THE IMPACT OF POOR HEALTH ON EDUCATION: NEW EVIDENCE USING GENETIC MARKERS*

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Abstract

This paper examines the influence of health conditions on academic performance during adolescence. To account for the endogeneity of health outcomes and their interactions with risky behaviors we exploit natural variation within a set of genetic markers across individuals. We present strong evidence that these genetic markers serve as valid instruments with good statistical properties for ADHD, depression and obesity. They help to reveal a new dynamism from poor health to lower academic achievement with substantial heterogeneity in their impacts across genders. Our investigation further exposes the considerable challenges in identifying health impacts due to the prevalence of comorbid health conditions and endogenous health behaviors with clear implications for the health economics literature.

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1 Introduction

The discovery of the human genome, a sequence of approximately three billion chemical "letters" that make up human DNA, the recipe of human life, is considered a milestone in the history of science and medicine that might have the potential to influence social science research. Consider the following question that has been investigated in the psychology, education, economics, sociology and public health literatures: Does health status affect educational outcomes? While numerous studies report that students who are obese or depressed perform poorly relative to their classmates, factors other than health could be responsible for this repeatedly observed, but potentially spurious association. To credibly claim that obesity and depression have a deleterious effect on student performance in schools one must first overcome the inherent endogeneity when considering health and education. Further, accurate measures of health are difficult to obtain and overcoming biases arising from measurement error represents a second hurdle for applied researchers.

This study overcomes these challenges by considering an instrumental variables approach, where the instruments are selected based on a growing body of evidence in several neuroscientific fields that have identified genetic markers which possess significant associations with specific diseases and health behaviors. While there has long been scientific evidence suggesting that the association between genetic factors and health is substantial,[1] only recently has it been possible to collect measures of genetic markers. Since genetic markers are formed at conception, they are predetermined to any outcomes including those that occur during pregnancy and at birth. Genetic markers truly fit the definition of "nature". Using this "nature filter", the health variables being instrumented will be isolated from most nurture influences or choice-based inputs such as schools parents choose for their kids, neighborhood families select to reside in, peers kids choose to associate with, among other factors that threaten the identification of education production function parameters. When the variations in health variables that include clinical measures of depression, ADHD and obesity are due only to the differences in genetic coding, these variations are much less likely to be correlated with the environments surrounding an individual, allowing us to recover consistent estimates

of the impacts of a vector of health measures on academic performance.[2] While our identification strategy relies on scientific findings, the results suggest that further study of social environments might have to be invoked to understand the root of heterogeneous impacts of health on academic performance, which seems to place the question squarely back under the realm of social sciences.

Specifically, our empirical identification strategy is based on a large body of evidence in several fields that explain the role of specific genes in the operation of a region of the brain along the medial forebrain bundle which is responsible for reward and pleasure.[3] This region is distinct from those that are known to process, develop and retain knowledge. Evidence that different regions of the brain are activated (or correlate) with different economic decisions has been found using fMRI technology in a studies of intertemporal choices (e. g. McClure, Laibson, Loewenstein and Cohen [2004]). The growing evidence in the biomedical literature that presents a significant association between certain genes in this reward system with particular health behaviors and health status such as smoking, alcohol usage, obesity, ADHD, depression and schizophrenia cannot be denied.

It is worth stating explicitly that the goal of this analysis is not to report a causal link between genes and health broadly defined. While we exploit the strong neural correlations between a set of genetic markers and certain health outcomes and behaviors, we do not wish to delve into the often complicated and sometimes controversial debate on how genes affect behavior. For example, the popular press is occasionally filled with stories on the discovery of a gene that specifically codes for obesity or depression that are often quickly refuted by medical authorities.

This study extends the burgeoning literature in economics that seeks to explain the strong correlation between education and health in three directions.[4] First, we present empirical evidence on a causal link running from health to academic performance. Due to biases associated with omitted variables, few studies have either empirically estimated the causal impact of health on education outcomes[5] nor focused on mental health conditions despite evidence that their incidence is substantially larger than physical disorders in adolescence.[6] Exceptions include Currie and Stabile [2005] which presents evidence from sibling fixed effects regressions that the negative impacts on test scores and educational attainment from a specific mental disorder, hyperactivity are quantitatively

larger than those from physical health limitation. Behrman and Lavy [1998] as well as Glewwe and Jacoby [1995] use market instruments such as prices for health. They respectively find that the impact of child health on cognitive achievement varies as a function of the assumptions made concerning parental choices and that much of the impact of child health on school enrolment proxies for unobserved variables. Using an experimental approach, Kremer and Miquel [2004] overcome the omitted variable bias problem by randomly assigning health treatments to primary schools in Kenya. Their analysis displays a mixed picture as improved health from the treatment significantly reduced school absenteeism but did not yield any gains in academic performance.

Second we take a close look at empirical measures of health. The dynamic relationships between health disorders and health behaviors revealed through our analysis clearly present a major empirical challenge. This challenge has not been clarified earlier since the majority of the literature linking health to education focuses on a single measure or proxy of an individual's health such as birth weight due to data limitations.[7] Since an individuals' health consists of many physical and mental health measures including standing heart rate, blood pressure, mental clarity, etc. that constitute a rich vector which not only would be difficult to convert to a single index, but would such a single index exist it is unlikely to be well proxied by measures such as BMI or birthweight.

Third, we make a clear separation of health outcomes from health behaviors. This distinction is not apparent in earlier empirical studies which estimate equations derived from models that either exclusively treat adolescents as a "child" whose parents make all her health and education choices or indistinguishable from "adults" that make all the decisions by themselves. In contrast, we introduce a model that treats adolescents as "adolescents" since they only make a subset of all the decisions. For example, we postulate that a teenager would make decisions such as whether or not to smoke or have sex, while their parents make important human capital investment decisions such as which neighborhood to reside in, which school their child should be sent to, the type of health insurance to purchase and number of visits to health care providers. This hybrid in decision-making is not only more realistic but helps disentangle the impact of health status (a state variable) from health behavior (a control variable) that are treated as equivalent in the earlier literature. Since health

behaviors only explain a limited amount of the variation in health status, they are poor proxies for health status and increase biases due to endogeneity as they may also proxy for non-health preferences such as peer group composition. Further health behaviors could result from as well as cause certain particular health state, which has important policy implications. For example, adolescents may decide to smoke since the nicotine in cigarettes may help self-medicate against craving for food or some mental illnesses. Accounting for the pathway between health status and health behavior is necessary for proper interpretation of our coefficient estimates and could reveal their dynamism that has been understudied in earlier work.

Our empirical analysis reaches four major conclusions:

1) Genetic markers show a great deal of promise as a means to identify the impact of health on education. The individual markers and their two by two polygenic interactions that we consider are highly correlated with each health behavior and status in the study. Moreover consistent with Mendel's hypothesis that the hereditary factors for different genes are independent, statistical tests demonstrate that these markers are not related to each other and only affect academic performance through health outcomes.[8] Further, genetic markers offers several advantages as instrumental variables since concerns regarding reverse causality and spurious correlations are greatly minimized. While this strategy permits statistical identification as we discuss in Section 5.2.3 our instrumental variables estimates should be interpreted as reduced form parameters.

2) The impact of poor health outcomes on academic achievement is substantial. Depression and obesity both lead to a decrease of 0.45 GPA points on average, which is roughly a one standard deviation reduction. However, there is substantial heterogeneity in the impact of health on academic performance across genders. The academic performance of female students is strongly and negatively affected by poor physical and mental health conditions. The estimated magnitudes are substantially smaller for male students and not a single poor health measure has a statistically significant impact.

3) To accurately estimate the impact of health status, it is important to account for endogenous health enhancing or health deteriorating behaviors. We find that treating the stock of lifetime smoking as exogenous leads to substantially different impacts of adverse health status on education.

Cigarette smoking is endogenous and we find that accounting for this choice reduces the negative impact of depression inattention and ADHD by over 50% for the full sample and females. In addition, ignoring the endogeneity of smoking makes the negative impact of depression on males statistically significant.

4) The presence of high comorbidity of health disorders is striking, thus the importance of accounting for it. Comorbidity is defined as having two or more diagnosable conditions at the same time. For example, research has suggested that between 50 to 65 percent of children with ADHD have one or more comorbid conditions such as depression (Pliszka et al. [1999]). Unless the exogenous genetic or environmental factors can be clearly disentangled between these disorders, estimating the causal impact of one disorder in the absence of related health states may not provide accurate results. Since many individuals suffer from more than one disorder, ignoring related illnesses may lead to some misleading conclusions. In our analysis, we find striking differences in the estimated impacts of depression and obesity when one examines a single health state in isolation, which is often the case in most studies.

The rest of the paper is organized as follows. In Section II, we provide an overview of the scientific literature linking genes to health behaviors and health outcomes. An overview of the data we employ in this study is provided in Section III. The framework that guides our understanding of how education and health interact in adolescence is described in Section IV. We discuss the identification strategy and estimating equations in this section. Our results are presented and discussed in Section V. A concluding Section summarizes our findings and discusses directions for future research.

2 Scientific Primer on Genetic Markers

As it was not possible until recently to collect data on genetic markers, empirical researchers in the social sciences traditionally chose to either ignore or assumed the unobserved heterogeneity conferred by variation in genetic inheritance is fixed over time for the same individual or across siblings or

twins. Yet recent advances in fields of molecular and behavioral genetics, most notably through the decoding of the human genome (Venter et al. [2002]) permits researchers to elucidate how differences in the genetic code correlate with differences in specific behaviors or outcomes across individuals. While researchers were able to identify the genetic code for a number of inherited traits and diseases such as eye color, cystic fibrosis, and Huntingdon's disease, most products of inheritance have been found to be polygenic, caused by the interaction of numerous genetic markers. The health outcomes and behaviors we consider in this paper are thought to be polygenic with researchers associating approximately 160 genes with obesity (Perusse et al. [2005]).[9] For these disorders researchers have focused their attention on genes involved in the reward pathway of the brain. This pathway is closely linked to primal drives such as feeding and sex, and has been shown to have a powerful effect on decision making among higher mammals including humans. For example, in a well-known study (Olds [1956]), rats that were given the choice of food versus stimulation of their reward system by electrodes ended up starving to death rather than lessening the stimulation of their pleasure center.

Since the reward system of the brain has been found to be closely linked to numerous human activities such as addiction much research has focused on how variation in different components of the pathway might make an individual more or less predisposed to addiction. In general, this system operates when activities such as feeding or sex are undertaken. A region of the brain known as the ventral tegmental area (VTA) is activated and neurons (brain cells) in the VTA release signaling molecules known as neurotransmitters (in this case dopamine[10]) to another area of the brain known as the nucleus accumbens (NA). These signals pass through the synapses (small gaps separating neurons) until they eventually reach the frontal cortex, where most "decisions" are made. Increases in the synapse of either neurotransmitters or receptor neurons for them allow for a much stronger signal to be sent.[11] Since the response of these neurons to nicotine and other substances has been shown to vary between individuals, it has been hypothesized that genetic differences could explain why different individuals report different levels of "highs" when smoking cigarettes, which is the underlying idea of having a genetic predisposition. In addition, since the VTA-NA pathway is important in regulating pleasure and, therefore, emotion, a number of behavioral traits including

depression, food binging and ADHD have been linked to this pathway.

The genes selected in this study operate either in the liver or in one of the two critical neurotransmission pathways in the reward pathway, the dopamine and serotonin systems. These markers include the, i) Dopamine Receptor D2 locus (DRD2), ii) SLC6A3 locus (DAT), iii) Tryptophan hydroxylase locus (TPH) and iv) CYP2B6 locus (CYP). Each person inherits from each parent a single copy known as an allele for each marker. Alleles can differ by the particular building blocks, or base pairs, that make up all DNA or the number of repeats, or base pairs in a row that repeat themselves. An individual who inherits 2 of the same (different) allele is considered to be homozygous (heterozygous) for that marker. Different allelic combinations are often called polymorphisms.

The DRD2 gene is believed to code for the density of D2 dopamine receptors on neurons in the brain, including those in the VTA and NA. The D2 receptor is one of at least five physiologically distinct dopamine receptors (D1-D5) found on the synaptic membranes of neurons in the brain. The DRD2-A1 allele has been associated with a reduced density of dopamine receptors.[12] Several researchers postulate that the reduced density of dopamine receptors explains the higher associations individuals with DRD2-A1 alleles (A1/A1 or A1/A2) have with compulsive and addictive behaviors including smoking, depression and obesity, relative to individuals with two DRD2-A2 alleles.[13]

The dopamine transporter (DAT) gene (SLC6A3) encodes a reuptake protein that regulates synaptic levels of dopamine in the brain.[14] Variability in the length of the DAT gene is believed to positively influence levels of the reuptake protein in the brain.[15] Individuals with shorter variants of the SLC6A3 gene have diminished dopamine reuptake and greater availability of synaptic dopamine. It has been suggested that by having more synaptic dopamine these individuals receive smaller benefits from substances that stimulate dopamine transmission.

The tryptophan hydroxylase gene (TPH) is a member of the serotonergic neurotransmission system and plays a crucial role in the regulation of mood and impulsivity. This particular gene is involved in the biosynthesis of serotonin, another neurotransmitter that operates in conjunction with the brain's reward system. Serotonin activity has been linked to a number of behavioral and physical conditions including depression, appetite, and addictive behavior.[16]

The CYP genes as a group code for enzymes present in various body organs, primarily the liver which break down a number of drugs and toxins including nicotine. Polymorphisms of these genes have been linked to across population differences in smoking, alcoholism, and response to anti-depression medications.[17]

Finally, different allelic combinations when interacted, can potentially have powerful effects. For example, the level of endogenous synaptic dopamine depends not only on the amount of dopamine released but also on the number of receptors that dopamine can bind to (proxied by the DRD2 gene) as well as the amount of reuptake protein (proxied by the length of the SLC6A3 allele). Similarly, one could imagine that the rate of metabolism determined by the CYP2B6 gene interacts with both the TPH and DRD2 genes.

3 Data

This paper uses data primarily from the Georgetown Adolescent Tobacco Research (GATOR) study. GATOR is a unique longitudinal data set of adolescents that combines information from a series of 5 survey questionnaires given over four years of high school (1999-2003) along with measures of the four genetic markers described in the preceding section.[18]

The study began in 1999 when researchers selected five high schools from the same county in Northern Virginia.[19] Within each school, administrators provided the names and mailing addresses of the complete 9th grade class roster of students. Project information packets, consent forms, a brief demographic/response form and an explanatory cover letter from the school principal were then mailed to 2120 students' homes to recruit study participants.[20] To increase participation rates, up to three waves of mailings were sent and telephone calls were placed to encourage parents to respond. Of the 72% of the parents/guardians (1533 of 2120) who responded to the mailings, three quarters (1151) provided written consent for their adolescent to participate in the study. 99% of the 1151 adolescents who had parental consent to participate provided assent themselves. These mailings also ask the responding parent on their smoking history, age, gender, education, and

biological relationship to the survey participant.

Biological samples were collected using buccal swabs from which DNA was extracted via standard phenol-chloroform techniques. DNA was extracted from buccal cells to avoid a selective exclusion of subjects with blood and injection phobia. Since the method to genotype varies across markers different assays were conducted.[21] In all assays, 20% of the samples were repeated for quality control.[22]

The survey questionnaires provide basic information on student demographic characteristics (i.e. race, gender, etc.), academic performance as measured by GPA (waves 3-5 only), reports on physical activity, detailed information on smoking patterns and smoking history within the household and across a complete set of family members.

Surveys were administered by a GATOR staff member to students who provided assent during a classroom common to all students.[23] Participants were initially surveyed in the spring of 9th grade and resurveyed in both the fall and spring of the 10th grade and in the spring of both the 11th and 12th grades. The rates of participation at the four follow-ups from baseline were about 95%, 96%, 93% and 89% respectively. Participants received $5 gift certificates to media stores to acknowledge their time and participation in this study.

The GATOR data contains numerous questions on health and health behavior. Each survey contained standard epidemiological questions related to self-reported experimentation with, and current use of, cigarettes. Each participant who reported having smoked a cigarette provided additional information on both recent and lifetime cigarette use. From this information, we constructed two variables that represented whether an adolescent was currently smoking cigarettes and years of being a cigarette smoker. A current smoker was defined as having smoked a cigarette within the past month and over one hundred cigarettes over the lifetime. Using this information on being a current smoker with self-reported smoking histories we constructed a conservative measure of number of years of smoking.

With the exception of the survey in the fifth wave, participants completed The Center for Epidemiologic Studies-Depression Scale (CES-D), a 20-item self-report measure of depressive symp-

toms. Items on the CES-D are rated along a 4-point Likert scale to indicate how frequently in the past week each symptom occurred (0 = never or rarely; 3 = very often). The sum of these items is calculated to provide a total score where higher scores indicate a greater degree of depressive symptoms. To determine whether an individual may be depressed, we followed findings from earlier research with adolescent samples (Roberts, Lewinsohn, and Seeley [1991]) who suggest using gender and age appropriate dichotomous cutoff scores (> 24 for female adolescents, > 22 for male adolescents) to ascertain the presence of clinically significant levels of depressive symptoms.

The Current Symptoms Scale-Self Report Form (CSSF), a well-standardized, 18-item self-report measure were used to assess symptoms of Attention-deficit/hyperactivity disorder (ADHD) from DSM-IV (Barkley and Murphy [1998]) in the second wave survey.[24] This form allows participants to rate their recent behavior regarding how often they experience symptoms of inattention (9 items) and hyperactivity-impulsivity (9 items) on a 4-point Likert scale (0 = never or rarely; 3 = very often). Typical diagnostic criteria (endorsement of at least moderate severity on at least six symptoms from either the inattention or hyperactivity-impulsivity subscale) was used to determine the likely presence or absence of clinically significant ADHD symptoms. In the final wave of the GATOR survey participants provided self reports of their height and weight. These measures were used to construct body mass index and we applied standard definitions for being obese (BMI>30).[25]

In total we have information on academic performance as measured by GPA, genetics, health outcomes and health behaviors for 893 study participants. Approximately 90% of these students (807 students) completed the survey in all three years. The top panel of Table 1 presents summary statistics of the time invariant characteristics of the 893 participants in our study. The sample is predominately Caucasian and the largest minority population are Asians. The percentage of African Americans and Hispanics in the student body of the schools in our sample vary between 2.07% to 12.20% and 5.54% to 19.3% respectively. The AD and HD subscale averages fell within standard ranges for adolescent samples. Over 40% of the students report that at least one of their parents was either currently smoking or was an active smoker during their childhood. Finally, the majority of responding parents are biological mothers and possesses a college degree.

The bottom panel of Table 1 presents information on time varying controls and outcomes. Neither GPA nor percentage of students who have a household member that smokes have any substantial change in summary statistic over the three years. In contrast, the number of individuals who currently smoke and have tried smoking rises rapidly during the same period. The percentage of daily smokers in this sample is similar to national averages calculated using the NELS88 (Miller [2005]). The percentage of depressed adolescent in our sample is slightly higher than the 1999 estimate of the fraction of the adolescent population being clinically depressed (12.5%) from the U.S. Department of Health and Human Services. Summary statistics on one year lagged smoking and depression are included since we use these predetermined measures in our empirical analysis since one could postulate that the answers from the psychological questionnaires used to diagnose these conditions could be influenced by current academic performance or another factor which simultaneously affects responses and current academic performance. Finally, we supplemented the GATOR survey data with information from other sources to improve measures of the students' neighborhood and school.[26]

4 Empirical Framework

4.1 The Dynamics From Health to Education

In this section, we present a three-stage model that guides our empirical analysis. The first two stages of our model incorporate elements from three competing theories in three distinct disciplines that explain the heterogeneity in health behaviors across individuals. Economics contributes the standard model of health investment (starting with Grossman, 1972). This model postulates that individuals make inter-temporal decisions trading off immediate satisfactions for future benefits. Different time discount factors and value of life could result in different health choices. Psychologists claim that the heterogeneous health behaviors arise from different environment or situational factors that individuals encounter. Natural scientists hypothesize that genetic variations in single

or multiple genes are associated with health differences across the population.

Stage 1, at the beginning of period T (T_0), adolescents choose whether or not to (continue to) engage in a risky behavior such as smoking, drinking alcohol or using narcotic drugs given their demographics, discount rates, the value of life, genetic markers and home and school environments as well as their current health status (H_{iT-1}). Adolescent i at time T_0 chooses action or behavior k if the immediate satisfaction it provides exceeds the aggregation of the current cost and the perceived future cost to her. The immediate satisfaction that adolescent i derives from action k could be affected by her current health status[27] and her genetic predispositions. The immediate cost of taking action k includes both pecuniary components such as price of cigarette and non-pecuniary components such as how difficult it is to take action k. For instance a teenager may face obstacles in acquiring cigarettes or narcotic drugs that can be measured as time spent. The obstacle faced are determined by neighborhood, school and family environment inputs. For example, increased parental monitoring might make cigarette smoking more costly; a drug infested neighborhood might make drug usage less difficult. The perceived future costs usually depend on the discount rates and the value of life, which may vary with current health status (healthy people are more patient in general) and genetic predispositions. Since the data contains no information on this matter, wlog we assume a non-binding monetary budget constraint for ease of exposition. As a result adolescent i's choice of k is a function of the market price for k that's available to i (p_k) and the health status at time T_0 (H_{iT-1}), given i's endowed predisposition to taking action k — that is, the set of genes (G^k) associated with k and the environment variables that are included in the matrix X_{1iT}.

$$k_{iT} = k(X_{1iT}, H_{iT-1}, p_k, G^k, \epsilon^k_{iT}) \tag{1}$$

where ϵ^k_{iT} captures an independent random shock. This stage of the model can be easily generalized to treat k as a vector of behaviors that are either health enhancing (i.e. proper diet and regular exercise) or health deteriorating (i.e. smoking and drinking).

Stage 2, at time T_1, altruistic parents select a level of health input l_{iT} for adolescent i, given the teenager's observed health behaviors $\tilde{K_{iT}}$ (not necessarily equal to k_{iT}) at the beginning of

this period and revealed health status H_{iT-1}, that provides the highest indirect utility for their household V_{iT}^{l}:

$$V_{iT}^{l} \equiv V_{iT}(X_{2i}, C_{lT}, H_{iT-1}, \tilde{K}_{iT}, G_{i}^{H}), \text{ for each } l \text{ available to } i\text{'s family} \tag{2}$$

where X_{2i} are person-specific and environmental characteristics of the child i; C_{lT} is the cost of health input l at time T which include the cost of insurance payment and the wage-rate forgone when taking care of child i's sickness etc.; and G_{i}^{H} is a vector of genetic markers that provide endowed predispositions to the current state of health status. Given the history of health behaviors chosen by adolescent i and the health inputs chosen by i's parents, health production functions translate these elements into a vector of health outputs as follows

$$H_{iT} = g(X_{2iT}...X_{2i0}, k_{iT}...k_{i0}, l_{iT}...l_{i0,}\ G_{i}^{H}, H_{i0,}\epsilon_{iT}^{H}...\epsilon_{i0}^{H}) \tag{3}$$

where $X_{2iT}...X_{2i0}, k_{iT}...k_{i0}, l_{iT}...l_{i0}$ and $\epsilon_{iT}^{H}...\epsilon_{i0}^{H}$ are the full history of individual and environmental characteristics, health behaviors, health inputs and independent random shocks to health production respectively. Child i's initial health stock at the start of life is represented by H_{i0}.

We assume here a display of single-mindedness in parental preference on child health. That is,

$$U(H_{it}^{1}, \bullet) \geq U(H_{it}^{2}, \bullet) \quad if\ H_{it}^{1} > H_{it}^{2}. \tag{4}$$

We also assume a discrete set of health input levels (i.e. health insurance packages) all well within the budget constraint. By this, we leave out extreme cases where parents have to choose between putting enough food on the table and paying the kid's medical bills. Since our data has no health input information, this assumption places no constraints on the estimation equations. Under these two assumptions, parents will always choose l^{*} that leads to the highest possible level of health for child i.

Stage 3, at the end of period T, T_2, parents choose a set of education inputs (i.e. school quality, employing tutors, etc.) based on the health status of their child. Parents select among these inputs the optimal school j^{*} for child i which provides the highest indirect utility for their household V_{ij}^{*},

$$V_{ij} \equiv V_{ij}(X_{3i}, C_{j}, Q_{j}, A_{iT-1}, I_{i}), \text{ for each } j \text{ available to child } i \tag{5}$$

where X_{3i} are observable person-specific and family characteristics of the child i; C_j is the cost of attending school j, which include the cost if living in a good school district; Q_j is school-specific characteristics; A_{iT-1}indexes child i's measured achievement at the stage of decision making; and I_i is child i's innate abilities. The availability of schools to a child is described by the school admission rules in the local areas where parents can commute to work daily.

Conditional on the selection of school j in the third stage, the standard education production model states that child i in school j at time T gains human capital as measured by a score on an achievement test or report card. The general conceptual model depicts this level of achievement A_{ijT} to be a function of the full history of family, community, school inputs and own innate abilities. Current achievement can be expressed as

$$A_{ijT} = f(X^e_{iT}...X^e_{i0}, Q_{j_T}...Q_{j_o}, H_{iT}, I_i, \epsilon_{iT}...\epsilon_{i0}) \tag{6}$$

where X^e_{it} is a vector of community variables, individual and family characteristics in year t, Q_{jt} is a vector of school characteristics, I_i is a vector of unobserved heterogeneity including such factors as student innate abilities, parental tastes, determination, among others and $(\epsilon_{iT}...\epsilon_{i0})$ are the full history of independent random shocks assumed to have zero mean and no serial correlation.[28]

4.1.1 Health as an Education Input

There are three popular explanations put forth in the health economics literature for the observed positive relationship between health and education. The first model considers education an investment in the future as paying large dividends the longer one lives, thus incentivizing individuals to stay healthy and live longer (Becker [1993]). The second model postulates that education is a critical component in a health production function, thus, educated individuals are better equipped to stay healthy (Grossman [1972]). The third explanation suggests that the relationship exists because both health status and education are directly related to an unobserved variable such as time discounting (Fuchs [1982]) or one's family background (Rosenzweig and Schultz [1983]). However, there's no formal economic model postulating how health enters into the education production process as an

input. As a result, we hypothesize below the possible channels under which health status (H_{iT}) potentially affect education.

First, it may affect the physical energy level of a child which determines the time (including classroom attendance and after school educational activities) that can be used for learning. For example, obesity has been found to be the largest determinant of absenteeism (Schwimmer et al. [2003]). Second, it affects the child's mental status that may have a direct impact on academic performance. For example, obesity is associated with obstructive sleep apnea which impacts energy levels and neurocognitive impairment (See et al. [2006]) and being obese may also cause low self esteem which leads to classroom disengagement that may reduce academic performance. Other health status such as being diagnosed with ADHD or clinical depression may directly affect a child's attention span, which adversely affects her academic outcomes. Third, a child's health status may affect the way her teachers, parents and peers treat her; this in part shapes the learning environment that she encounters. For example, obese children are often less popular among their peers and teachers . Depressed children are associated with personal distress, and if the state lasts a long time or occur repeatedly, they can lead to a circumscribed life with fewer friends and sources of support (Klein et al. [1997]). The first two channels directly affect own health input (both physical and mental) in the education process while the latter scenario influences a child's education outcome through other inputs such as peer quality and teacher attention that is the result of a certain health status.

Ideally we would like to disentangle the effect of obesity on education (the structural parameter) from that which is due to the impact of the environment resulting from being obese. If parents, schools or peers are responding to negative health outcomes by increasing investment into other inputs this may offset the deleterious effects of poor health on achievement. Conversely the response of these individuals could move in a direction that reinforces the deleterious impact of health such as discrimination. For example, parents may decide not to invest or invest less in a child's education due to observed health status of their child. Since our data lacks information on family and school inputs as well as peers, we will obtain a combined (reduced form parameter) impact of health on

education.

4.2 The Estimating Equations

Linearizing the achievement relationship (equation 6) yields

$$A_{ijT}=\beta_{0T}+\beta_{1T}X^e_{iT}+\beta_{2T}H_{ij_T}+\beta_{3T}Q_{j_T}+\beta_{4T}I_i+(\sum_{t=0}^{T-1}\alpha_{0t}+\alpha_{1t}X^e_{it}+\alpha_{3t}Q_{j_t}+\alpha_{4t}I_i+\delta_{it})+\epsilon_{iT} \quad (7)$$

where $\delta_{it} = \alpha_{5t}\varepsilon_{it}$ for some coefficient α_{5t}. The components of equation (7) may include higher order and interaction terms. We re-express the achievement function as

$$A_{ijT} = \beta_0 + \beta_1 X_{iT} + \beta_2 H_{iT} + \beta_3 Q_{jT} + \tilde{\epsilon}_{iT} \quad (8)$$

where the vector X contains individual and family characteristics (gender, race, residential smoking status, responding parent characteristics), the vector H is a vector of variables that captures current predetermined health measures.[29] Similarly both the health production function in equation (3) and the decision to engage in health behavior equation (1) can be expressed as follows:

$$H_{iT} = \gamma_0 + \gamma_1 X_{iT} + \gamma_2 k_{jT} + \gamma_3 G^H_i + \tilde{\epsilon}^H_{iT} \quad (9)$$

$$k_{iT} = \delta_0 + \delta_1 X_{iT} + \delta_2 H_{iT} + \delta_3 G^k_i + \tilde{\epsilon}^S_{iT} \quad (10)$$

Instrumental variable methods are used to estimate the above system of equations ((8) - (10)) to generate consistent estimates of the causal impact of health on education (β_2). Our identification relies on the assumption that the vectors of genetic markers that impact health behaviors and health outcomes (G^k_i and G^H_i) are unrelated to unobserved components of equation (8). While there is absolutely no evidence for the former assumption that the markers considered in this study have any impact on the education production process, it remains possible.[30]

5 Results

5.1 Basic Patterns in the Data

5.1.1 Losing the genetic lottery?

We begin by demonstrating that Mendel's law of independent assortment is supported by the GATOR data and that there is substantial unique variation from each of the markers and their interactions. Summary information on the genetic markers in our data is provided in Table 2. The DAT genotypes are classified with indicator variables for the number of 10-repeat alleles (zero, one, or two). We include indicator variables for the available AA, AC and CC genotypes of the TPH gene. Similarly, the DRD2 gene is classified as A1/A1, A1/A2 or A2/A2. Finally, we include indicator variables for the available CC, CT and TT genotypes of the CYP gene. The first column of Table 2 provides the raw number of individuals who possess each particular marker. Excluding the TPH gene, the majority of individuals in our data are homozygous of A2/A2 (for the DRD2 gene), CC (for the CYP gene) and have two ten repeat alleles of the DAT gene. For each of these genes the heterozygous combination is the next most populated and the remaining homozygous combinations of the CYP (TT) and DRD2 (A1A1) genes are rarest. For the TPH gene there is nearly an equal number of people who possess either the heterozygous AC or homozygous CC combination, with AA being the rarest.

The entries in the remaining columns of Table 2 indicate the number of people in each row that also possess one of the rare polymorphisms of the other genes along with the conditional probability of possessing this combination. Each cell in the table is populated with at least two individuals and there does not exist any systematic relationship between the different genetic polymorphisms.[31] Thus, having a rare polymorphism for one gene does not make it more or less likely that you would have a rare allele combinations in another gene. These results are consistent with Mendel's law of independent assortment and are encouraging as they do not lend support to correlations between markers of different genes.

5.1.2 Candidate Genes for Adolescent Health

To justify our four sets of genetic markers and their two by two polygenic interactions to explain health behavior and status we begin by examining whether there are indeed simple differences in health measures between individuals with different genetic markers. Table 3 presents summary information on health measures for each genetic marker. Each cell contains the conditional mean, standard deviation and odds ratio of alternative health outcomes for individuals that possess a particular marker.

For each genetic marker, there exists a substantial difference in the occurrence rate of at least one of the health outcomes and behaviors.[32] Individuals with the AA polymorphism of the TPH gene have substantially higher propensities (relative to the AC and CC markers) for smoking and obesity respectively. For the CYP gene, those with the rare TT polymorphism are significantly more likely to be diagnosed with inattention (AD) and hyperactivity (HD) relative to those with the common CC marker. For the DRD2 gene, individuals with the common A2A2 allele are significantly less likely to be diagnosed as depressed or obese relative to DRD2 markers that contain an A1 allele. Among the DAT gene, individuals with one 10-repeat allele (DAT1) are more likely to be diagnosed with ADHD and less likely of a depression diagnosis. Individuals that have no 10- repeats (DAT0) are associated with slightly higher smoking rates. These results clearly demonstrate that the four sets of genetic markers have statistically significant associations with our health measures.

5.1.3 Health and Education Outcomes in Adolescence

The well known positive association between good health and educational outcomes is also observed in the data. As indicated in Appendix Table 2, individuals diagnosed with ADHD, depression and obesity respectively have on average GPA scores that are 0.26, 0.18 and 0.43 lower than their counterparts. These differences are statistically significant (one sided t-tests). The raw GPA gap of individuals with ADHD or obesity relative to those not diagnosed increases from grades 10 to 12 by approximately 20%. While the gap between depressed and non-depressed children does not

vary through grades, cigarette smokers close their GPA gap with non-smokers from 0.58 in grade 10 to 0.49 in grade 11 and 0.37 in grade 12. This is somewhat misleading as many adolescents start smoking over time. These new smokers have substantially higher GPA scores than long-term smokers. Between grade 10 and grade 12 long-term smokers consistently have GPA scores that are approximately one half point lower relative to non-smokers.

Not only do smokers have lower GPA scores but they also have a higher propensity of being diagnosed with negative health status. Individuals with each health disorder are significantly more likely to be smokers at the 1% significance level.[33] The largest gaps occur for individuals diagnosed with either inattention or ADHD whose smoking rate is over 250% higher than the remaining population (33% of individuals with ADHD smoke versus 13% of the remaining individuals and 39% of individuals with AD smoke versus 12% of the remaining population). The propensity to smoke is twice as high among adolescents with hyperactivity (HD) relative to those not diagnosed with this disorder. Lastly, adolescents diagnosed as obese or depressed are associated with approximately 50% greater smoking propensities versus the remaining sample.

Comorbid conditions potentially pose a major statistical challenge for identification. Table 4 presents some summary information on the presence of comorbordities in our full sample.[34] Column 1 of Table 4 displays the number of individuals (and marginal distribution) in each wave who smoke or have been diagnosed with either AD, HD, ADHD, obesity or depression. Across each row we present the number of individuals (and conditional frequency) who also engage in smoking or suffer other poor health outcomes. Not only are adolescents who are diagnosed with ADHD more likely to smoke but they also have a higher rate of being diagnosed as either clinically depressed or obese than their cohorts (one sided t-tests). This result is not unique to ADHD as we find that individuals diagnosed with any of these health disorders are significantly more likely to engage in smoking than those not diagnosed in grade 12.

Since health disorders and risky health behaviors are more common among individuals diagnosed with one particular disorder than among the remaining population we will investigate whether estimates of the impacts of a disorder vary if we do not control for comorbidities. The majority of

the literature on the impacts of health generally include only single outcome measure such as obesity, smoking or birthweight in their analysis. Estimates of the impact of health disorders may vary if there are both strong correlations between included and omitted health outcomes and if the omitted health outcomes have a significant impact on the dependent variable. Our genetic instruments are unlikely to be unique to specific disorders as they are associated with the same region of the brain.[35] Thus even with the genetic instruments, excluding significant comorbid conditions may result in estimates of the impacts of included disorder proxying for the effects of the omitted outcomes.

5.2 Estimates of the Empirical Model

Ordinary least squares estimates of equations (8) that ignore the endogeneity of health outcomes and smoking behavior are presented in the top panel of Table 5.[36] In our analysis we consider two different health vectors. The first health vector includes depression, obesity and ADHD. The results are reported in columns 1 - 3. The second health vector (results reported in columns 4 -6) includes depression and obesity but decomposes the diagnosis of ADHD into being clinically inattentive (AD) or clinically hyperactive / impulsive (HD). Results for the full sample are presented in columns 1 and 4, for the sample of females in columns 2 and 5 and the male sample in columns 3 and 6.

As shown in column 1 of Table 5, the impact of each health disorder in the first vector is negatively and significantly associated with academic performance for the full sample. The negative impact of obesity is larger than the magnitude of the other health outcomes. On average obese individuals have a GPA 0.34 points lower, an effect that is larger than that from any race or family variable. Columns 2 and 3 present the results for the subsample of females and males respectively and each health outcome is negatively and significantly related to achievement. The negative impact of obesity is approximately eight times the magnitude of being depressed for females. In contrast to the results for the girls, the magnitude of the coefficients does not vary across the health outcomes for boys. Finally, both the negative impact of the household smoking environment variable and positive impact of whether the biological parent is present is nearly twice as large for boys than for

girls.

Decomposing the impact of ADHD into its components, columns 4 to 6 of Table 5 indicate that AD was responsible for the negative coefficient of ADHD in column 1. For the full sample, HD is positively associated with academic performance but the coefficient is not significant at the 10% level. The impact of obesity relative to depression remains large for females but for boys in column 6 there is a strong negative association between AD and GPA. Interestingly among Asians, females performed significantly better than their Caucasian counterparts.

5.2.1 Endogenous Health Outcomes and Health Behaviors: First-stage Estimates

A potential challenge exists in selecting an appropriate subset of the markers in our data to serve as instruments. The scientific literature provides some (arguably weak) guidance as the evidence tends to be inconsistent across studies.[37] We present and report results from a parsimonious set of instruments selected by forward stepwise estimation and we used twelve different sets using alternative selection criteria to verify the robustness of our findings.[38] We do not vary our instrument set across gender so that any observed difference in terms of health effects is not the result of the selection of different instrument sets that are gender variant.

Statistically, for the markers to serve as instruments they must possess two statistical properties. First, they must have a substantial correlation with the potentially endogenous health variables. Second, they must be unrelated to unobserved determinants of the achievement equation. Table 6 presents results from two specification tests that examine the statistical performance of the instruments for each health equation and sample.

In the top panel of Table 6 we present estimates of the F-statistics of the joint significance of the instruments in the first stage regressions. For each health outcome and health behavior with each sample, the instrument set is jointly statistically significant at a level above current cutoffs for weak instruments.[39] Since our 2SLS estimates (presented in the next sub-section) are over-identified, we use a J-test to formally test the overidentifying restrictions. This test is the principle method to

test whether a subset of instruments satisfy the orthogonality conditions. The associated p-values for these tests are presented in the bottom panel of Table 6. The smallest of the five p-values is a reassuring 0.21, provides little evidence against the overidentifying restrictions. In addition many of the p-values are large and exceed 0.5.

5.2.2 Endogenous Health Outcomes and Health Behaviors: Second-stage Estimates

Two stage least squares (2SLS) results for the achievement equation (8) for the two health vectors is presented in Table 7. Column one presents results for the full sample and depression and obesity are significantly related to academic performance. The impact of depression is approximately three times larger than the OLS estimate presented in Table 5. When ADHD is broken into components (AD and HD) the impact of depression decreases by roughly a third but remains statistically significant as shown in column 4. Hyperactivity and impulsiveness is positively related to academic performance and is significant at the 20% level. In contrast, the portion attributable to AD is negatively related to GPA and statistically significant at the 20% level. These impacts would appear drastic in light of the prevalence of the over prescription of behavioral drugs among school-age children (Eberstadt [2004]).

The results for the subsample of females in columns 2 and 5 are most striking. With health vector one, only obesity is significantly related to academic performance. With health vector two, both depression and obesity lead to significant decreases in GPA. The impact of depression is substantially larger than that obtained using OLS. In contrast, for the subsample of males in columns 3 and 6, health outcomes are no longer statistically significant once we correct for their endogeneity. For each sample and health vector we checked whether health status should be treated as endogenous by testing the null hypothesis that the OLS and 2SLS estimates are equal using a Hausman-Wu test.[40] We can reject the Null of exogeneity of health outcomes for each health vector with each sample at the 5% level.[41]

There are several additional differences between the estimates for males and females.[42] Asian

girls are associated with higher GPA scores among females. Hispanic boys have significantly lower GPA among the males. The magnitude in the 2sls estimates increases relative to OLS for the boys but diminishes by approximately 40% for girls. We should emphasize that our variable indicating whether a smoker resides in the household is a proxy for family environment that we lack direct information on. Concerns regarding whether a smoker residing in the home may represent inheritability of genes from biological parents were examined. First, the raw association between biological parents having been regular smokers and the presence of a smoker in the household is 35%, within the households that smoke approximately 65% of the smokers are other family members. Second, we replicated the analysis in Table 7 excluding this proxy for home environment, the magnitude as well as the statistical significance of the health disorders were unchanged for all three samples and two health vectors.

To demonstrate the robustness of our results, Appendix Tables 4 presents results for the male and female subsample that correspond to their preferred instruments sets using stepwise estimation on those subsamples. While the first stage properties in Appendix Table 4 are improved, a eyeball test confirms that there are no important statistical differences between these estimates and those using the instruments set constructed for the full sample with health vector 1 in Table 7. Similarly, combining the separate instrument sets for males and females and estimating the system of equations for the full sample yields no observable differences. For females with health vector 2, the negative impact of AD increases substantially and becomes statistically significant. Similarly, the impact of depression and obesity increases by 25% with this alternative instrument set. Overall, the results continue to demonstrate that females suffer large decreases in their GPA when they have been diagnosed with depression or are obese; whereas no significant relationships exist for the males.

5.2.3 Discussion

The parameter estimates we obtain should be viewed as reduced form coefficients that might include dynastic effects.[43] Information on parental and teacher investment as well as peer group composition

is not available to disentangle the impact of the health condition as explained by genes from that of the response from the environment to the health conditions as explained by genes. While this appears unsatisfying, this limitation is also implicitly shared by other empirical strategies used to estimate the impact of health on education which generally either treat genetics as part of a big blackbox that can be eliminated under strong assumptions or propose the use of alternative instrumental variables such as an individual's phenotype. The availability of genes as instrumental variables for the first time makes it crystal clear the level of difficulty in obtaining structural parameter estimates and the importance of detailed accurate information on health and education inputs.[44] Further, structural parameters of this kind even if they could be obtained, may quickly become invalid every time a new (medical) treatment is developed that changes the occurrence rate or severity of these disorders' negative impacts.

The use of exact measures of genes permits us to enter what traditionally has been a blackbox in empirical economics. Studies that exploit variation within siblings or within twins not only assume that the set of genetic factors do not vary between pairs but implicitly the *impacts* of these factors and unobserved (to the analyst) family investments are constant between family members. Most unsatisfying is that one can not test the validity of these two assumptions and if they are refuted biases could increase from differencing.[45] Increasing scientific evidence shows that monozygotic human twins are discordant in many physical traits and diseases which is not only ascribed to environmental factors but also epigenetic modifications.[46] Epigenetics refers to DNA and chromatin modifications that play a critical role in regulation of various genomic functions. Essentially a substantial degree of epigenetic variation can be generated during the mitotic divisions of a cell in the absence of any specific environmental factors. This variation which results primarily from stochastic events is either assumed to be the same in the sibling and twin differencing strategies or has zero impacts on outcomes. In the social sciences, researchers often consider sibling fixed effects model as they potentially control for (unobserved) parental characteristics and could allow the researcher to exploit a genetic lottery between family members. Yet, this empirical strategy does not effectively deal with endogeneity bias that results from either parents adjusting their fertility

patterns in response to the (genetic) quality of their earlier children or which results from differential time varying investments across siblings. These two factors have strong empirical support in the social science literature and within evolutionary biology models of human fertility.[47] Further, this empirical strategy is inconsistent with many underlying economic models (Rosenzweig and Wolpin, [2000]) and it implicitly imposes an assumption of strict exogeneity on the explanatory variables in the model which rules out the possibility that predetermined characteristics of the siblings may either purposely or inadvertently influence each other. This assumption directly contradicts the available evidence that indicates sibling behavior is a stronger risk factor for health behavior than parental behavior (Rajan et al, [2003], Vink et al. [2003] and Avenevoli and Merikagnas [2003]).

As noted, the use of genes as an instrument presents a challenge in regards to intergenerational transmission. It is well known that offspring of parents with psychological problems are more likely to develop these disorders. For example, it has been estimated that 40% of children with depressed parents experience psychiatric disorders by the age 20 (Beardslee et al. [1998]). Data from the Minnesota Twin Family Study finds a weak positive association between maternal depression and offspring depression but does not find any evidence of an association between paternal depression with either maternal or offspring depression. The mechanism by which parental disorders influence offspring psychopathology has not been clearly established. While we lack direct information on parental diagnoses, we use knowledge of comorbidities to construct proxies. That is, we use parental smoking to proxy for parental health. We estimated variants of our principal empirical model where we separately as well as jointly included variables on whether the responding parent is currently smoking or has ever smoked as well as whether the subject reported that at least one of his biological parents smoked in their lifetime as additional control variables. Our results were both quantitatively and qualitatively robust to the inclusion of these parental health measures. This result is not a surprise since our genetic markers possess several properties that increase our confidence in their conceptual validity as instruments. First, the genes we consider are pleiotropic and second they cannot credibly account for the majority of the variation in the diagnoses of these health disorders. Thus, even if a parent possessed the same markers for any of these four genes as their child, this

would neither guarantee that they suffer from the same disorders nor that these particular genes would affect the parent and child in similar fashion.

Our coefficient estimates may also capture a dynastic effect of the impact of health disorders. Without more detailed data on parental diagnoses as well as parental genes we can not separate out the portion of the impact that is uniquely brought on by the child's condition. As a result, this effect may include the impact of family environments provided by depressed parents whose depression can be explained by exactly the same set of genes and genetic interaction terms that we selected to explain the child's depression in our study. This dynastic effect is of policy relevance since individuals are in general not randomly assigned to families and policymakers are generally interested in the total impact of these disorders. Similarly if the assortative mating process is stable, then the dynastic effect is important to recover since kids with certain disorders will increasingly come from families that also have this disorder. It is also worth noting that there is limited evidence that individuals seek out partners with similar genetic makeup. Animal studies on mate choice have shown that both signals of genetic quality and genetic diversity play important roles whose relative weight varies according to the respective ranges of these characteristics in the study population.[48] The pursuit of genetic diversity serves to weaken intergenerational correlations, especially on adverse health attributes.

A concern may exist regarding the conceptual validity of the instruments since dynastic effects may suggest that the genetic markers we consider influence academic outcomes through channels outside of child health status. Conceptually since the estimating equations used in Table 7 include predetermined outcomes of the responding parent such as education as explanatory variables, should the identical markers manifest in the same manner within a family we are directly accounting for all these predetermined impacts of genes on parental outcomes that subsequently affect the child's education. Empirically the quantitative and qualitative patterns of our 2sls results are robust to the exclusion of information on the parents and other family members education, smoking and age which further increases our confidence in the validity of the instruments.

To summarize, the genetic markers we employ in our study are predetermined to any interaction

that the adolescents have with the environment, even those interactions such as pre-natal care that occur in utero and affect measures such as birth weight and APGAR scores. They possess strong correlations with certain health disorders and health outcomes. At present there is no detectable evidence that they are correlated with genetic factors that associate with inputs to either innate ability or the development of intelligence. We are not ruling out the possibility that these genes affect the acquisition of intelligence but rather we are assuming that these genes neither directly enter the education production process nor correlated with genes directly involved in production of these education outcomes. The assumptions underlying these markers for identification are supported by both statistical tests and the scientific literature. Not only can these assumptions be tested but we argue that this strategy imposes substantially weaker assumptions on the relationship between nature, nurture and adolescent outcomes than other empirical strategies used in the literature. Despite these advances substantially richer data would be needed to recover the structural parameter.

5.3 Accounting for Endogenous Cigarette Smoking Matters

With genetic markers as instruments we can investigate the extent to which smoking is a choice variable. Past research in economics has suggested that smoking could proxy for an individuals' discount rate and have implicitly assumed that smoking does not reflect a choice.[49] Treating cigarette smoking as an exogenous input to health outcomes presents striking changes to our results. Table 8 presents 2SLS estimates of equations (8) and (9) that assume this choice is exogenous. Notice that the magnitude of all health outcomes in Table 8 increases markedly from those presented in Table 7, where smoking was treated endogenous. Most surprising is that by treating smoking as an exogenous behavior, the estimates on the impact of depression, HD and obesity become statistically significant for males. The results suggest that being obese leads boys to score 0.8 points higher on their GPA. The sign and magnitude of this estimated impact seems implausible. For the full sample and subsample of girls, the estimated impact of depression nearly doubles in magnitude. In addition, ADHD becomes statistically significant for the full sample. Finally, the estimates on

AD and HD for girls become implausibly large but continue to offset one another. The implausible magnitude of these coefficients are a result of both limited independent variation to separately identify impacts and the use of smoking as an invalid exclusion restriction.

We conducted a Hausman test of each health status equation for each vector in Table 8 by comparing it to the corresponding equation in Table 7. We can reject the Null of exogeneity for years of cigarette smoking, suggesting that smoking is indeed a choice variable. Our investigation into the endogeneity of smoking shows that despite the use of genes as instruments for the health outcomes, the different ways of accounting for the smoking decision leads to very different results. This could result from the fact that genes associated with smoking tendency are also associated with health disorders and that smoking may have a direct impact on our health disorders.[50]

To further investigate whether smoking patterns do indeed have different relationships with diagnosed health disorders between the genders we present OLS and 2SLS estimates of the impacts of smoking on each health outcomes for each sample and health vector in Appendix Table 5.[51] Whereas smoking is positively associated with each health outcome when treated as exogenous (in the bottom panel), the 2sls estimates present different patterns. Smoking is positively related to depression and negatively related to obesity once we account for endogeneity as reported in column 1. Further, boys who smoke are significantly less likely to be diagnosed with hyperactivity but more likely to be diagnosed with depression and inattention. In contrast, females who smoke are less likely to be diagnosed with depression. These gender differences add a further layer of complexity and support the possibility that smoking patterns account for some of the gap in the impacts of health disorders on education between the genders.

5.4 Accounting for Comorbid Health Outcomes Matters

We now consider what, if any, effect it would have on our estimates if we followed the usual practice of ignoring comorbid conditions and only include one health outcome in the achievement equation at a time. Two stage least squares estimates are presented in Table 9, where each entry refers to

the point estimate of that health behavior from a system of equation that included the achievement equation, the particular health outcome and health behavior.

Examining results from separate regressions using the full sample, we would conclude that inattention is positively and HD negatively related to GPA, which is the opposite of the pattern reported in Table 7. The results for the subsample of boys completely change when comorbid conditions are omitted. Obesity, AD and HD are all positively related to academic performance and the magnitude of the impact for obesity is extremely large. Similarly, for the full sample and subsample of girls the impact of depression is approximately 40% larger as it may be capturing a portion of the negative impact of obesity or ADHD. Taken together, the results of Table 8 and Table 9 illustrate the need to account for a greater set of related health outcomes and endogenous behaviors in any analysis. Even with exogenous instruments such as genes to correct for the endogeneity of health status, the omission of comorbid conditions and behaviors may present a misleading picture of the causal relation between particular health states and academic performance among other outcomes.[52]

Due to the high comorbidity in health conditions as demonstrated in our study and the lack of exogenous variations that can explain one particular condition only, the coefficient for one particular health condition such as obesity may reflect the composite effect of several health conditions, thus the reliability of that coefficient is dependent on the rich controls we have on most of the comorbid health conditions. Without the rich information on health, most of the exogenous variations cannot identify the impact of one condition only.[53]

6 Conclusions

Understanding the consequences of growing up in poor health for adolescent development is an important research question. This question is particularly interesting to policymakers since part of the explicit rationale for programs such as Medicaid is to improve the development of children. However, it is challenging to address due to endogeneity that arises from omitted variables and

measurement error problems pertaining to health.

In this paper, we use information on genetic markers to overcome these challenges and identify the causal effect of health on education via an instrumental variables strategy. The explicit use of genetic markers in empirical social science research is becoming possible due to an ever increasing understanding of how genetic inheritance relates to individual health outcomes as well as knowledge from the human genome project. This knowledge increase the conceptual validity of the instrument since i) the markers are inherited at conception prior to any interaction with the environment eliminating concerns related to reverse causality, ii) a large literature reports robust correlation between the markers and health variables we consider in this study suggesting that the correlations are not spurious, iii) studies of genetic inheritance indicate that the assignment of the markers we consider are independent of hereditary factors associated with the development of intelligence, and iv) while these genes are pleiotropic they only influence academic outcomes through adolescent health status channels as we directly account for predetermined parental education outcomes. Empirically, statistical tests confirm the strong correlations in the first stage relationship as well as do not support the overidentifying restrictions. Hausman tests further speaks to the strength of our instruments as the IV estimates are statistically different from the OLS estimates which also indicates that we should treat health as an endogenous input to education. Further, the quantitative and qualitative patterns of our empirical results are robust to the inclusion of information on the parents and other family members in the estimating equations.

Using these genes as a novel source of identification we find that the impact of poor health on academic achievement is large. Depression and obesity both lead to a 0.45 point decrease on GPA, which is roughly a one standard deviation reduction in performance. There exists substantial heterogeneity in the impacts of health status on academic performance as female adolescents are strongly adversely affected by negative physical and mental health conditions, whereas males are not significantly impacted.

Several results from our empirical investigation have important implications for the health economics literature. First, we find in explaining health status researchers must account for comorbid

health disorders. Since many individuals suffer from more than one disorder, ignoring related illnesses may lead to some misleading conclusions regarding the impacts of one particular disorder when it is examined in isolation. This issue is particularly challenging since one can not easily overcome biases when measuring a particular health state with error using an instrumental variables strategy, unless there exists an instrument that can clearly disentangle the variation between related comorbid disorders such as ADHD and depression. Second, we make a clear separation of health outcomes (a state variable) from health behavior (a control variable) in our theoretical and empirical analysis. Empirically we find that treating health behaviors as exogenous or ignoring comorbid conditions would lead to either different signed estimates or substantially larger impacts of health on education. Since health behaviors only explain a limited amount of the variation in health status and could result from as well as cause certain particular health states, accounting for this pathway is important as it could reveal a dynamic relationship that could be examined in future work.

The results also suggest that future research is needed to improve our understanding on why females and not males are so adversely affected by poor health outcomes. For example, responses to a variety of psychological questionnaires can be used to shed light on possible differences between females and males in their self-perception. Future research could also incorporate additional dynamics such as how parents, teachers and peers respond to an individual's changing health state to explore more deeply some of the sources for this heterogeneity.

Finally, measures of genetic markers could also be used in other lines of research in the social sciences. One could use them as a source of identification to assess the impact of health as a form of human capital on many outcomes such as labor market activity, marriage and educational attainment. Researchers could also investigate whether nurture inputs or family characteristics can offset the impact of genetic predispositions. In conclusion, recent years have witnessed an explosion of findings on the causes and correlates of health outcomes and behaviors in neurobiology, which could offer a promising source of predetermined exogenous variations to help identify the impact of health on a set of outcomes of great interest to economists.

Notes

[1]The importance of genetic factors to behavioral characteristics and health outcomes has been noted throughout history. The passage of physical and disease traits from parents to offspring was first explicitly studied and modeled by Gregor Mendel in the 19th century. Since this work more sophisticated studies of laboratory animals as well as comparisons between monozygotic and dizygotic human twins demonstrate that behavioral characteristics and economic as well as health outcomes were in part linked to genetic inheritance. Most recently, Cutler and Glaeser [2005] compares the correlation of health behaviors between monozygotic and dizygotic twins and conclude that approximately 72% of the variation in obesity and 30% of the variation in cigarette smoking are due to genetic factors.

[2]These impacts should be viewed as reduced form parameters and our analysis will clarify the difficulties in estimating the structural health parameter. In Section 5.2.3 we discuss issues surrounding identification that include intergenerational transmission, potential dynastic effects, assortative matching and ideal data requirements. We also discuss how using genes as instruments to identify the impacts of health offers several benefits over alternative empirical approaches, most importantly we can directly test the identifying assumptions.

[3]This evidence summarized in Section 2 suggests that possessing the genetic markers considered in our study indeed increases the sensitivity of individuals being diagnosed with certain health disorders. Second, there is no detectable evidence that the markers we consider are correlated with other genetic factors that associate with either innate ability or the development of intelligence. Note, we are not ruling out the possibility that these genes affect outcome measures of intelligenece but rather we are assuming that these genes neither directly enter or correlate with the genes directly involved in the education production process; they only affect achievement through health.

[4]This correlation has been explained in three ways that are not necessarily mutually exclusive. The first hypothesis is that education increases health through productive or allocative efficiency (Grossman [1972], Kenkel [1991]). The second hypothesis is the converse that poor health results

in little education (Perri [1984], Currie and Hyson [1999]). Finally, others have suggested that this correlation could be caused by a third unobserved variable (e.g. discount rate) that affects both education and health (Fuchs [1982]).

[5]Grossman and Kaestner [1997] note that the majority of the empirical literature reports correlations and focuses on the effect of education on health. Strauss and Thomas [1998] present a survey of the literature on the relationship between health and income. More recently, Bleakley (2006) presents evidence that cohorts who were exposed to a large scale public health intervention against hookworm in childhood were associated with larger gains in income and higher rates of return to schooling later in life.

[6]Chapter 3 of Mental Health: A Report of the Surgeon General clearly states that "approximately one in five children and adolescents experiences the signs and symptoms of a DSM-IV disorder during the course of a year".

[7]For example, see Behrman, Rosenzweig, and Taubman [1994], Currie and Hyson [1999], Behrman and Rosenzweig [2004] or Almond, Chay and Lee [2005].

[8]Note, more recent evidence suggests that not all hereditary factors assort independently but that those which are located close together on the same chromosome tend to be inherited as a unit, not as independent entities. This property is termed linkage in the genetics literature. This does not threaten our analysis as the markers we consider are i) not in close proximity to those markers believed to associate with intelligence, and ii) not located close to each other. Further, the National Center for Biotechnology Information in their online science primer on the genome, interpret the evidence on linkage as being random across individuals but note that some regions on the chromosone are more likely to have links than others.

[9]Our literature survey indicated that studies have examined whether associations exist between approximately 300 different genes and ADHD (e. g. Comings et al. [2000] examined 42 in their study alone).

[10]Dopamine has been called the “pleasure” chemical of the brain because people who are electrically stimulated in the limbic dopaminergic centers of the brain report intense feelings of well-being

and sometimes orgasm.

[11]Certain food and drugs such as nicotine or caffeine can have an especially powerful effect on the reward center of the brain as they mimic or potentiate the effects of neurotransmitters that occur there naturally. This process is often described as a molecular "hijacking" of the reward pathway. For example, nicotine has been shown to increase levels of synaptic dopamine by stimulating dopamine release in the VTA (Di Chiara and Imperato [1988]) and inhibiting dopamine reuptake in the reward pathway (Carr et al. [1992]).

[12]This finding was first reported in Blum et al. [1991].

[13]See Audrain-McGovern [2004] and Epstein et al. [2002] and the references within for evidence on these associations.

[14]Bannon, Granneman, and Kapatos [1995] presents an overview of the SLC6A3 gene. The SLC6A3 gene has been implicated in Parkinson's disease (Seeman and Niznik [1990]), attention deficit disorder (Cook et al. [1995]) and Tourette's syndrome (Connors et al. [1996]).

[15]The length is associated with the number of variable tandem repeats on each marker. Each repeat increases the amount of reuptake protein. The majority of individuals have SLC6A3 alleles with lengths of 9 or 10 base pairs, where the length is positively associated with levels of DAT protein. Note the SLC6A3 loci may also take the form of 7- repeat, 8-repeat, 11-repeat or 12-repeat; each of which is extremely rare in both the population and our sample.

[16]See Lucki [1998] for evidence of these associations.

[17]See Lerman et al. [2001, 2003] for a discussion.

[18]At present, these are the only genes that have been collected for the full sample. For subsets of approximately two hundred subjects information on the COM-T, CYP2A6 and OPRM1 genes are also available. As we discuss later in this section we use specific assays for each gene product as these methods are substantially more accurate (lower misclassification rates) than newer technologies which can provide information on large sections of the genome or gene expression.

[19]A total of 21 high schools exist in this county. Using data from the NCES CCD we did not find any significant differences in student demographics or standard school input measures between

schools included and excluded from the sample. Note, we cannot identify this county by name, but is large and affluent as it contained over 950,000 residents with a median household income of $70,000 in 1995.

[20]Students who the principals indicated special class placement, such as a severe learning disability or difficulty speaking and understanding the English language were excluded from the study. In total 273 students or 11% of the total population were excluded.

[21]For example in conducting SLC6A3 genotyping the following assay was conducted. DNA (25 ng) was mixed with primers (20 pmol), GeneAmp PCR buffer (10 mM tris-HCl pH 8.3, 50 mM KCl, 1.5 mM MgCl2 , and 0.0001% gelatin; Perkin Elmer, Norwalk, CT), Amplitaq DNA polymerase (2.5 μ; Perkin Elmer, Norwalk, CT), and 2'-deoxynucleotides-3'-triphosphates ($144\mu M$; Pharmacia, Piscataway, NJ) in 50-μl total volume. The reaction conditions included an initial melting step (94^0C; 4 min) followed by 35 cycles of melting (94^0C; 1 min), annealing (65^0C; 1 min), and extending (72^0C; 1 min). The VNTR repeat was then determined with a 4% agarose gel electrophoresis (3:1 nusieve:agarose). The authors would be happy to provide full details on the assays for the other markers by request. Note each assay was validated by confirming a polymorphic inheritance pattern in seven human family lines encompassing three generations.

[22]Quality control procedures included positive and negative controls with each assay and independent repeat genotyping for 20% of the results. The rate of discordance was less than 5%, and ambiguous results were not reported. In total, genetic information was obtained for 1032 subjects.

[23]Students without parental consent completed classroom assignments during the administration of these surveys. Classroom teachers and school administrative personnel did not participate in the survey portion of the research, nor were they permitted to view participants' responses. Students were identified on the completed survey by an identification number and during each wave a member of the research team read aloud a set of instructions, emphasizing confidentiality to promote honest responding, and encouraged questions if survey items were not clear. To minimize missing data, make-up days were scheduled for those adolescents who were absent during the regular survey administration. Further, surveys were mailed to the homes of students who had either switched

schools or dropped out of school.

[24]Barkley and Murphy [1998] describe the scoring algorithmn. Being diagnosed with ADHD means that an individual has been diagnosed with either AD or HD. It also does not make a distinction between individuals with one or both disorders. It is important to state explicitly that we are not focusing on diagnosed cases but rather on responses to questions which are used to construct a diagnosis known only to researchers.

[25]Our results are robust to alternative cutoffs for obesity, ADHD and depression.

[26]Data at the school level was obtained from the CCD and neighborhood information was obtained from US census records at the zip code level.

[27]Research has also suggested that individuals with ADHD employ nicotine to enhance cognitive function (e.g. Coger et al. [1996], Levin et al. [1996] and Pomerleau et al. [1995]).

[28]Boardman and Murnane [1979] present a clear discussion of the model underlying education production functions.

[29]This model is commonly used in the economics of education literature and alternatively one could include lagged measures of achievement in the specification. These modelling decisions place implicit assumptions on the effects of all previous observed and unobserved influences in the current period. The empirical validity of these alternative assumptions has only recently been tested (Ding and Lehrer [2005], Todd and Wolpin [2005]). Note that since parents may choose to make investments in their children based on their health status, our estimates should be viewed as an upper bound of the health impact on academic performance if the investment is positively related to good health. Conversely, if the investment is negatively related to good health, our estimates provide a lower bound.

[30]Plomin et al. [2006] and de Quervain and Papassotriopoulos [2006] present recent surveys on which genes are believed to be associated with intelligence and memory ability respectively. Researchers have found no links between several of the genes in this study and either intelligence (i.e. Moises et al. [2001]) or cognitive ability (e.g. Petrill et al. [1997]).

[31]Statistically, to determine whether there were links between markers of different genes we

conducted regressions and tests for homogeneity of odds ratios to see whether possessing a given marker increased the odds of possessing a specific marker for a different gene. We did not find any evidence indicating a systematic relationship between markers of any two of these genes.

[32]In addition, we conducted simple linear regressions by gene of health outcomes on discrete indicators for possessing each allele combination. The regression results are available by request. Several relationships are statistically significant and we denote statistically different odds ratios with an asterik in Table 3.

[33]Results from one sided t-tests.

[34]Note the high prevalance of comorbid conditions is not unique to this sample. This is a well known empirical regularity in the medical literature particularly among mental health conditions. For example, Biederman et al. [1995] report that 70% of adults with ADHD are treated for depression at some point in their life.

[35]Recall, from the scientific literature that these disorders are believed to be polygenic and that there is no unique depression or obesity or ADHD gene. Pharmaceutical companies are now in the process of examining the use of nicotine patches to deal with ADHD. Ritalin, which is currently prescribed to children with ADHD was originally developed as an anti-depressant.

[36]The full set of estimates from the system of equations are available by request.

[37]These studies tend to use very small unrepresentative clinical samples and suffer from low statistical power. Since it is not possible (and probably unethical) to engage in random mutations of an individual genetic code we argue it is best to treat genetic predispositions as a form of neural correlates with health behaviors and health status.

[38]To examine the robustness of our results we have now considered twelve different instrument sets for the equations. One set involved the use of the complete set of the markers in our study, another set was constructed based on our reading of the neuroscientific literature up to May 2005 and the remaining ten sets were constructed from stepwise estimation using alternative selection criteria. Our empirical results (available upon request) are robust to the instrument set for the full sample and sub-sample of males. The statistical significance of the estimates of the negative impact

of inattention (AD) on GPA for females varies across instrument sets for females. yet it should be noted that in those specifications where AD entered significantly the first stage F statistic was fairly small which could indicate that the standard erros are too small due to weak instruments.

[39]We report equation by equation results in Table 6 to demonstrate that the results are not driven by the instruments performing well in some health equations and not in others. For information on cutoffs, see Stock and Yogo [2005]. Note that weak identification could result in the 2SLS estimates being inconsistent and biased towards the OLS estimates and Hahn and Hausman (2003) show that the finite sample bias of these estimates is inversely related to the first stage F statistic. Yet as we will demonstrate in the next section with second stage estimates, a Hausman test rejects the consistency of the OLS estimates.

[40]In the event of weak instruments (as well as overfitting), the 2SLS estimates would be biased towards the OLS estimates. Note an examination of each health equation indicates that higher levels of parental education are positively associated with ADHD and depression but negatively related to obesity and cigarette smoking.

[41]We also considered the more efficient 3sls estimation of equation (8) where we accounted for the one way error component structure of $\tilde{\epsilon}_{iT}$ in running GLS. There are limited efficiency gains and the results available on request indicate no substantial differences in the magnitude or significance of any of our results moving from 2sls to 3sls.

[42]Appendix Table 3 presents comorbidities by gender. There are substantially fewer girls diagnosed with both AD and HD relative to boys. In contrast, there are more females diagnosed solely with depression.

[43]From a statistical perspective, the 2sls estimate can take a causal interpretation as a local average treatment effect (LATE) provided the conditions described in Angrist, Imbens and Rubin [1996] are satisfied. This allows for heterogeneous impacts of health across individuals and the LATE parameter is simply the average causal effect on education that can be attributed to the health disorders for the subset of the population whose health disorders are induced by the chosen set of genetic markers and their interactions (or, at least, a mechanism that the genetic markers

reflect).

[44]Yet this parameter is likely of policy interest unless one were to believe that all educational inputs are caused by health outcomes, the particular genes we employ should not be correlated with most nurture inputs. For instance, if one is to believe that the health resulted inputs rather than health itself (i.e. the different investment from parents, peers and teachers a child receives if in poor health and the family environment from parents who experience similar poor health outcomes) are the major cause for the impact of health on education, then any investigation on intervening policies should focus on those inputs.

[45]The notion that estimates with samples of twins may increase biases is discussed in Bound and Solon [1999] and Neumark [1999] in the context of estimating the returns to education.

[46]For example, while 80% of the variation in schizophrenia is assumed to be heritable only half of monozygotic twin pairs in which at least one twin has the disease, share the disorder. In total, only 10% of diseases are assumed to be due strictly to heritable genetic factors. Gringas and Chen [2001] discuss the mechanisms that lead monozygotic twins to be genetically different.

[47]For example birth order, birth spacing and sex composition have been shown to affect differential levels of investment by parents into children (e. g. Hanushek [1992], Black, Devereux, and Salvanes [2005] and Conley and Glauber [2005]). The use of prior birth outcomes based on both sex composition and neo-natal or infant mortality to influence subsequent fertility decisions is well-established (i.e. Angrist and Evans [1998] and Preston [1985]).

[48]Roberts and Gosling [2003] use experiments with rodents to reach this conclusion and note that genetic diversity is desired since it increases reproductive success.

[49]This idea is due to Farrell and Fuchs [1982] and subsequent studies such as Evans and Montgomery [1994] have tried to use smoking as an instrument for education in wage equations. Hammermesh [2000] argues that smoking behavior is a measure of family background and is unlikely to be a valid instrument for education.

[50]Appendix Table 6 presents 2sls estimates of the smoking equation. Note inattention only impacts smoking for males, whereas depression is associated with more smoking for the full sample

and males.

[51]While simple t-tests between the genders indicate that there are no systematic differences in in current smoking and year smoked, among those diagnosed with either depression, ADHD, AD, or HD boys smoked cigarettes with significantly more tar and nicotine content. Males with mental disorders may use the nicotine in the cigarettes to self-medicate against these disorders since nicotine is well known to have a positive effect on attention and indirect effects on the dopaminergic system, potentially reducing symptoms of ADHD and depression (Conners et al. [1996]). While it may appear unlikely that only males would self-medicate with tobacco, a recent survey in the psychiatric literature (Perkins et al. [1999]) concludes that gender differences in the motivation for tobacco consumption and maintenance exist in both human and animal populations. Smoking differs from other health behaviors such as drug or alcohol use as it is not known to impair judgment and the detrimental health impacts come much later in life relative to drug use, thus appears to be less damaging in the present.

[52]This may be due to the fact that the genes are associated with more than one health outcome in a vector. But if genetic markers cannot separate one health outcome from another, it is hard to imagine that any nurture or environmental factor could break the statistical association between these disorders. This issue does not have a simple solution.

[53]Similarly, the variation generated from a randomized intervention that provides a vaccination for a unique medical condition when used to identify on one condition may have effects on other conditions as well. If we rely on the randomized hookworm vaccination to identify the effect of the hookworm condition rather than the effect of vaccination, then the assumption that the vaccination has no significant effect on other conditions is called for. Thus it's not clear that a randomized medical treatment can help identify the effect of a single condition.

References

[1] Almond, D., K. Y. Chay and D. S. Lee (2005), "The Costs of Low Birth Weight," forthcoming in *Quarterly Journal of Economics*.

[2] Angrist, J. D. and W. Evans. (1998), "Children and Their Parents' Labor Supply: Evidence from Exogenous Variation in Family Size," *American Economic Review*, 88, 450-477.

[3] Angrist, J. D., G. W. Imbens, and D. B. Rubin (1996), "Identification of Causal Effects Using Instrumental Variables," *Journal of the American Statistical Association*, 91 444-472.

[4] Audrain-McGovern, J., D. Rodriguez, K. P. Tercyak, J. Cuevas, K. Rodgers and F. Patterson (2004), "Identifying and Characterizing Adolescent Smoking Trajectories," *Cancer Epidemiology Biomarkers & Prevention,* 13, 2023-2034.

[5] Audrain-McGovern, J., K. P. Tercyak, P. Goldman and A. Bush (2002), "Recruiting Adolescents into Genetic Studies of Smoking Behavior," *Cancer Epidemiology Biomarkers & Prevention,* 11, 249-52.

[6] Avenevoli, S., K. Ries, K. R. Merikangas (2003), "Familial Influences on Adolescent Smoking, *Addiction,* 98, 1-20.

[7] Barkley, R. A. and K. R. Murphy (1998), *Attention-Deficit/Hyperactivity Disorder: A Clinical Workbook* (2nd ed), New York: The Guilford Press.

[8] Bannon M. J., P. Sacchetti and J. G. Granneman (1995), "The Dopamine Transporter: Potential Involvement in Neuropsychiatric Disorders" in F. E. Borroni and D. J. Kupfer (eds.), *Psychopharmacology: The Fourth Generation of Progress,* New York: Raven Press Ltd., 179-188.

[9] Beardslee W. R., S. Swatling, L. Hoke, L, P. C. Rothberg, P. van de Velde, L. Focht and D. Podorefsky (1998), "From Cognitive Information to Shared Meaning: Healing Principles in Prevention Intervention," *Psychiatry,* 61(2), 112-130.

[10] Becker, G. S. (1993), *Human Capital: A Theoretical and Empirical Analysis with Special Reference to Education* (3d edition), Chicago: The University of Chicago Press.

[11] Behrman, J. R. and M. R. Rosenzweig (2004), "Returns to Birthweight," *Review of Economics and Statistics*, 86, 586-601.

[12] Behrman, J. R. and V. Lavy (1998), "Child Health and Schooling Achievement: Association, Causality and Household Allocations," *CARESS Working Papres 97-23*, University of Pennsylvania.

[13] Behrman, J. R., M. R. Rosenzweig and P. Taubman (1994), "Endowments and the Allocation of Schooling in the Family and in the Marriage Market: The Twins Experiment," *Journal of Political Economy,* 102, 1131-1174.

[14] Biederman J., S. Faraone, E. Mick and E. Lelon (1995), "Psychiatric comorbidity among Referred Juveniles with Major Depression: Fact or Artifact?," *Journal of American Academy of Child and Adolescent Psychiatry,* 34, 579-590.

[15] Black, S., P. Devereux, and K. Salvanes (2005), "The More the Merrier? The Effect of Family Size and Birth Order on Children's Education," *Quarterly Journal of Economics*, 120, 669-700.

[16] Bleakley, H. C. (2006), "Disease and Development: Evidence from Hookworm Eradication in the American South." forthcoming in *Quarterly Journal of Economics*.

[17] Blum, K., E. P. Noble, P. J. Sheridan, O. Finley, A. Montgomery, T. Ritchie, T. Ozkaragoz, R. J. Fitch, F. Sadlack, D. Sheffield, T. Dahlmann, S. Halbardier and H. Nogami (1991), "Association of the A1 Allele of the D2 Dopamine Receptor Gene with Severe Alcoholism," *Alcohol,* 8, 409-416.

[18] Boardman, A. E. and R. J. Murnane (1979), "Using Panel Data to Improve Estimates of the Determinants of Educational Attainment," *Sociology of Education,* 52, 113-121.

[19] Bound J. and G. Solon (1999), "Double Trouble: On the Value of Twins-Based Estimation of the Return to Schooling", *Economics of Education Review*, 18, 169-182.

[20] Carr, L. A., J. K. Basham, B. K. York and P. P. Rowell (1992), "Inhibition of Uptake of l-methyl-4-phenylpyridinium Ion and Dopamine in Striatal Synaptosomes by Tobacco Smoke Components," *European Journal of Pharmacology,* 215, 285-287.

[21] Coger, R. W., K. L. Moe and E. A. Serafetinides (1996), "Attention deficit disorder in adults and nicotine dependence: Psychobiological factors in resistance to recovery?", *Journal of Psychoactive Drugs*, 28, 229-240.

[22] Comings, D. E., R. Gade-Andavolu, N. Gonzalez, S. Wu, D. Muhleman, H. Blake, F. Chiu, E. Wang, K. Farwell, S. Darakjy, R. Baker, G. Dietz, G. Saucier and J. P MacMurray (2000), "Multivariate Analysis of Associations of 42 Genes in ADHD, ODD and Conduct Disorder," *Clinical Genetics,* 58(1), 31-40.

[23] Comings, D. E., S. Wu, C. Chiu, R. H. Ring, R. Gade, C. Ahn, J. P. MacMurray, G. Dietz and D. Muhleman (1996), "Polygenic Inheritance of Tourette Syndrome, Stuttering, Attention Deficit Hyperactivity, Conduct, and Oppositional Defiant Disorder: The Additive and Subtractive Effect of the Three Dopaminergic Genes–DRD2, D beta H, and DAT1," *American Journal Of Medical Genetics,* 67, 264-288.

[24] Conley, D. and R. Glauber, (2005) "Parental Education Investment and Children's Academic Risk: Estimates of the Impact of Sibship Size and Birth Order from Exogenous Variation in Fertility," *NBER Working Paper w11302.*

[25] Conners, K. C., E. D. Levin, E. Sparrow, S. C. Hinton, D. Erhardt, W. H. Meck, J. E. Rose and J. March (1996), "Nicotine and Attention in Adult Attention Deficit Hyperactivity Disorder (ADHD)", *Psychopharmacology Bulletin*, 32, 67-73.

[26] Cook, E. H. Jr., M. A. Stein, M. D. Krasowski, N. J. Cox, D. M. Olkon, J. E. Kieffer and B. L. Leventhal (1995), "Association of Attention-Deficit Disorder and the Dopamine Transporter Gene," *American Journal of Human Genetics,* 56, 993-998.

[27] Currie, J. and M. Stabile (2005), "Child Mental Health and Human Capital Accumulation: The Case of ADHD," forthcoming in *Journal of Health Economics.*

[28] Currie, J. and R. Hyson (1999), "Is the Impact of Health Shocks Cushioned by Socioeconomic Status? The Case of Low Birthweight," *American Economic Review,* 89, 245-250.

[29] Cutler, D. and E. Glaeser (2005), "What Explains Differences in Smoking, Drinking and Other Health-Related Behaviors?" *NBER Working Paper w11100.*

[30] de Quervain, D. J.-F. and A. Papassotiropoulos (2006), "Identification of a Genetic Cluster Influencing Memory Performance and Hippocampal Activity in Humans ," *Proceedings of the National Academy of Sciences USA,* 103, 4270-4274.

[31] Di Chiara, G.. and A. Imperto (1988), "Drugs Abused by Humans Preferentially Increase Synaptic Dopamine Concentrations in the Mesolimbic System of Freely Moving Rats," *Proceedings of the National Academy of Sciences USA,* 85, 5274-5278.

[32] Ding, W., and S. F. Lehrer (2005), "Accounting for Unobserved Ability Heterogeneity within Education Production Functions," *mimeo*, Queen's University.

[33] Eberstadt, M. (2004), *Home-Alone America: The Hidden Toll of Day Care, Behavioral Drugs, and Other Parent Substitutes*, New York: Sentinel Press.

[34] Evans, W. and E. Montgomery (1994), "Education and Health: Where There's Smoke There's an Instrument," *NBER Working Paper w4949.*

[35] Epstein, L. H., J. L. Jaroni, R. A. Paluch, J. J. Leddy, H. E. Vahue, L. Hawk, E. P. Wileyto, P. G. Shields, and C. Lerman (2002), "Dopamine Transporter Genotype as a Risk Factor for Obesity in Smokers," *Obesity Research*, 10(12), 1232-1240.

[36] Farrell, P. and V. R. Fuchs (1982), "Schooling and Health: The Cigarette Connection," *Journal of Health Economics,* 1, 217-230.

[37] Fuchs, V. R. (1982), "Time Preference and Health: An Explanatory Study" in Victor R. Fuchs ed. *Economic Aspects of Health*, University of Chicago Press for NBER, Chicago.

[38] Glewwe, P. and H. Jacoby (1995), "An Economic Analysis of Delayed Primary School Enrollment in a Low-Income Country-the Role of Early Childhood Nutrition," *Review of Economics and Statistics*, 77, 156-169.

[39] Gringras, P. and W. Chen (2001), "Mechanisms for Differences in Monozygous Twins," *Early Human Development,* 64, 105-117.

[40] Grossman, M. and R. Kaestner (1997), "Effects of Education on Health," in J. R. Behrman and N. Stacey eds. *The Social Benefits of Education*, University of Michigan Press, Ann Arbor.

[41] Grossman, M. (1975), "The Correlation between Health and Schooling," in *Household Production and Consumption*, Ed N. E. Terleckyj, Studies in Income and Wealth, Vol. 40, Conference on Research in Income and Wealth. New York: Columbia University Press for the National Bureau of Economic Research,

[42] Grossman, M. (1972), "On the Concept of Health Capital and the Demand for Health," *Journal of Political Economy*, 80, 223-255.

[43] Hahn, J., Hausman, J. 2003. Instrumental Variable Estimation with Valid and Invalid Instruments., *MIT Department of Economics Working Paper No. 03-26.*

[44] Hamermesh, D. (2000), "The Craft of Labormetrics," *Industrial and Labor Relations Review*, 53, 363-380.

[45] Hanushek, E. (1992), "The Trade-off between Child Quantity and Quality," *Journal of Political Economy*, 100 84-117.

[46] Imbens, G. W. and J. D. Angrist (1994), "Identification and Estimation of Local Average Treatment Effects," *Econometrica,* 62, 467-475.

[47] Kenkel, D. (1991), "Health Behavior, Health Knowledge and Schooling," *Journal of Political Economy*, 99, 287-305.

[48] Klein, D. N., P. M. Lewinsohn and J. R. Seeley (1997a), "Psychosocial Characteristics of Adolescents with a Past History of Dysthymic Disorder: Comparison with Adolescents with Past Histories of Major Depressive and Non-Affective Disorders, and Never Mentally Ill Controls," *Journal of Affective Disorders*, 42, 127-135.

[49] Kremer M. and E. Miguel (2004), "Worms: Identifying Impacts on Education and Health in the Presence of Treatment Externalities," *Econometrica,* 72, 159-217.

[50] Lerman, C., N. E. Caporaso, A. Bush, Y. Zheng, J. Audrain, D. Main and P. G. Shields (2001), "Tryptophan Hydroxylase Gene Variant and Smoking Behavior," *American Journal of Medical Genetics*, 105, 518-520.

[51] Lerman, C., and W. Berrettini (2003), "Elucidating the Role of Genetic Factors in Smoking Behavior and Nicotine Dependence," *American Journal of Medical Genetics (Neuropsychiatric Genetics)*, 118B, 48-54.

[52] Levin, E. D., K. C. Conners, E. Sparrow, S. C. Hinton, D. Erhardt, W.H. Meck, J.E. Rose and J. March (1996), "Nicotine Effects on Adults with Attention-Deficit/Hyperactivity Disorder", *Psychopharmacology*, 123, 55–63.

[53] Lucki, I., (1998), "The Spectrum of Behaviors Influenced by Serotonin," *Biological Psychiatry*, 44, 151-162.

[54] Mays Jr., H. L. and G. E. Hill, "Choosing Mates : Good Genes versus Genes that are a Good Fit", *Trends in Ecology and Evolution,* 10(19), 554-559.

[55] McClure, S. M., D. I. Laibson, G. Loewenstein and J. D. Cohen (2004), "Separate Neural Systems. Value Immediate and Delayed. Monetary Rewards," *Science*, 306, 503-507.

[56] Miller, D. C. (2005), "Adolescent Cigarette Smoking: A Longitudinal Analysis Through Young Adulthood," *NCES Working Paper #2005333.*

[57] Moises H. W., R. M. Frieboes, P. Spelzhaus, L. Yang, M. Kohnke, O. Herden-Kirchhoff, P.Vetter, J. Neppert, and I. Gottesman (2001), "No Association between Dopamine D2 Receptor Gene (DRD2) and Human Intelligence.," *Journal of Neural Transmission*, 108, 115-121.

[58] Neumark, D. (1999), "Biases in Twin Estimates of the Return to Schooling," *Economics of Education Review*, 18, 143-148.

[59] Olds, J. (1967), "The Limbic System and Behavioral Reinforcement," *Progress in Brain Research*, 27, 144-64.

[60] Perri, T. J. (1984), "Health Status and Schooling Decisions of Young Men", *Economics of Education Review,* 3, 207-213.

[61] Perusse, L., T. Rankinen, A. Zuberi, Y. C. Chagnon, S. J. Weisnagel, G. Argyropoulos, B. Walts, E. E. Snyder and C. Bouchard (2005), "The Human Obesity Gene Map: The 2004 Update," *Obesity Research*, 13, 381-490.

[62] Petrill, S. A., R. Plomin, G. E. McClearn, D. L. Smith, S. Vignetti, M. J. Chorney, K. Chorney, L. A. Thompson, D. K. Detterman, C. Benbow, D. Lubinski, J. Daniels, M. Owen and P. McGuffin (1997), "No Association between General Cognitive Ability and the A1 Allele of the D2 Dopamine Receptor Gene," *Behavior Genetics*, 27(1), 29-31.

[63] Pliszka S. R., C. L. Carlson, J. M. Swanson (1999), ADHD with Comorbid Disorders: Clinical Assessment and Mangement, New York: Guilford Press.

[64] Plomin, R., J. K. J. Kennedy and I. W. Craig (2006), "The Quest for Quantitative Trait Loci Associated with Intelligence ," forthcoming in *Intelligence.*

[65] Pomerleau, O. F., K. K. Downey, F. W. Stelson and C. S. Pomerleau (1995), "Cigarette Smoking in Adult Patients Diagnosed with Attention Deficit Hyperactivity Disorder", *Journal of Substance Abuse*, 7(3), 373-378.

[66] Preston, S. H. (1985), Mortality in Childhood: Lessons from WFS, in John G. Cleland and John Hobcraft (eds.), Reproductive Change in Developing Countries, Oxford: Oxford University Press, pp. 46-59.

[67] Rajan, K. B., B. G. Leroux, A. V. Peterson Jr., J. B. Bricker, R. M. Andersen, K. A. Kealey, and I. G. Sarason (2003), "Nine-year Prospective Association Between Older Siblings' Smoking and Children's Daily Smoking," *Journal of Adolescent Health*, 33, 25– 30.

[68] Roberts, C. S. and M. L. Gosling (2003), "Genetic Quality and Similarity Interact in Mate Choice Decisions by Female Mice," *Nature Genetics,* 35, 103-106.

[69] Roberts, R. E., P. M. Lewinsohn, and J. R. Seeley (1991), "Screening for Adolescent Depression: Comparison of Depression Scales," *Journal of the American Academy of Child and Adolescent Psychiatry*, 30, 58-66.

[70] Rosenzweig, M. R. and K. I. Wolpin (2000), "Natural "Natural Experiments" in Economics," *Journal of Economic Literature*, 38, 827-874.

[71] Rosenzweig, M. R. and T. P. Schultz (1983), "Estimating a Household Production Function: Heterogeneity, the Demand for Health, Inputs, and Their Effects on Birth Weight," *Journal of Political Economy*, 91, 723-746.

[72] See, C. Q, E. Mensah and C. O. Olopade (2006), "Obesity, Ethnicity, and Sleep-Disordered Breathing: Medical and Health Policy Implications," *Clinics in Chest Medicine,* 27, 521-533.

[73] Seeman, P., and H. B. Niznik (1990), "Dopamine Receptors and Transporters in Parkinson's Disease and Schizophrenia," *The FASEB Journal*, 4, 2737-2744.

[74] Sibley, D. R. and F. J. Monsma, Jr. (1992), "The Molecular Biology of the Dopamine Receptors", *Trends in Pharmacological Science*, 13, 61-68.

[75] Schwimmer, J. B., T. M. Burwinkle and J. W. Varni (2003), "Health-Related Quality of Life of Severely Obese Children and Adolescents," *Journal of American Medical Association*, 289, 1813-1819.

[76] Stock, J. H. and M. Yogo (2005), "Testing for Weak Instruments in Linear IV Regression," in J.H. Stock and D.W.K. Andrews (eds), Identification and Inference for Econometric Models: Essays in Honor of Thomas J. Rothenberg, Cambridge University Press..

[77] Strauss, J. and D. Thomas (1998), "Health, Nutrition, and Economic Development," *Journal of Economic Literature*, 36(2), 766-817.

[78] Todd, P. E. and K. I. Wolpin (2004), "The Production of Cognitive Achievement in Children: Home, School and Racial Test Score Gaps," *mimeo*, University of Pennsylvania.

[79] Venter, J. C., M. D. Adams, E. W. Myers, P. W. Li, R. J. Mural, G. G. Sutton, H. O. Smith, M. Yandell, C. A. Evans, R. A. Holt, J. D. Gocayne, P. Amanatides, R. M. Ballew, D. H. Huson, J. Russo Wortman, Q. Zhang, C. D. Kodira, X. H. Zheng, L. Chen, M. Skupski, G. Subramanian, P. D. Thomas, J. Zhang, G. L. Gabor Miklos, C. Nelson, S. Broder, A. G. Clark, J. Nadeau, V. A. McKusick, N. Zinder, A. J. Levine, R. J. Roberts, M. Simon, C. Slayman, M. Hunkapiller, R. Bolanos, A. Delcher, I. Dew, D. Fasulo, M. Flanigan, L. Florea, A. Halpern, S. Hannenhalli, S. Kravitz, S. Levy, C. Mobarry, K. Reinert, K.Remington, J. Abu-Threideh, E. Beasley, K. Biddick, V. Bonazzi, R. Brandon, M. Cargill, I. Chandramouliswaran, R. Charlab, K. Chaturvedi, Z. Deng, V. Di Francesco, P. Dunn, K. Eilbeck, C. Evangelista, A. E. Gabrielian, W. Gan, W. Ge, F. Gong, Z. Gu, P. Guan, T. J. Heiman, M. E. Higgins, R. Ji, Z. Ke, K. A. Ketchum, Z. Lai, Y. Lei, Z. Li, J. Li, Y. Liang, X. Lin, F. Lu, G. V. Merkulov, N. Milshina, H. M. Moore, A. K Naik, V. A. Narayan, B. Neelam, D. Nusskern, D. B. Rusch, S. Salzberg, W. Shao, B. Shue, J. Sun, Z. Yuan Wang, A. Wang, X. Wang, J. Wang, M. Wei, R. Wides, C. Xiao,

C. Yan, A. Yao, J. Ye, M. Zhan, W. Zhang, H. Zhang, Q. Zhao, L. Zheng, F. Zhong, W. Zhong, S. C. Zhu, S. Zhao, D. Gilbert, S. Baumhueter, G. Spier, C. Carter, A. Cravchik, T. Woodage, F. Ali, H. An, A. Awe, D. Baldwin, H. Baden, M. Barnstead, I. Barrow, K. Beeson, D. Busam, A. Carver, A. Center, M. Lai Cheng, L. Curry, S. Danaher, L. Davenport, R. Desilets, S. Dietz, K. Dodson, L. Doup, S. Ferriera, N. Garg, A. Gluecksmann, B. Hart, J. Haynes, C. Haynes, C. Heiner, S. Hladun, D. Hostin, J. Houck, T. Howland, C. Ibegwam, J. Johnson, F. Kalush, L. Kline, S. Koduru, A. Love, F. Mann, D. May, S. McCawley, T. McIntosh, I. McMullen, M. Moy, L. Moy, B. Murphy, K. Nelson, C. Pfannkoch, E. Pratts, V. Puri, H. Qureshi, M. Reardon, R. Rodriguez, Y. Rogers, D. Romblad, B. Ruhfel, R. Scott, C. Sitter, M. Smallwood, E. Stewart, R. Strong, E. Suh, R. Thomas, N. Tint, S. Tse, C. Vech, G. Wang, J. Wetter, S. Williams, M. Williams, S. Windsor, E. Winn-Deen, K. Wolfe, J. Zaveri, K. Zaveri, J. F. Abril, R. Guigó, M. J. Campbell, K. V. Sjolander, B. Karlak, A. Kejariwal, H. Mi, B. Lazareva, T. Hatton, A. Narechania, K. Diemer, A. Muruganujan, N. Guo, S. Sato, V. Bafna, S. Istrail, R. Lippert, R. Schwartz, B. Walenz, S. Yooseph, D. Allen, A. Basu, J. Baxendale, L. Blick, M. Caminha, J. Carnes-Stine, P. Caulk, Y. Chiang, M. Coyne, C. Dahlke, A. Deslattes Mays, M. Dombroski, M. Donnelly, D. Ely, S. Esparham, C. Fosler, H. Gire, S. Glanowski, K. Glasser, A. Glodek, M. Gorokhov, K. Graham, B. Gropman, M. Harris, J. Heil, S. Henderson, J. Hoover, D. Jennings, C. Jordan, J. Jordan, J. Kasha, L. Kagan, C. Kraft, A. Levitsky, M. Lewis, X. Liu, J. Lopez, D. Ma, W. Majoros, J. McDaniel, S. Murphy, M. Newman, T. Nguyen, N. Nguyen, M. Nodell, S. Pan, J. Peck, M. Peterson, W. Rowe, R. Sanders, J. Scott, M. Simpson, T. Smith, A. Sprague, T. Stockwell, R. Turner, E. Venter, M. Wang, M. Wen, D. Wu, M. Wu, A. Xia, A. Zandieh, and X. Zhu (2001), "The Sequence of the Human Genome," *Science*, 291, 1304-1351.

[80] Vink, J. M, Willemsen, G. Gonneke and D. I. Boomsma (2003), "The Association of Current Smoking Behavior with the Smoking Behavior of Parents, Siblings, