 (0.007)	−0.001 (0.005)	−0.003 (0.007)	0.004+ (0.002)	0.007 (0.005)	−0.160** (0.054)	0.041 (0.061)
Age	−0.008** (0.003)		−0.004* (0.002)		0.000 (0.003)		−0.003* (0.001)		−0.096*** (0.029)	
Male = 1	0.159*** (0.033)		0.034 (0.021)		0.197*** (0.029)		0.043** (0.015)		1.336*** (0.308)	
R-squared	0.073	0.005	0.025	0.000	0.042	0.000	0.021	0.003	0.038	0.001

Standard errors are clustered by twinship. Linear probability models were estimated for dichotomous outcomes.
***p < 0.001, **p < 0.01, *p < 0.05, + p < 0.1.

Table 5
Estimated correlation matrix with genetic, shared environment, and individual environment contributions, Add Health Twins, N = 746.

	Estimated Correlation Matrix					Genetic Contributions			
	Smoking Propensity	Drinking Propensity	Vigorous activity per week	BMI		Smoking Propensity	Drinking Propensity	Vigorous activity per week	BMI
Smoking Propensity	1.00				Smoking Propensity	0.29			
Drinking Propensity	0.22	1.00			Drinking Propensity	0.03	0.11		
Vigorous activity per week	0.01	0.05	1.00		Vigorous activity per week	0.03	0.08	0.27	
BMI	−0.08	−0.07	−0.11	1.00	BMI	0.02	−0.12	−0.12	0.77
	Shared Environment Contributions					Individual Environment Contributions			
	Smoking Propensity	Drinking Propensity	Vigorous activity per week	BMI		Smoking Propensity	Drinking Propensity	Vigorous activity per week	BMI
Smoking Propensity	0.34				Smoking Propensity	0.37			
Drinking Propensity	0.10	0.30			Drinking Propensity	0.09	0.59		
Vigorous activity per week	−0.04	0.01	0.04		Vigorous activity per week	0.02	−0.04	0.69	
BMI	−0.07	0.04	0.04	0.06	BMI	−0.03	0.01	−0.03	0.17
Model estimation details									
Estimated parameters	38								
Degrees of freedom	2948								
-2loglikelihood	6467.586								
AIC	571.5858								
BIC	−10989.2274								

Table 6
Estimated correlation matrix with genetic, shared environment, and individual environment contributions, MIDUS Twins, N = 1506.

	Estimated Correlation Matrix					Genetic Contributions			
	Smoking Propensity	Drinking Propensity	Vigorous activity per month	BMI		Smoking Propensity	Drinking Propensity	Vigorous activity per month	BMI
Smoking Propensity	1.00				Smoking Propensity	0.27			
Drinking Propensity	0.33	1.00			Drinking Propensity	0.14	0.33		
Vigorous activity per month	−0.12	0.10	1.00		Vigorous activity per month	−0.03	0.21	0.25	
BMI	−0.03	−0.08	−0.09	1.00	BMI	−0.03	0.02	−0.02	0.64
	Shared Environmental Contributions					Individual Environment Contributions			
	Smoking Propensity	Drinking Propensity	Vigorous activity per month	BMI		Smoking Propensity	Drinking Propensity	Vigorous activity per month	BMI
Smoking Propensity	0.47				Smoking Propensity	0.26			
Drinking Propensity	0.17	0.39			Drinking Propensity	0.02	0.28		
Vigorous activity per month	−0.07	−0.09	0.03		Vigorous activity per month	−0.02	−0.02	0.72	
BMI	0.04	−0.10	0.02	0.06	BMI	−0.04	0.00	−0.09	0.30
Model estimation details									
Estimated parameters	36								
Degrees of freedom	5990								
-2loglikelihood	13,200.03								
AIC	1220.032								
BIC	−26478.119								

Table 7
Estimated correlation matrix with genetic, shared environment, and individual environment contributions, MTR Twins, N = 2306.

