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Limited Common Origins of Multiple Adult Health-Related Behaviors: Evidence from U.S. Twins

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Keywords
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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 the empirical evidence on the underlying causes of the vast within-population variation in health-related behaviors is mixed. While many potential causes of behaviors have been identified—such as schooling, genetics, and environments—little is known on how much of the variation across multiple health-related 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. Based on the results of both within identical twin regressions and multivariate behavioral genetic models, we find that aside from smoking and drinking, most behaviors are not strongly correlated among individuals. 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, mostly childhood, environments–suggesting 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.

Keywords: health-related behaviors; twins; smoking; alcohol consumption; schooling; genes.

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 related to other European countries (Preston et al., 2011; Rehm et al., 2007).

An important question for understanding how health behaviors determine 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 in fact causal, or if schooling is determined by unobserved characteristics that also determine health behaviors. The findings from these studies suggest that while schooling is associated with better health behaviors, schooling may not be a cause of health-related behaviors (Amin et al., 2015; Behrman et al., 2011).

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; 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, they have not sought to determine if variation across multiple behaviors is due to a common set of genetic endowments or childhood environments.

In this paper we use data on U.S. twins to investigate the degree to which multiple health 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 and waist circumference – 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.

2 Background

Health-related behaviors are not just limited to smoking, drinking, and physical activity but can encompass "any action, or deliberate inaction, by an individual that affects [their] own health" (Cawley and Ruhm, 2011). 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 health that affect 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 just 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 morely 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 behaviors. They find that around 30% of the educational gradient in health 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 behaviors, the study design was limited by an inability to identify whether the education health relationship is causal. A more recent literature using data on identical twins has tried to determine if these associations are in fact causal, or if schooling is determined by unobserved characteristics that also determine health 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 that bias estimates of the education health relationship in conventional analyses (Kohler

et al., 2011). By using within-MZ twin estimates, one can purge the cross-sectional associations between schooling and health of bias from these unobserved factors. The findings from these studies suggest that while schooling is associated with better health behaviors, schooling may not be a cause of variation in health behaviors (Amin et al., 2015; Behrman et al., 2011). Similarly, Cutler and Glaeser try to empirically confirm 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 among the health 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 genetic perspective. Under this paradigm, health-related behaviors are additively determined by genetic endowments, common (shared by sibling) environments, and individual idiosyncratic environments. Many behavior 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 twin pairs reports that smoking initiation has a heritability of 44%–implying that, subject to the assumptions of the behavioral genetic 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 to 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 role of genetics may also vary with social and environmental changes. 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 a more correlated with health-related behaviors as the populations of individuals that engage in those behaviors becomes more select.

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 found that being physically active in childhood was 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).

Research in multiple disciplines has identified many potential causes of health-related behaviors in adulthood. While schooling, genetics, and environments have been shown to be related to various health behaviors individually, the extent to which these factors determine multiple behaviors remains an open question. To address this gap in the literature, we use three datasets on U.S. twins to investigate the degree to which multiple health behaviors can be explained by an underlying common set of determinants. 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 (McGinnis and Foege, 1993; Mokdad et al., 2004), 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.

3 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).

3.1 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 twin zygocity across the three datasets. Zygocity in the Add Health data was initially self reported by the twins but was later confirmed by DNA testing. In the MIDUS data, twin pairs were given a separate survey and asked to self report their zygocity as either monozygotic or dizygotic. Finally, the zygocity 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, dropping opposite-sex DZ twins.

3.2 Schooling

For all three datasets individuals categorically reported their highest level of completed schooling. Based on these responses, we created continuous indicators 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 to 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).

3.3 Health-related behaviors

While Add Health asked a number of questions on smoking and drinking, we selected measures of "peak" smoking and drinking to be consistent with the other datasets. For smoking, we created a continuous variable of the number of cigarettes that an individual reported smoking per day during the heaviest period of smoking in their life. Similarly, drinking was measured as the number of drinks per day than an individual reported drinking during their heaviest year of drinking—we preferred drinks per day rather than the number of days an individual drank any alcohol per week, since this measure may better capture binge drinking patterns, which have been show to be related to poor health (Viner and Taylor, 2007). 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. Due to the difficulty in measuring diet, we proxied the combined effects of diet and physical activity as unhealthy weight–measured by waist circumference. We preferred waist circumference to BMI since studies have found that it is more closely related to unhealthy weight and the risk of mortality and cardiovascular disease (Yusuf et al., 2005).

MIDUS asked individuals about their smoking and drinking habits for their heaviest years of use. Based on these questions we created a continuous variable for the number of cigarettes smoked per day during the heaviest year of smoking and the average number of drinks consumed per day during the heaviest year of drinking. For physical activity, 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 included measured waist circumference.

The MTR asked individuals to report the number of cigarettes the smoked per day when they were 25, 30, 35, 40, and 45 years old. Based on these answers we created a peak smoking measure that was simply the max cigarettes per day smoked across these age groups. Unfortunately, the MTR did not ask about drinks per day, rather they asked the number of days an individual drank per week for each of the age groups listed above. We created a peak drinking measure that was the maximum number of days per week an individual drank across all age groups. The MTR also did not have measurements of waist circumference so we used a continuous measure of BMI to capture variation in unhealthy weight.

3.4 Missing values and sample size

For Add Health, the total wave 4 twin sample consisted of 531 complete MZ or same-sex DZ twin pairs. 153 twin pairs (29%) were dropped for missing information for one or both members of the twinship for a final sample of 378 twin pairs (207 MZ twin pairs and 171 DZ twin pairs). The total MIDUS twin sample for waves 1 and 2 pooled consisted of 1085 complete twin pairs. 348 twin pairs (32%) were dropped for missing information on the key covariates for one or both members of the twinship for a final sample size of 737 twin pairs (407 MZ twin pairs and 330 same-sex DZ twin pairs). Finally, the MTR was had an initial twin sample of 1,399 complete twin pairs. 227 twin pairs (16%) were dropped for missing information on the key covariates for a final sample of 1,172 twin pairs (657 MZ twin pairs and 515 same-sex DZ twin pairs).

4 Methods

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

4.1 Within-MZ Twin 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 characterstics (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 twin 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. The plausibility of these estimates depends on the size of the within-twin differences in both schooling and each outcome; in Appendix figures 1-3 we graph the within-twin distributions and find a wide range of differences across twin pairs. For example, for a health-related behavior y_i for individual i, the regression of y_i on schooling would be:

$$y_i = \beta_0 + \beta_1 \,\text{schooling}_i + \beta_2 \,\text{age}_i + \beta_3 \,\text{male}_i + \beta_4 \; + \gamma z_i + \epsilon_i \tag{1}$$

where z_i are the unobserved parental, family, genetic, and child environmental characteristics discussed above. The β_1 is the association between schooling and behavior y, but it is not be the causal effect, since both schooling and behavior y are affected by z. By comparing the within-MZ twin difference in both

schooling and health behaviors, we can instead estimate the following regression for twinship j:

$$(y_{1j} - y_{2j}) = \beta_1(\text{schooling}_{1j} - \text{schooling}_{2j}) + \gamma(z_{1j} - z_{2j}) + (\epsilon_{1j} - \epsilon_{2j}) \quad (2)$$

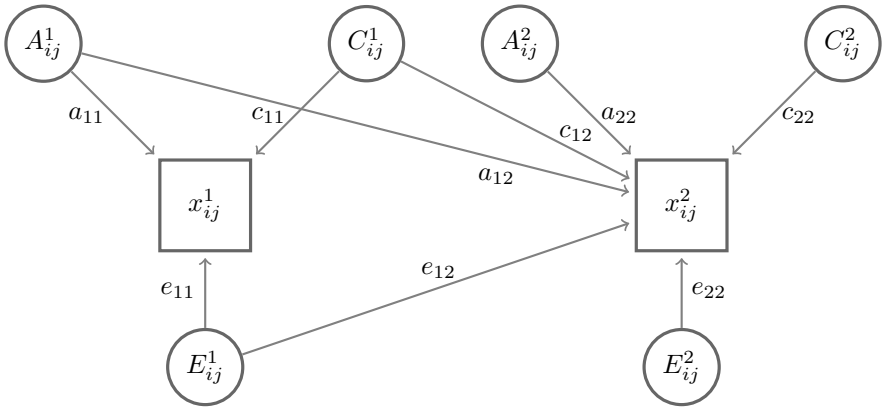
Since MZ twins have identical genes at birth, parental and family backgrounds, and childhood environments, $z_{1j} - z_{2j}$ cancels out, removing the confounding from these unobserved factors.

These models have a few potential problems. First, we have to assume that the source of the within-MZ twin 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 twin regression, biasing the estimated effect towards zero (Bound and Solon, 1999). 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 therfore estimated regression of the form (2) for each of the health-related behaviors (we first converted each health-related behavior to z-scores to make the estimated schooling effect comparable across all the behaviors).

4.2 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 enviornmental 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 Figure 1 (figure is shown for only two health-related behaviors for clarity, but this approach generalizes to any number of health-related behaviors). Here, x_{ij}^1 through x_{ij}^4 are four observed behaviors for individual i in twin pair j 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

Figure 1: Path diagrams for the multivariate ACE model



a_{11}, c_{11}, e_{11}, a_{22}, c_{22}, and e_{22}) and the A, C, E factors of the other behaviors (paths a_{12}, c_{12}, and e_{12}).

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 simple correlation matrices of all the behaviors (estimated in the beginning of the analysis) as a function of all 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 (1992).

We determine the role of a common set of genetic, shared environment, and individual environmental factors by using the model presented in Figure 1 to first estimate the observed correlation matrices as a function of all the path coefficients.[1], then split the correlation matrices into the correlation due to genetic factors, the correlations due to shared environments, and the correlations due to individual environments. Large coerrelation coefficients for these factor-specific matrices would therefore imply that a common set of factors are influencing multiple behaviors.

[1]The observed and estimated correlation matrices are slightly different since the behavioral genetics models estimates the correlations under the constraints that the correlations among MZ twin 1, MZ twin 2, DZ twin 1, and DZ twin 2 individuals should be identical Since twin number was randomly assigned, there is no systematic relationship between twin number and the correlations. Similarly, there is no reason to suspect that correlation between behaviors within individuals is different depending on whether the individual belongs to an MZ or DZ twinship. However, in practice random sample error results in slightly different correlation matrices. Since the implied correlation matrices were estimated with the a, c, and e paraemeters that provided the best fit under the identical correlation constraint, the implied matrix is slightly different from the observed matrices, which was estimated pooling all individuals regardless of their twin number or zygocity.

5 Results

Table 1 presents a descriptive overview of the three twin samples. The MIDUS and MTR samples are on average middle aged (47.1 years old for MTR and 47.6 for MIDUS) while the individuals in the Add Health are slightly younger (28.9 years). All three datasets have a greater share of women compared to men–this difference is especially pronounced for the Minnesota Dataset (64.9% female). Most of our analyses focus specifically on differences within twin pairs and would not be biased by the sex composition of the samples. Across all four of the identified health-related behaviors, we observe a common pattern: the average levels of each behavior are fairly high but there is also substantial variation in the behavior. For example, the average number of cigarettes smoked per day is around 6 in the Add Health, 10 in the MIDUS, and 11 in the Minnesota datasets. Yet, the standard deviation in each sample is larger than the mean, implying a wide distribution in smoking behavior. We observe a similar pattern for drinking, and physical activity. Based on the standard Centers for Disease Control and Prevention cutoffs for BMI and waist circumference, the samples are on average slightly overweight.

Figures 1, 2, and 3 graph the correlation matrix of the selected health behaviors for all three datasets (all variables are in z-scores). 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 datasets, the most striking initial result is the lack of correlation among many of the behaviors. For example, smoking and physical activity has a correlation of -0.023 in the Add Health data and a correlation of -0.084 in the MIDUS data–implying that individuals that smoke are only very slightly less likely to engage in physical activity. Similarly, the correlation of drinking and unhealthy weight is 0.0056 in the Add Health data, 0.025 in the MIDUS data and -0.019 in the MTR data. 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 behaviors since the behaviors themselves do not correlate highly. This general lack of correlation between the health behaviors is consistent for almost every pairwise comparison except for one: smoking and drinking. Despite different birth cohorts and measurements of drinking and smoking, we find a remarkably similar correlation between smoking and drinking across all three datasets (0.33 in the Add Health, 0.33 in the MIDUS, and 0.27 in the MTR). In the following section, we investigate the role of schooling, genetics, and the childhood and adolescent environment to the covariation between health behaviors, paying special attention to smoking and drinking.

In Tables, 2-4, we show the results from the OLS and within-twin fixed effect regressions of each health-related behavior on years of schooling (we standardized all variables to z-scores to make the magnitude of the schooling effect comparable across behaviors). Focusing on just the OLS regressions, we find the commonly

reported conclusion of an association between schooling and better health behaviors. In the Add Health dataset, a one year increase in schooling is associated with a decrease in cigarettes smoked per day, an increase in the times an individual engages in vigorous activity per week, and a lower waist circumference. This pattern of association between schooling and health behaviors is largely similar in the other two datasets as well: in the MIDUS dataset schooling is associated with less smoking, fewer drinks per day, more vigorous activity per week, and a lower waist circumference. 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 twin regressions, providing a more robust evaluation of the schooling-health behavior relationship.[2] 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-twin estimates are substantially smaller in magnitude and most lost statistical significance. For example, the relationship between schooling and smoking moved from -0.087 to -0.036 in the Add Health data, from -0.073 to -0.020 in the MIDUS data, and from -0.086 to -0.051 in the Minnesota data (for this last dataset the within-MZ effect is still significant). In the Add Health and Minnesota datasets the coefficient for the unhealthy weight outcomes moved from -0.058 to 0.012 in the Add Health and -0.035 to 0.007 in the Minnesota twins. Not every relationship diminished or lost statistical significance. In the MIDUS dataset, the OLS and within-MZ coefficients are comparable for vigorous activity per month (0.033 vs 0.032, the within-MZ is not significant however) and waist circumference (-0.042 vs -0.046), suggesting that schooling may be related to diet and physical activity behavior; however, this conclusion is not observed for Add Health or Minnesota datasets, where the MZ estimates no longer indicate a significant relation between schooling and physical activity or waist circumference.

While the above results from the schooling regressions (Tables 2-4) suggest that schooling may be related to some health behaviors, we find almost no support for the hypothesis that schooling affects all four of the behaviors examined. Focusing specifically on smoking and drinking, tthe two most correlated health behaviors, we find that the schooling effect is much larger in magnitude for smoking than for drinking in two of the three datasets (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 health 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 genetic model, and the genetic, shared environment, and individual environment

[2]For the AddHealth and MIDUS datasets 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.

specific correlation matrices. These second two matrices estimate the correlation between the behaviors that arise from a common set of genes or a shared environments. The diagonals of the genetic, environmental, and individual matrices represent the fraction of variance in each specific behavior that is consistent with genetic endowments and environmental factors.

Across all three datasets, 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 26% of the variance among the Add Health twins, 41% among the MIDUS twins, and 17% of the variance among the MTR twins. Similarly, genetic endowments are consistent with a large fraction of the variance in unhealthy weight: 58% in Add Health, 62% in MIDUS, and 67% in MTR. The role of the shared, mostly childhood, environment is less pronounced across the datasets. The shared, environment is consistent with 17% of the variance in cigarettes per day and 34% of drinks per day for the Add Health sample. We observe a relatively similar pattern in the MTR data, with 25% of the variance in smoking and 23% of the variance in drinking consistent with shared environmental factors. For the MIDUS twins, the shared environment is related to 19% of the variance in smoking but only 11% of the variance in drinking. While the magnitudes vary across the datasets, the patterns 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 datasets and all behaviors, most 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, the majority 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 the Add Health and Minnesota twins, we find that a large portion of this correlation is consistent with a common environmental factor (environmental correlation is 0.17 for the Add Health and 0.14 in the Minnesota–the shared environment correlation between smoking and drinking is small in the MIDUS data: r=0.08).

For the other pairwise comparisons, the role of a common set of genetic endowments and environments is inconsistent across the three datasets. For example, we find that a common set of genetics is consistent with the covariation in smoking and drinking among the MIDUS twins (r=0.20), but this correlation is not present in the Add Health or MTR datasets. We also find a moderate genetic correlation between cigarette smoking and BMI in the MTR dataset (r=0.17) 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.

5.1 Robustness

We conducted a number of robustness checks. First, we re-estimated all the models with dichotomized versions of our main variables (pack per day smoker, heavy drinker, physically inactive, unhealthy waist circumference (for the Add Health and MIDUS datasets), and obese (for the MTR dataset).[3] Our main conclusions were unchanged, although, as expected, the magnitude of the correlations and estimated coefficients were slighty diminished since information is lost from dichtomizing the variables. 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.[4] We find that measurement error in the MTR dataset does not affect our conclusions, with the coefficient actually becoming smaller for some outcomes.

Our conclusions were also unchanged under many alternative classifications of our main variables. We found no changes when we when we defined smoking as the number of days per month an individual smoked rather than the number of cigarettes per day; similarly, our conclusions were consistent when measuring drinking as the number of times per month an individual drank rather than the number of drinks per sitting. While we preferred the use of waist circumference to BMI (as mentioned previously, studies have found that it is more closely related to the risk of mortality than BMI (Yusuf et al., 2005), our conclusions were mostly consistent when using BMI in the Add Health and MIDUS datasets–the main exception is that we found a much larger component of genetic contributions to BMI than we did waist circumference. This is not cause for worry, however, since this is likely due by the strongly heritable height component of BMI (studies consistently estimate that the hertiability of height is between 80-90% (Silventoinen et al., 2003). Our results were also consistent when looking at just moderate physical activity and a measure that combined both moderate and vigorous physical activity.

6 Discussion

Health-related behaviors are significant contributors to morbidity and mortality in the United States, yet the empirical evidence on the underlying causes of the vast within-population variation in health-related behaviors is mixed. While many potential causes of behaviors have been identified—such as schooling, genetics, and environments—little is known on how much of the variation across multiple health-related behaviors is due to a common set of causes. Using three data sources on American twins, we do not find evidence that schooling, or a common set of genetic endowments or environments are a common cause of

[3] Appendix figures 4-6

[4] Appendix tables 1-3

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 datasets, the key health 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 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 behaviors is the relationship between smoking and drinking (drinks per sitting or day): across all three of the datasets, 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 surprising, they are consistent with prior studies that use within MZ-twin designs including (Kohler et al., 2011; Behrman et al., 2011; Fujiwara and Kawachi, 2009; Amin et al., 2015). These papers generally find that the cross-sectional associations between schooling and healthy 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 Roayer, 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-twin 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 figures 1-3 we show the distributions of within-twin differences in schooling and each of the behaviors–these graphs highlight the wide range of differences on which the within-MZ twin is estimated over).

Finally, based on the results of the behavioral genetic analyses, we find that the greatest portion of variance for each health behavior is related to behavior specific factors, suggesting that the causes of health behaviors are largely idiosyncratic. We also find that genetic endowments are consistent with significant portions of the variance in most of the health behaviors. These two results have been found in other behavioral genetic studies on the heritability of individual behaviors (Bauman et al., 2012; Vink et al., 2005; Walters, 2002; Min et al., 2013)–these studies find small contributions from environments, reasonably large genetic contributions, and large idiosyncratic error 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 moderate 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-twin difference in schooling was unrelated to the within-twin difference in health 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 genetic models and is not needed for the within-MZ twin estimates. After controlling for any unobserved difference between twins through the within-twin 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. Twin 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, twin 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, it has been 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-twin estimates is due to magnifying of measurement error when comparing within-twins (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 dataset suggest that measurement error is not driving our results (Appendix table 7).

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 anal-

yses 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 behaviors should consider each behavior uniquely to try and identifying causes of poor health-related behaviors. 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.

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Figures and tables

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

	Add Health Twins N = 756		MIDUS Twins N = 1,474		MTR Twins N = 2,344	
	Mean or n	SD or %	Mean or n	SD or %	Mean or n	SD or %
Age	28.92	1.62	47.85	12.39	47.07	5.62
Sex						
Male	370	48.94	634	43.01	822	35.07
Female	386	51.06	840	56.99	1,522	64.93
Zygocity						
MZ	414	54.76	814	55.22	1,314	56.06
DZ	342	45.24	660	44.78	1,030	43.94
Cigarettes per day	6.09	9.25	10.08	14.86	10.82	15.56
Drinks per sitting	3.47	3.56	2.34	2.77		
Days drink per week					1.84	2.11
Vigorous activity per month			6.41	5.39		
Vigorous activity per week	2.84	3.58				
Waist Circumference (in)	38.05	7.38	35.38	5.61		
BMI					25.84	4.66

Notes: Data are shown for the total number of people (number of twin pairs is the sample size divided by 2). All three datasets did not have consistent questions on drinking, physical activity, and unhealthy weight so different measures are shown across datasets

Figure 2: Correlation matrix and scatter plots for the selected health behaviors, Add Health Twins, N = 756. All variables are shown as z-scores.

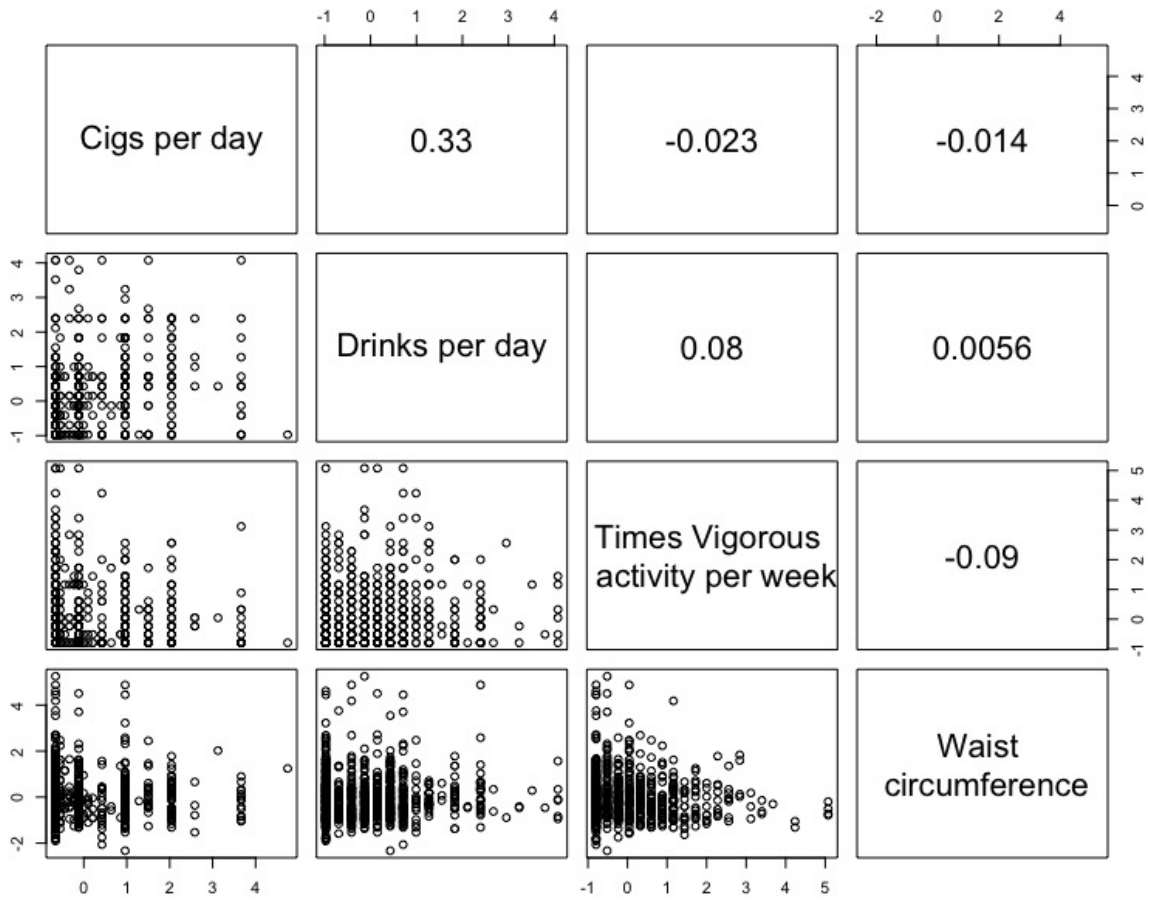


Figure 3: Correlation matrix and scatter plots for the selected health behaviors, MIDUS Twins, N = 1,474. All variables are shown as z-scores.

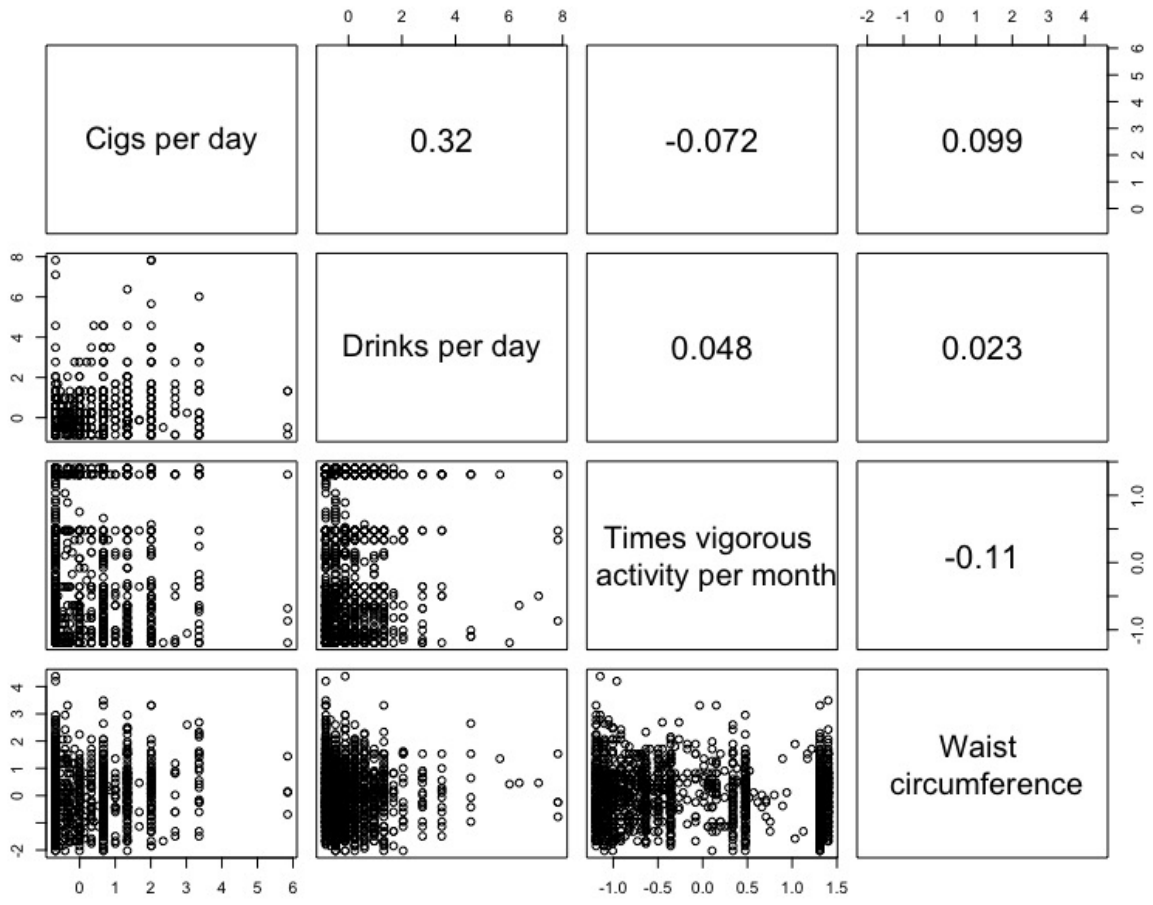


Figure 4: Correlation matrix and scatter plots for the selected health behaviors, MTR Twins, N = 2,344. All variables are shown as z-scores.

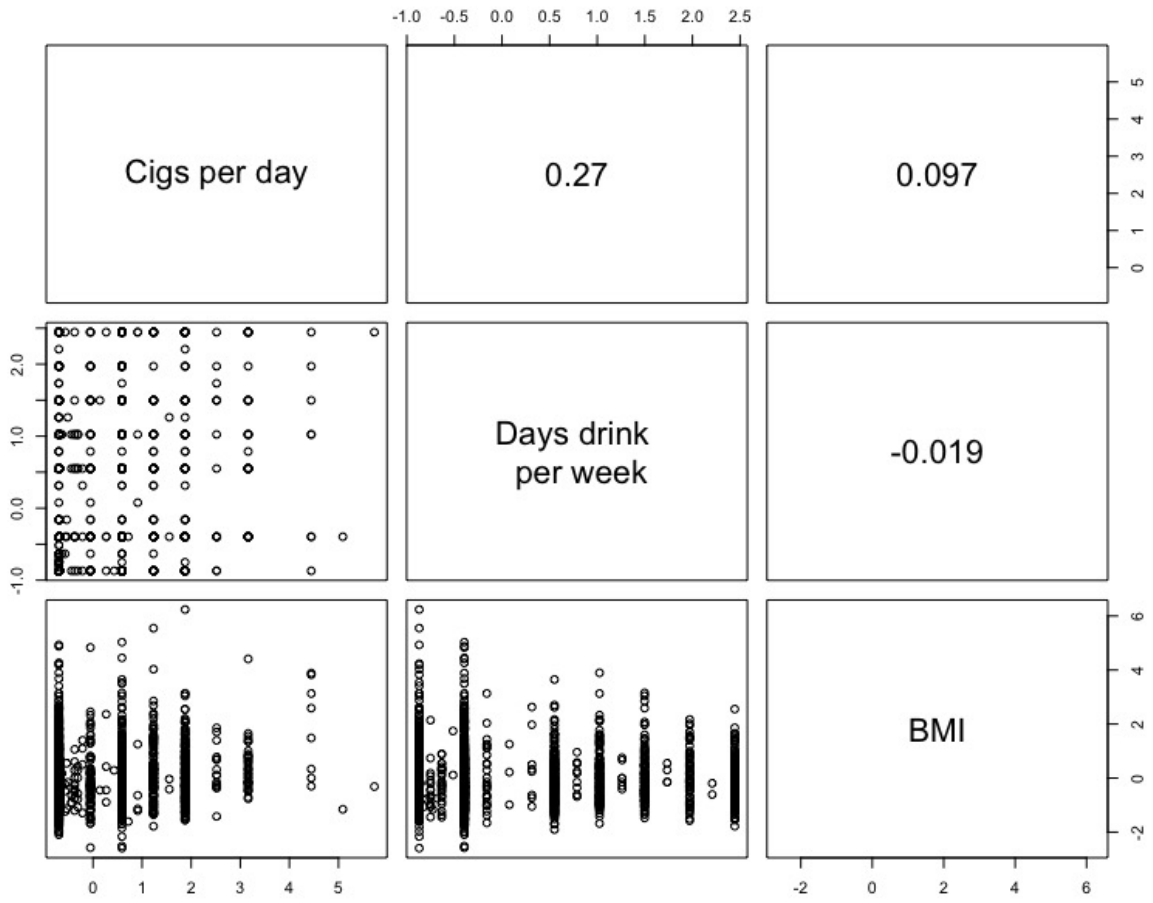


Table 2: Estimated OLS and within-twin regressions of smoking, drinking, physical activity, and unhealthy weight on schooling, Add Health Twins, N = 414

	Cigs per day		Drinks per day		Vig act per week		Waist circumference	
VARIABLES	OLS	FE	OLS	FE	OLS	FE	OLS	FE
Years of schooling	-0.087***	-0.036	-0.004	0.038	0.068**	0.029	-0.058**	0.012
	(0.017)	(0.027)	(0.020)	(0.040)	(0.025)	(0.044)	(0.022)	(0.019)
Age	0.094**	0.062	0.043	-0.170	0.018	0.505	0.008	-0.052
	(0.033)	(0.270)	(0.035)	(0.310)	(0.031)	(0.317)	(0.038)	(0.399)
Male	0.143		0.447***		0.216*		0.266*	
	(0.109)		(0.108)		(0.107)		(0.122)	
R-squared	0.077	0.006	0.058	0.005	0.046	0.013	0.037	0.001

Standard errors are clustered by twinship

*** p<0.001, ** p<0.01, * p<0.05, + p<0.1

Table 3: Estimated OLS and within-twin regressions of smoking, drinking, physical activity, and unhealthy weight on schooling, MIDUS Twins, N = 814

	Cigs per day		Drinks per day		Vig act per month		Waist circumference	
VARIABLES	OLS	FE	OLS	FE	OLS	FE	OLS	FE
Years of schooling	-0.077***	-0.014	-0.041**	0.000	0.039**	0.034	-0.035*	-0.047**
	(0.015)	(0.024)	(0.014)	(0.015)	(0.015)	(0.027)	(0.015)	(0.016)
Age	0.009*	0.052	-0.019***	0.018	-0.016***	-0.222	0.017***	-0.009
	(0.003)	(0.137)	(0.003)	(0.109)	(0.003)	(0.162)	(0.003)	(0.124)
Male	0.376***		0.527***		0.207**		0.716***	
	(0.081)		(0.077)		(0.075)		(0.076)	
R-squared	0.092	0.001	0.139	0.000	0.063	0.007	0.196	0.016

Standard errors are clustered by twinship

*** p<0.001, ** p<0.01, * p<0.05, + p<0.1

Table 4: Estimated OLS and within-twin regressions of smoking, drinking, and unhealthy weight on schooling, MTR Twins, N = 1,314

	Cigs per day		Days drink per week		BMI	
VARIABLES	OLS	FE	OLS	FE	OLS	FE
Years of schooling	-0.086***	-0.051*	-0.004	-0.008	-0.035**	0.007
	(0.010)	(0.025)	(0.012)	(0.018)	(0.011)	(0.013)
Age	-0.014*		-0.008		-0.021***	
	(0.006)		(0.006)		(0.006)	
Male	0.384***		0.654***		0.291***	
	(0.069)		(0.073)		(0.065)	
R-squared	0.085	0.011	0.103	0.000	0.041	0.000

Standard errors are clustered by twinship

*** p<0.001, ** p<0.01, * p<0.05, + p<0.1

Table 5: Implied correlation matrices from the multivariate ACE models, Add Health Twins, N = 756

	Total Correlation Matrix					Genetic Correlation			
	Cigs per day	Drinks per day	Vig act per week	Waist circumference		Cigs per day	Drinks per day	Vig act per week	Waist circumference
Cigs per day	1.00				Cigs per day	0.26			
Drinks per sitting	0.34	1.00			Drinks per sitting	0.05	0.02		
Vigorous activity per week	-0.02	0.08	1.00		Vigorous activity per week	-0.02	-0.02	0.25	
Waist Circumference	-0.02	0.00	-0.09	1.00	Waist Circumference	0.03	-0.07	-0.10	0.58

	Shared Environmental Correlation					Individual Error Correlation			
	Cig per day	Drinks per day	Vig act per week	Waist circumference		Cigs per day	Drinks per day	Vig act per week	Waist Circumference
Cigs per day	0.17				Cigs per day	0.56			
Drinks per sitting	0.19	0.34			Drinks per sitting	0.10	0.63		
Vigorous activity per week	0.00	0.07	0.04		Vigorous activity per week	0.00	0.03	0.70	
Waist Circumference	-0.04	0.03	0.04	0.08	Waist Circumference	-0.01	0.04	-0.03	0.33

Table 6: Implied correlation matrices from the multivariate ACE models, MIDUS Twins, N = 1,474

	Total Correlation Matrix					Genetic Correlation			
	Cigs per day	Drinks per day	Vig act per week	Waist circumference		Cigs per day	Drinks per day	Vig act per week	Waist circumference
Cigs per day	1.00				Cigs per day	0.41			
Drinks per day	0.31	1.00			Drinks per day	0.20	0.44		
Vigorous activity per month	-0.07	0.05	1.00		Vigorous activity per month	-0.03	0.05	0.13	
Waist circumference	0.10	0.02	-0.10	1.00	Waist circumference	0.12	-0.07	-0.01	0.62

	Shared Environmental Correlation					Individual Error Correlation			
	Cig per day	Drinks per day	Vig act per week	Waist circumference		Cigs per day	Drinks per day	Vig act per week	Waist Circumference
Cigs per day	0.19				Cigs per day	0.40			
Drinks per day	0.08	0.11			Drinks per day	0.03	0.45		
Vigorous activity per month	-0.04	0.02	0.12		Vigorous activity per month	0.00	-0.02	0.75	
Waist circumference	-0.03	0.06	0.01	0.08	Waist circumference	0.01	0.03	-0.10	0.29

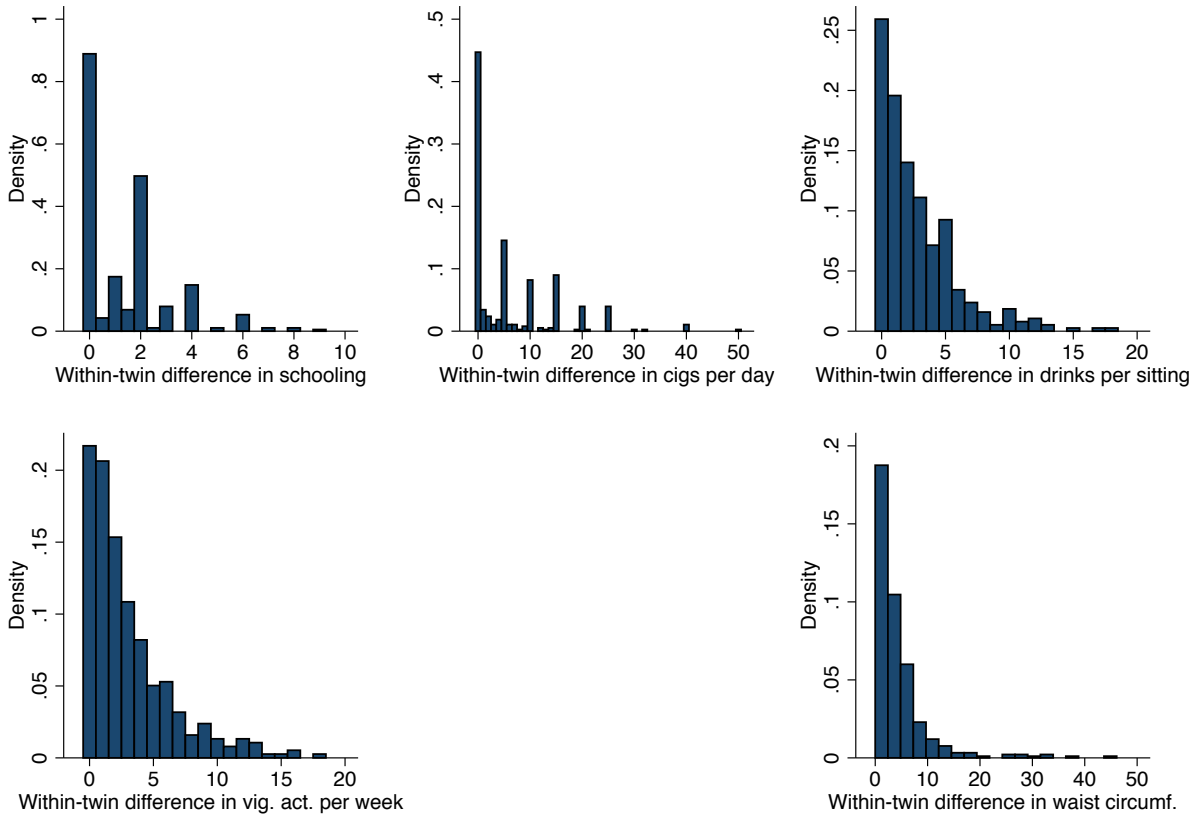
Table 7: Implied correlation matrices from the multivariate ACE models, MTR Twins, N = 2,344

	Total correlation matrix				Genetic correlation		
	Cigs per day	Days drink per week	BMI		Cigs per day	Days drink per week	BMI
Cigs per day	1.00			Cigs per day	0.17		
Days drink per week	0.26	1.00		Days drink per week	0.01	0.32	
BMI	0.10	-0.02	1.00	BMI	0.17	-0.01	0.67

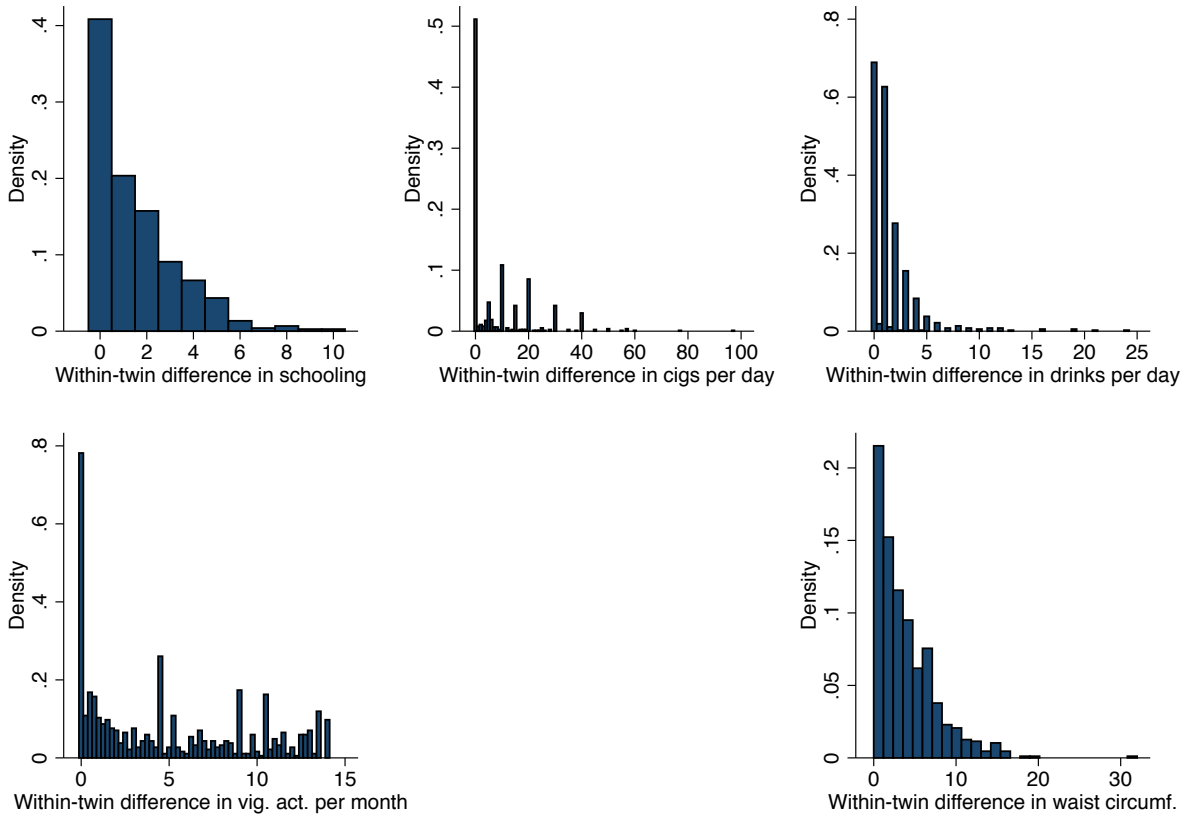
	Shared environmental correlation				Individual error correlation		
	Cigs per day	Days drink per week	BMI		Cigs per day	Days drink per week	BMI
Cigs per day	0.25			Cigs per day	0.58		
Days drink per week	0.16	0.23		Days drink per week	0.09	0.45	
BMI	-0.04	0.00	0.01	BMI	-0.03	-0.01	0.32

Appendix A: Within-twin distributions

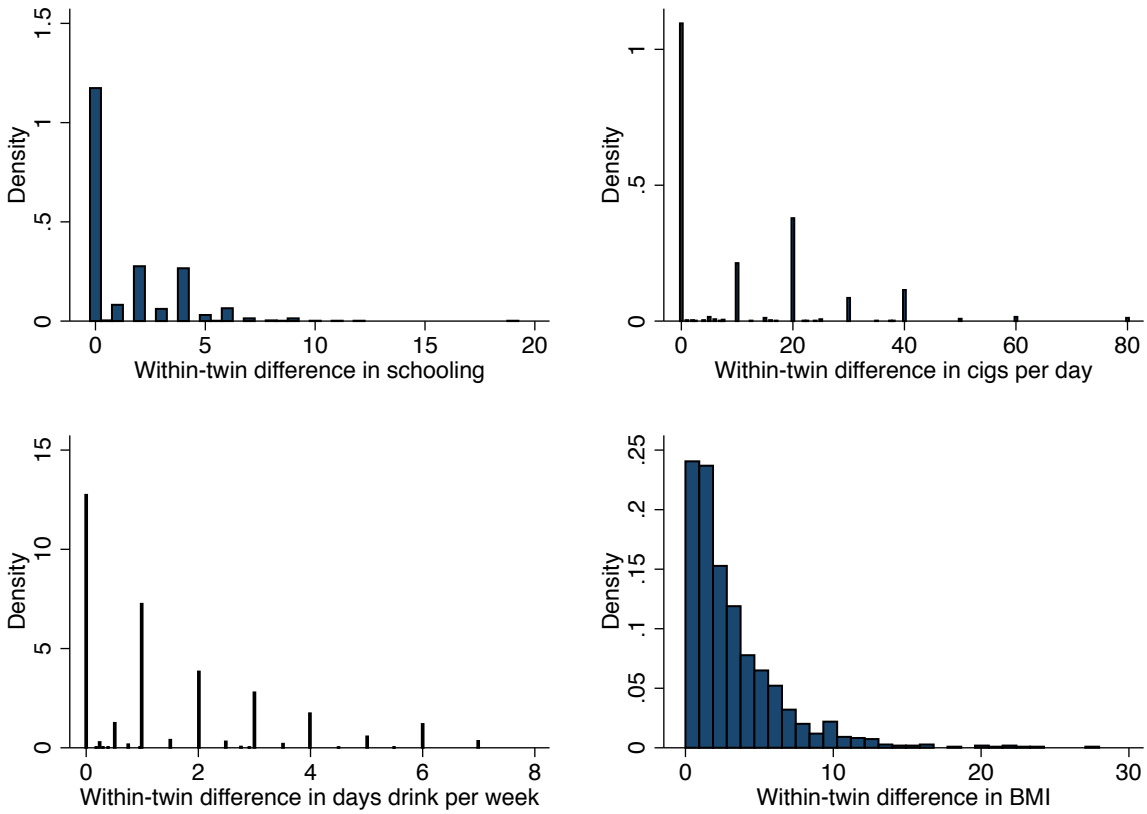
Appendix Figure 1: Within-MZ twin difference in health-related behaviors, Add Health Twins, N = 378 twin pairs



Appendix Figure 2: Within-MZ twin difference in health-related behaviors, MIDUS Twins, N = 737 twin pairs

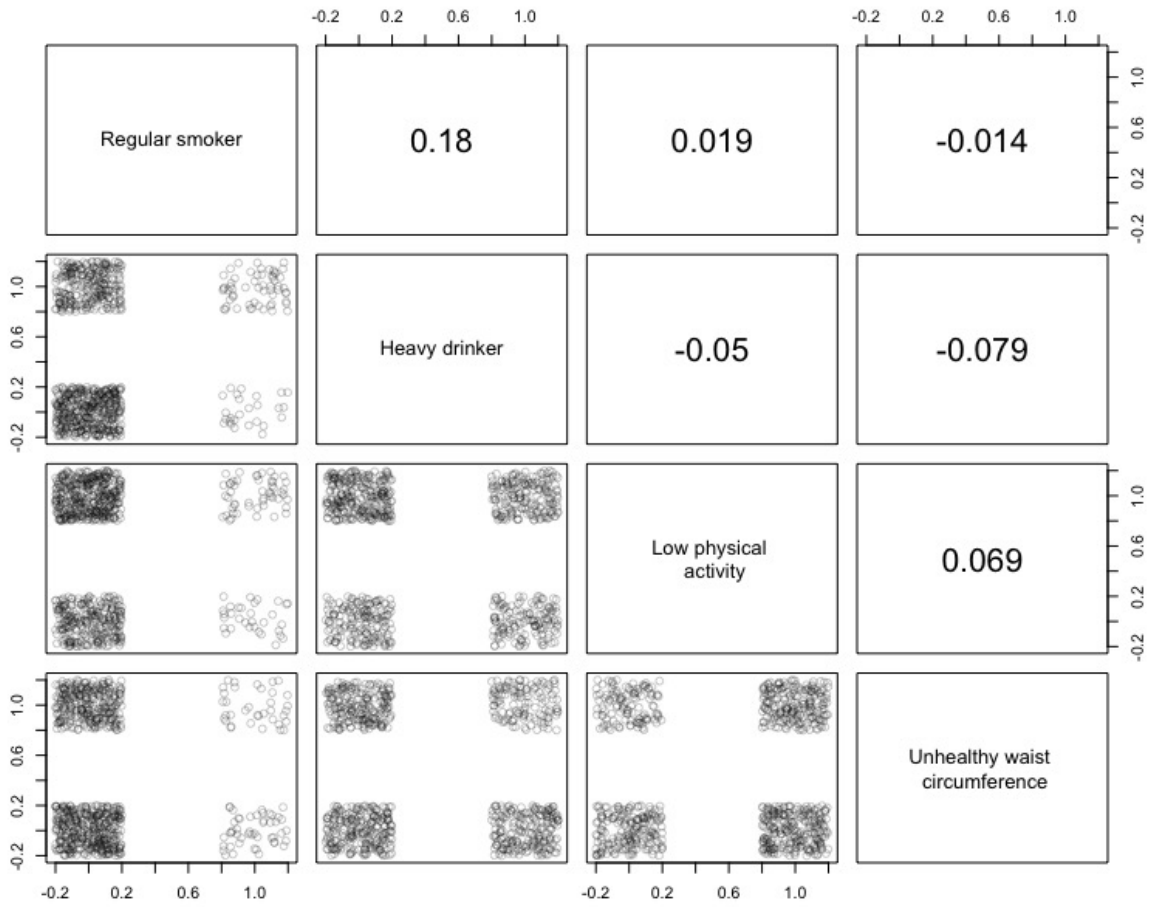


Appendix Figure 3: Within-MZ twin difference in health-related behaviors, MTR Twins, N = 1,712 twin pairs

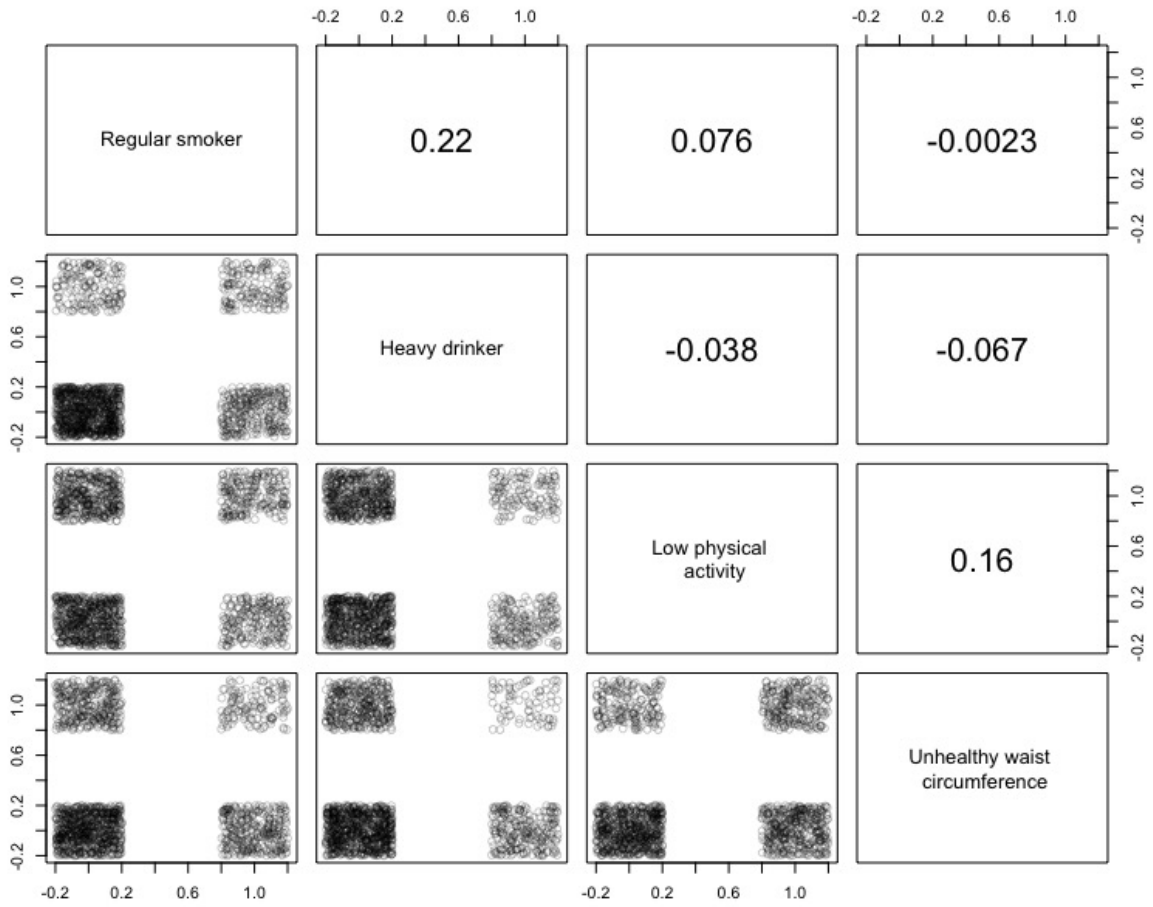


Appendix B: Dichtomous outcomes

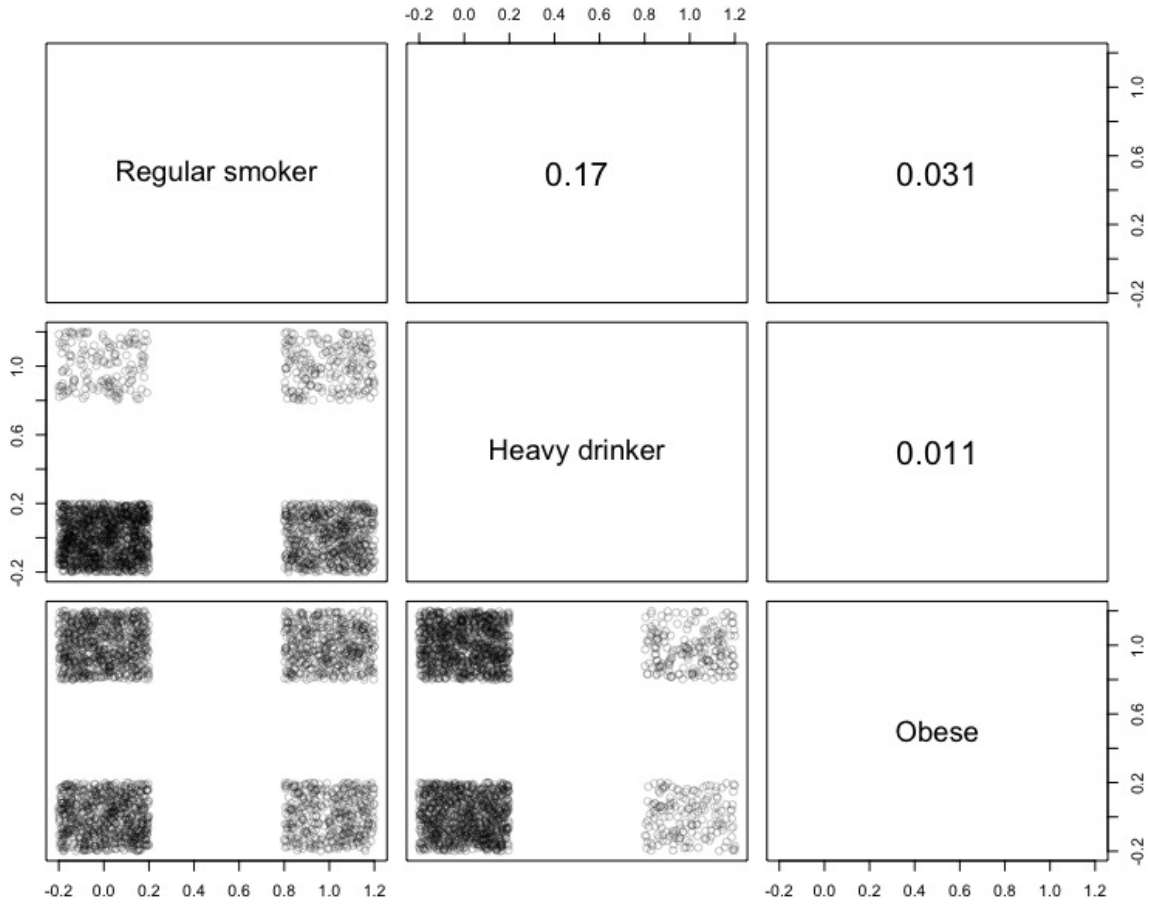
Appendix Figure 4: Correlation matrix and scatter plots for the selected health behaviors, Add Health Twins, N = 756. All variables are shown as z-scores.



Appendix Figure 5: Correlation matrix and scatter plots for the selected health behaviors, MIDUS Twins, N = 1,474. All variables are shown as z-scores.



Appendix Figure 6: Correlation matrix and scatter plots for the selected health behaviors, MTR Twins, N = 2,344. All variables are shown as z-scores.



Appendix Table 1: Estimated OLS and within-twin regressions of smoking, drinking, physical activity, and unhealthy weight on schooling, Add Health Twins, N = 756

	Cigs per day		Drinks per day		Vig act per week		Waist circumference	
VARIABLES	OLS	FE	OLS	FE	OLS	FE	OLS	FE
Years of schooling	-0.019**	0.003	-0.004	-0.001	-0.021*	-0.021	-0.030**	0.022
	(0.006)	(0.008)	(0.011)	(0.022)	(0.011)	(0.023)	(0.011)	(0.015)
Age	0.021*	-0.081	0.014	-0.193	0.004	-0.293+	0.011	-0.098
	(0.010)	(0.093)	(0.017)	(0.199)	(0.016)	(0.173)	(0.020)	(0.150)
Male	0.069*		0.151**		-0.103+		-0.156*	
	(0.034)		(0.056)		(0.053)		(0.062)	
R-squared	0.045	0.003	0.028	0.006	0.022	0.015	0.046	0.016

Standard errors are clustered by twinship

*** p<0.001, ** p<0.01, * p<0.05, + p<0.1

Appendix Table 2: Estimated OLS and within-twin regressions of smoking, drinking, physical activity, and unhealthy weight on schooling, MIDUS Twins, N = 1,474

	Cigs per day		Drinks per day		Vig act per month		Waist circumference	
VARIABLES	OLS	FE	OLS	FE	OLS	FE	OLS	FE
Years of schooling	-0.038***	-0.001	-0.020***	0.002	-0.013+	-0.016	-0.009	-0.021+
	(0.007)	(0.010)	(0.006)	(0.008)	(0.007)	(0.016)	(0.007)	(0.011)
Age	0.003*	-0.025	-0.007***	-0.073	0.005**	0.095	0.007***	0.094
	(0.002)	(0.074)	(0.001)	(0.064)	(0.002)	(0.092)	(0.002)	(0.069)
Male	0.150***		0.248***		0.010		-0.106**	
	(0.038)		(0.033)		(0.037)		(0.038)	
R-squared	0.079	0.000	0.141	0.004	0.022	0.005	0.048	0.014

Standard errors are clustered by twinship

*** p<0.001, ** p<0.01, * p<0.05, + p<0.1

Appendix Table 3: Estimated OLS and within-twin regressions of smoking, drinking, and unhealthy weight on schooling, MTR Twins, N = 2,344

	Cigs per day		Days drink per week		BMI	
VARIABLES	OLS	FE	OLS	FE	OLS	FE
Years of schooling	-0.041***	-0.018+	-0.004	-0.003	-0.013*	0.005
	(0.005)	(0.011)	(0.004)	(0.008)	(0.006)	(0.008)
Age	-0.008**		-0.002		-0.009**	
	(0.003)		(0.002)		(0.003)	
Male	0.171***		0.185***		0.239***	
	(0.031)		(0.026)		(0.034)	
R-squared	0.083	0.006	0.067	0.000	0.061	0.001

Standard errors are clustered by twinship

*** p<0.001, ** p<0.01, * p<0.05, + p<0.1

Appendix Table 4: Implied correlation matrices from the multivariate ACE models, Add Health Twins, N = 756

	Total Correlation Matrix					Genetic Correlation			
	Pack per day smoker	Heavy drinker	Inactive	Unhealthy waist cir.		Pack per day smoker	Heavy drinker	Inactive	Unhealthy waist cir.
Pack per day smoker	1.00				Pack per day smoker	0.06			
Heavy drinker	0.19	1.00			Consumes 3+ drinks per sitting	0.00	0.03		
Inactive	0.01	-0.05	1.00		Inactive	0.03	0.04	0.06	
Unhealthy waist cir.	0.00	-0.10	0.07	1.00	Unhealthy waist cir.	0.18	-0.01	0.08	0.56

	Shared Environmental Correlation					Individual Error Correlation			
	Pack per day smoker	Heavy drinker	Inactive	Unhealthy waist cir.		Pack per day smoker	Heavy drinker	Inactive	Unhealthy waist cir.
Pack per day smoker	0.19				Pack per day smoker	0.74			
Consumes 3+ drinks per sitting	0.14	0.31			Consumes 3+ drinks per sitting	0.05	0.65		
Inactive	0.01	-0.05	0.12		Inactive	-0.03	-0.04	0.81	
Unhealthy waist cir.	-0.15	-0.12	-0.01	0.13	Unhealthy waist cir.	-0.03	0.03	0.00	0.32

Appendix Table 5: Implied correlation matrices from the multivariate ACE models, MIDUS Twins, N = 1,474

	Total Correlation Matrix					Genetic Correlation			
	Pack per day smoker	Heavy drinker	Inactive	Unhealthy waist cir.		Pack per day smoker	Heavy drinker	Inactive	Unhealthy waist cir.
Pack per day smoker	1.00				Pack per day smoker	0.26			
Heavy drinker	0.22	1.00			Heavy drinker	0.10	0.36		
Inactive	0.09	-0.04	1.00		Inactive	0.10	0.09	0.05	
Unhealthy waist cir.	0.00	-0.07	0.17	1.00	Unhealthy waist cir.	0.12	-0.05	0.07	0.48

	Shared Environmental Correlation					Individual Error Correlation			
	Pack per day smoker	Heavy drinker	Inactive	Unhealthy waist cir.		Pack per day smoker	Heavy drinker	Inactive	Unhealthy waist cir.
Pack per day smoker	0.28				Pack per day smoker	0.46			
Heavy drinker	0.12	0.21			Consumes 3+ drinks per day	0.00	0.43		
Inactive	0.01	-0.11	0.12		Inactive	-0.02	-0.02	0.83	
Unhealthy waist cir.	-0.10	-0.05	0.01	0.04	Unhealthy waist cir.	-0.02	0.03	0.09	0.48

Appendix Table 6: Implied correlation matrices from the multivariate ACE models, MTR Twins, N = 2,344

	Total correlation matrix				Genetic correlation		
	Pack per day smoker	Heavy drinker	Obese		Pack per day smoker	Heavy drinker	Obese
Pack per day smoker	1.00			Pack per day smoker	0.15		
Heavy drinker	0.18	1.00		Heavy drinker	-0.09	0.09	
Obese	0.04	0.02	1.00	Obese	0.16	0.03	0.53

	Shared environmental correlation				Individual error correlation		
	Pack per day smoker	Heavy drinker	Obese		Pack per day smoker	Heavy drinker	Obese
Pack per day smoker	0.28			Pack per day smoker	0.57		
Heavy drinker	0.17	0.24		Heavy drinker	0.10	0.67	
Obese	-0.06	0.00	0.02	Obese	-0.06	-0.01	0.45

Appendix C: IV regression for measurement error

Appendix Table 7: Estimated OLS, within-twin, and within-twin IV regressions of smoking, drinking, physical activity, and unhealthy weight on schooling, MTR Twins, N = 1,314

	Cigs per day			Days drink per week			BMI		
VARIABLES	OLS	FE	FE IV	OLS	FE	FE IV	OLS	FE	FE IV
Years of schooling	-0.086***	-0.051*	-0.025	-0.004	-0.008	-0.010	-0.035**	0.007	0.018
	(0.010)	(0.025)	(0.031)	(0.012)	(0.018)	(0.028)	(0.011)	(0.013)	(0.022)
Age	-0.014*			-0.008			-0.021***		
	(0.006)			(0.006)			(0.006)		
Male	0.384***			0.654***			0.291***		
	(0.069)			(0.073)			(0.065)		

Standard errors are clustered by twinship

*** p<0.001, ** p<0.01, * p<0.05, + p<0.1