	Estimated Correlation Matrix				Genetic Contributions		
	Smoking Propensity	Drinking Propensity	BMI		Smoking Propensity	Drinking Propensity	BMI
Smoking Propensity	1.00			Smoking Propensity	0.15		
Drinking Propensity	0.33	1.00		Drinking Propensity	0.06	0.49	
BMI	0.04	−0.04	1.00	BMI	0.13	−0.02	0.68
	Shared Environment Contributions				Individual Environment contributions		
	Smoking Propensity	Drinking Propensity	BMI		Smoking Propensity	Drinking Propensity	BMI
Smoking Propensity	0.41			Smoking Propensity	0.44		
Drinking Propensity	0.18	0.14		Drinking Propensity	0.09	0.37	
BMI	−0.03	−0.01	0.00	BMI	−0.06	−0.01	0.32
Model estimation details							
Estimated parameters	24						
Degrees of freedom	6896						
-2loglikelihood	13,592.4						
AIC	−199.5957						
BIC	−35025.2406						

MIDUS, and 68% in MTR. The role of the shared, mostly childhood, environment is less pronounced for BMI and physical activity across the datasets. For example, the shared environment is consistent with 6% of the variance in BMI and 4% of vigorous activity for the Add Health sample. We observe a relatively similar pattern in the MIDUS data, with 6% of the variance in BMI and 3% of the variance in vigorous activity consistent with shared environmental factors. However, the results suggest that the childhood and adolescent environment plays an important role in smoking and drinking behavior in adulthood. One of the more surprising findings is that across all three samples and all behaviors, a large fraction of the variation in the each of the behaviors is due to individual idiosyncratic environments. While this term also captures measurement and specification errors, these results suggest that despite the potential role of schooling, genetics, and environments in explaining portions of the variation and covariation in these four behaviors, much of the variance is idiosyncratic and behavior specific.

The off-diagonal elements of the matrixes measure the correlation between behaviors consistent with a common set of genetic endowments or environments. As mentioned previously, the one pairwise comparison with a large correlation coefficient is smoking and drinking. For all three samples, we find that a large portion of this correlation is consistent with a common environmental factor (environmental contribution is 0.10 in the Add Health sample, 0.17 in the MIDUS sample, and 0.18 in the MTR sample).

For the other pairwise comparisons, the role of a common set of genetic endowments and environments is inconsistent across the three samples. For example, we find that a common set of genetics is consistent with the covariation in smoking and drinking among the MIDUS twins (contribution = 0.14), but this contribution is not present in the Add Health or MTR data. We also find a moderate genetic correlation between cigarette smoking and BMI in the MTR sample (contribution = 0.13) that is not present in the other two samples. The inconsistent correlations across the datasets for most of the pairwise comparisons of behaviors is not surprising, since many of these behaviors do not have strong overall correlations.

3.1. Robustness

We conducted a number of robustness checks. First, our results were consistent when using continuous measures of smoking and drinking. Our results were also consistent when looking at just moderate physical activity and a measure that combined both moderate and vigorous physical activity. As mentioned previously, the within-MZ regressions may be biased towards zero if there is measurement error in schooling. Although only available in the MTR dataset, we used co-twin reported schooling as an instrument for an individual's schooling and estimated instrumental variable regressions to reduce bias from measurement error. We find that measurement error in the MTR dataset does not affect our conclusions, with the coefficient actually becoming smaller for some outcomes (Appendix Table 3).

4. Discussion

Health-related behaviors are significant contributors to morbidity and mortality in the United States, yet evidence on the underlying causes of the vast within-population variation in behaviors is mixed. While many potential causes of health-related behaviors have been identified—such as schooling, genetics, and environments—the magnitude of the variation across multiple behaviors that is due to a common set of causes remains an open question. Using three data sources on U.S. twins, we do not find evidence that schooling, or a common set of genetic endowments or environments are a common cause of most health-related behaviors. Smoking and excessive alcohol consumption is the main exception: we find evidence that variation in both adult smoking and drinking is consistent with a common shared environment between twins (mostly the childhood environment). Overall, the results of our study suggest that the causes for health-related behaviors in adulthood are largely idiosyncratic.

Our first primary conclusion is that across all three samples, the key health-related behaviors investigated in this paper do not correlate as strongly as we, and probably many others, would have expected. While theories on the causes of health-related behaviors across many disciplines imply that many behaviors have a common underlying cause, and should therefore correlate, the patterns in our data are not consistent with this expectation. Individuals that smoke are not substantially less likely to be physically active or more likely to have unhealthy weight. Similarly, we observe very weak correlations between physical activity and unhealthy weight, and unhealthy drinking and physical activity. These findings suggest that individuals selectively engage in some unhealthy behaviors but not necessarily multiple behaviors. While perhaps surprising and counter-intuitive, this conclusion is consistent with research on the correlation between health behaviors using the Behavioral Risk Factor Surveillance System in the United States (Cutler and Glaeser, 2005). The one main exception to the lack of correlation across health-related behaviors is the relationship between smoking and drinking (drinks per sitting or day): across all three of the samples, we find that individuals who smoke more are

also more likely to drink more per sitting. This finding has precedent in the literature, with many studies documenting an association between the two behaviors (De Leon et al., 2007; Hagger-Johnson et al., 2013; Room, 2004). Despite the lack of correlation between many behaviors, the presence of a correlation between smoking and drinking is important, since smoking and heavy drinking are the two health-related behaviors associated with the largest burden of morbidity and mortality (McGinnis and Foege, 1993; Mokdad et al., 2004). Interventions aimed at the cause of this correlation may provide a strong way to improve population health.

Our second main conclusions is that the relationship between schooling and health-related behaviors is unlikely to be causal: while we initially find many strong associations between schooling and the health-related behaviors, most of these associations attenuate and become non-significant after controlling for unobserved differences shared between MZ twins. Schooling also seems an unlikely explanation for the relationship between smoking and drinking: while the size of the relationship between schooling and smoking is relatively large and consistent across datasets, this coefficient is very small for drinking–in some cases, the coefficient even suggests opposite associations, where more schooling makes an individual more likely to drink heavily. The results imply that schooling is questionable as a common cause of both smoking and drinking. Although these results may be surprising, they are consistent with prior studies that use within MZ-twins designs, including (Amin et al., 2015; Behrman et al., 2011, 2015; Fujiwara and Kawachi, 2009; Kohler et al., 2011). These papers generally find that the cross-sectional associations between schooling and health largely overstate the potential relationship–in many cases, the relationship becomes very small in magnitude and loses statistical significance. The estimates from this paper differ from studies of the effect of schooling that use natural experiments and instrumental variables (Clark and Royer, 2013; Lleras-Muney, 2005). Although most of these studies find that schooling has a plausibly causal effect on health, these results are only identified for very specific margins of the population, and thus are usually not generalizable to larger populations. Due to the wide range of within-twins differences in schooling and health-related behaviors, our results are identified for a larger subset of the population and come closer to estimating an average treatment effect (In Appendix Figs. 1–3 we show the distributions of within-twins differences in schooling and each of the behaviors–these graphs highlight the wide range of differences on which the within-MZ twins models are estimated over).

Finally, based on the results of the behavioral genetic analyses, we find that the greatest portion of variance for each health-related behavior is related to behavior-specific factors, suggesting that the causes of health-related behaviors are largely idiosyncratic. We also find that genetic endowments are consistent with significant portions of the variance in most of the behaviors. These two results have been found in other behavioral genetic studies on the heritability of individual behaviors (Bauman et al., 2012; Min et al., 2013; Vink et al., 2005; Walters, 2002)–these studies find small contributions from environments, reasonably large genetic contributions, and large individual environment contributions. However, we find that genetic endowments are not consistent with the covariation between the behaviors. The lack of support for a common set of genes that causes multiple unhealthy behaviors may arise if the elevated risk of mortality for individuals with these gene expressions resulted in selective genetic pressure over time–effectively selecting out such sets of genes. Despite the idiosyncratic origins of the health-related behaviors, we find consistent evidence that the correlation between smoking and unhealthy drinking is associated with a common environmental factor: a large part of the correlation between smoking and unhealthy drinking is consistent with a common source of the shared, mostly childhood, environment between twins. This finding suggests that modifying the childhood environment may provide a plausible policy solution to reduce both smoking and unhealthy drinking behavior in adulthood.

In interpreting the results of this study, it is important to address some limitations of our study design. In order for the within-MZ estimates to be causal, we have to assume that the cause of the within-twins difference in schooling was unrelated to the within-twins difference in behaviors, except through schooling, though the violation of this condition produces predictable bounds on the causal estimates (see: Kohler et al., 2011). Furthermore, the outcome variable for one twin cannot depend on the outcome variable for another twin beyond their joint dependence on genetic endowments and childhood environments, although the violation of this condition produces predictable biases that have been discussed extensively elsewhere (see: Kohler et al., 2011). For our estimates of the variance attributable to common environments, we also assume that the common environments of MZ twins are the same as the common environment of DZ twins. However, this assumption only applies to the behavioral genetics models and is not needed for the within-MZ twins estimates. After controlling for any unobserved difference between twins through the within-twins estimates, we assume that the population of twins is representative of the larger American population and that the underlying causes of schooling and health-related behaviors are the same for twins as for the American population. The samples are overwhelming white, and the results estimated might not be generalizable to the unique childhood contexts experienced by other race/ethnic groups in the United States or in other societies if there are interactive race/ethnic effects. Twins studies in general have been criticized for several reasons. For example, studies have found that MZs are not perfectly identical genetically, especially when considering epigenetic processes (Petronis, 2006). Although such considerations mean that the control for unobservable factors afforded by MZs is less than it would be if they also controlled for epigenetic processes, they do not negate the substantial advantages of twin controls over uncontrolled population-based studies that simply ignore genetic processes and unobserved childhood family background characteristics in exploring associations between risks and outcomes. Similarly, the validity of the so-called equal environment assumption, which holds that MZs share no more common environmental experiences than DZs, has been questioned (Joseph, 2002). Nevertheless, this hypothesis is testable and has generally been supported in the literature (Kendler et al., 1993). Moreover it is not relevant for the within-MZ estimates. Yet another criticism holds that modern genomic methods and detailed biological understanding of genomics have caused twins-based methods to become antiquated. However, considering that Genome Wide Association Studies (GWAS) often identify only very small single-gene effects on health and behaviors, twins and related study designs continue to be relevant to obtain a comprehensive assessment of the genetic and social determinants of health and health-related behaviors (Van Dongen et al., 2012). Finally, researchers have questioned whether twins samples are representative of the populations from which they were drawn. Once again, this hypothesis is testable, and studies have generally reported little or no differences between twins and singleton populations with the exception of birth weights. For example, a recent study that performed MRI brain scans found no significant differences between twins and unrelated, age- and sex-matched singletons in several brain structures (Ordaz et al., 2010). Moreover within-twins estimates control for the additive effect of whatever might be distinctive about being a twin. There is a threat

that the smaller coefficients and larger standard errors of the within-twins estimates is due to magnifying of measurement error (Bound and Solon, 1999). While the MTR data ask about co-twin data, allowing for the possibility of instrumenting, the other datasets did not permit this. While this is an important consideration, the results from instrumental variable regression for the MTR sample suggest that measurement error is not driving our results (Appendix Table 3). The MIDUS and MTR samples had a large degree of individuals dropped for incomplete data. In Appendix Table 4, we show the mean levels of the main variables for those included and excluded and find that most of the variables are similar with differences across smoking and sex. However, these differences would only bias our result if the estimated relationships displayed interaction effects with the unbalanced variables. Importantly, our results may still be biased if those excluded were different from the included sample in unobserved ways that related to both schooling and health-related behaviors. Similarly, if individuals were missing due to premature mortality resulting from multiple poor health-related behaviors, we may underestimate the covariation between poor behaviors, since those with the greatest correlation would be dropped. Given the average ages of the samples, however, the role of selective mortality is likely minor.

Despite these limitations, our study is one of the first to explicitly examine the role of schooling, genetic endowments, and environments as common causes of multiple health-related behaviors. By presenting analyses common to both economics and behavioral genetics, we are able to provide a rich examination of the relationship between multiple health-related behaviors and their causes. We find that most health-related behaviors in adulthood are largely idiosyncratic and likely not caused by single factors, whether that is schooling, genetics, or environments. Our results suggest that programs that categorically target all health-related behaviors in adulthood may not produce changes across all behavioral domains–policies to improve health-related behaviors might be most effective if targeted at specific behaviors. Similarly, research on the causes of health-related behaviors should consider each behavior uniquely. The one prominent exception to this pattern is the relationship between smoking and unhealthy drinking: although the environmental correlation between these two is modest, our results suggest that a common aspect of the childhood and adolescent environment is consistent with variation in both behaviors. Research and policy to identify and modify this source may provide a strong way to reduce the population health burden of smoking and heavy drinking.

Appendix

Appendix Table 1
Likelihood ratio tests of alternative twin models, Add Health, MIDUS, and MTR twins.

	# Parameters	-2logLL	Degrees of freedom	P-value
Add Health				
ACE	38	6467.586	2948	Reference
AE	28	6474.119	2958	0.769
CE	28	6540.775	2958	0.000
E	18	6880.66	2968	0.000
MIDUS				
ACE	36	13,200.03	5990	Reference
AE	26	13,233.94	6000	0.000
CE	26	13,279.41	6000	0.000
E	16	14,027.45	6010	0.000
MTR				
ACE	24	13,592.4	6896	Reference
AE	18	13,607.61	6902	0.019
CE	18	13,703.8	6902	0.000
E	12	14,422.4	6908	0.000

Appendix Table 2
Proportions, means, and standard deviations by zygosity.

	Add Health		MIDUS		MTR	
	MZ	DZ	MZ	DZ	MZ	DZ
Categorical						
Smoker						
Never	63.1%	57.5%	66.3%	62.2%	62.3%	54.6%
Ever	31.3%	37.1%	20.1%	23.1%	25.4%	32.2%
Heavy	5.6%	5.4%	13.6%	14.7%	12.3%	13.1%
Drinker						
Never	23.5%	21.6%	23.7%	24.0%	29.8%	28.9%
Ever	57.5%	57.8%	57.2%	50.6%	65.6%	63.5%
Heavy	18.9%	20.7%	19.1%	25.4%	4.6%	7.6%
Continuous						
Vigorous activity per week						
Mean	2.4	2.5				
SD	2.5	2.6				
Vigorous activity per month						
Mean			6.4	6.3		
SD			5.3	5.4		
BMI						
Mean	28.0	28.2	26.4	26.9	25.8	25.9
SD	7.3	7.3	4.9	5.3	4.6	4.7

Appendix Table 3
Estimated OLS, within-MZ twins, and within-MZ twins IV regressions of smoking, drinking, and BMI on schooling, MTR Twins, N = 1.294.

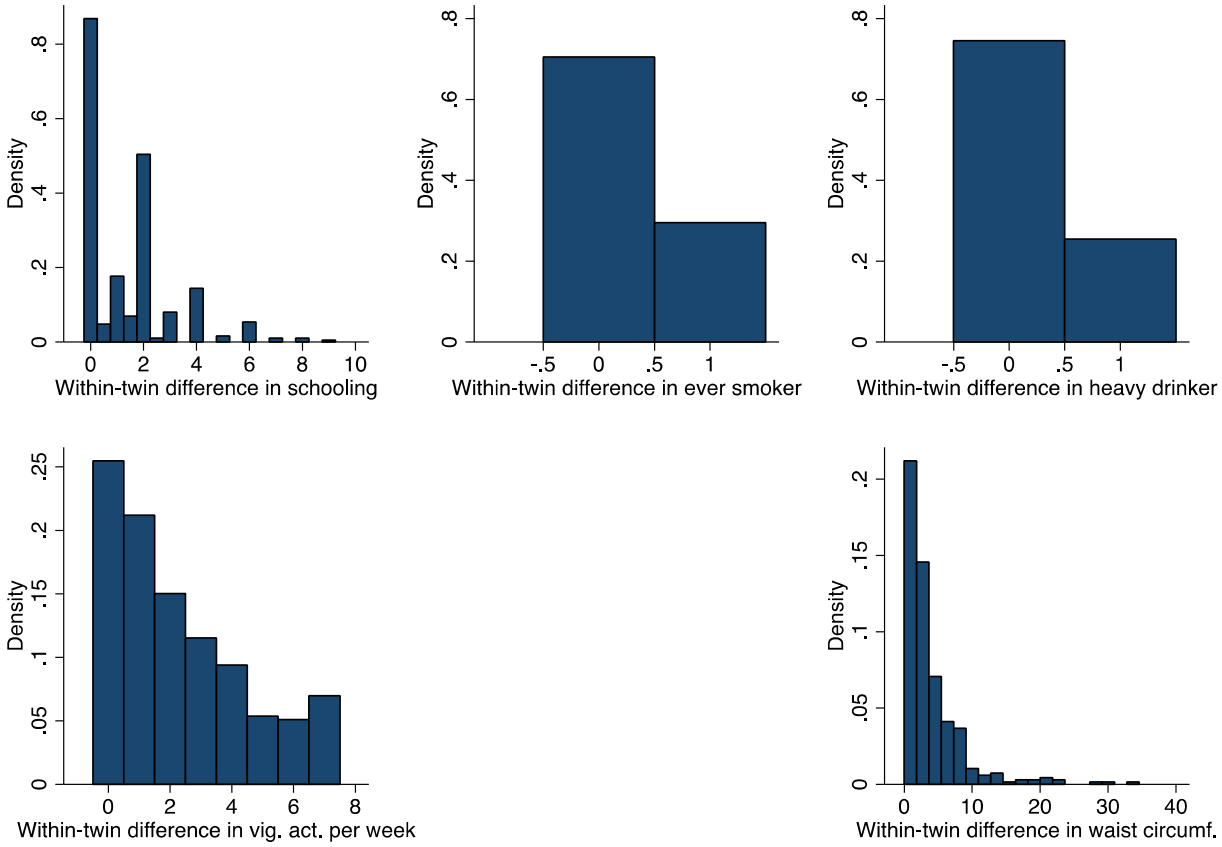
Variables	Ever Smoker			Heavy Smoker			Ever Drinker			Heavy Drinker			BMI		
	OLS	FE	FE IV	OLS	FE	FE IV	OLS	FE	FE IV	OLS	FE	FE IV	OLS	FE	FE IV
Years of schooling	−0.040*** (0.005)	−0.016 (0.010)	−0.012 (0.015)	−0.017*** (0.003)	−0.001 (0.007)	−0.001 (0.012)	−0.001 (0.005)	−0.003 (0.007)	0.002 (0.014)	0.004+ (0.002)	0.007 (0.005)	0.005 (0.008)	−0.160** (0.054)	0.041 (0.061)	0.096 (0.105)
Age	−0.008** (0.003)			−0.004* (0.002)			0.000 (0.003)			−0.003* (0.001)			−0.096*** (0.029)		
Male = 1	0.159*** (0.033)			0.034 (0.021)			0.197*** (0.029)			0.043** (0.015)			1.336*** (0.308)		
R-squared	0.073	0.005		0.025	0.000		0.042	0.000		0.021	0.003		0.038	0.001	

Standard errors are clustered by twinship. Linear probability models were estimated for dichotomous outcomes.
***p < 0.001, **p < 0.01, *p < 0.05, + p < 0.1.

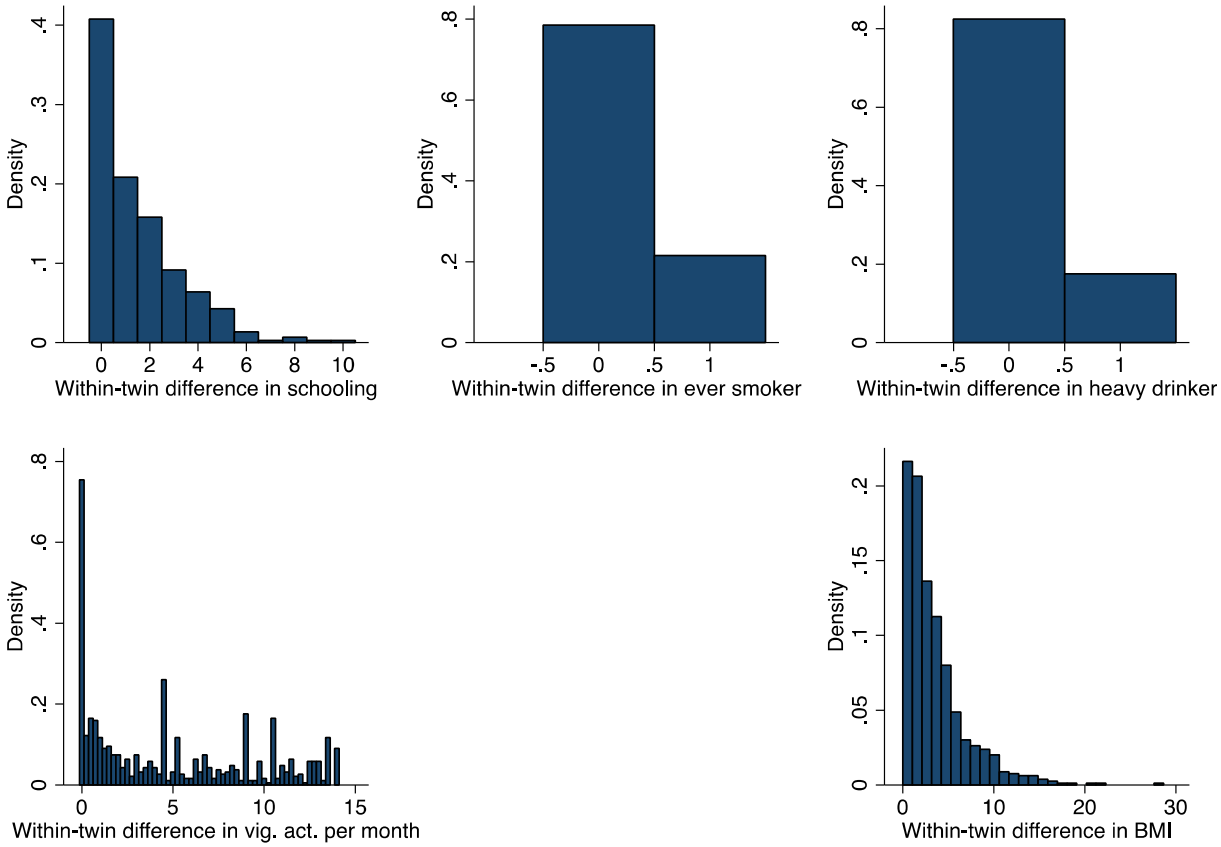
Appendix Table 4
Means and percentages for main variables for included and missing samples.

	Midus Wave 1			Midus Wave 2			MTR		
	In sample	Dropped	P-val	In sample	Dropped	P-val	In sample	Dropped	P-val
Age	45.0	43.5	0.038	52.9	54.7	0.009	47.1	47.3	0.360
Male	42.9%	44.1%	0.691	39.7%	45.4%	0.060	34.9%	40.5%	0.001
Years of schooling	13.7	13.6	0.726	14.3	14.2	0.704	13.7	13.4	0.003
Ever smoker	47.8%	46.1%	0.558	39.9%	45.5%	0.061	41.1%	45.0%	0.022
Heavy smoker	16.2%	23.8%	0.001	9.5%	12.9%	0.077	12.7%	14.3%	0.171
Ever drinker	84.9%	80.8%	0.065	57.6%	53.1%	0.137	70.6%	69.4%	0.432
Heavy drinker	29.2%	28.6%	0.853	6.6%	6.6%	0.970	5.9%	6.9%	0.245
BMI	26.2	40.6	0.000	27.4	35.2	0.000	25.8	26.0	0.274

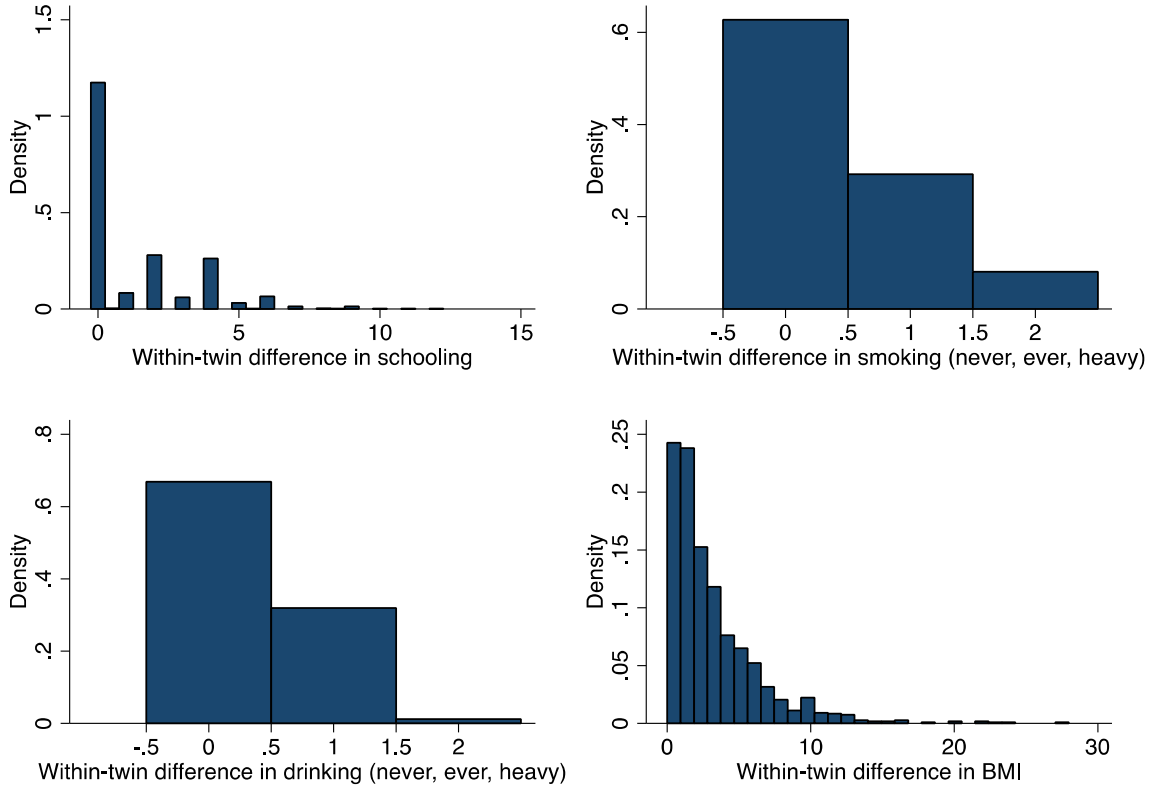
Notes: The Add Health data were not included due to the small amount of missingness (5.6%).



Appendix Fig. 1. Within-MZ twins difference in health-related behaviors, Add Health Twins, N = 373 twin pairs.



Appendix Fig. 2. Within-MZ twins difference in health-related behaviors, MIDUS Twins, N = 753 twin pairs.



Appendix Fig. 3. Within-MZ twins difference in health-related behaviors, MTR Twins, N = 1153 twin pairs.

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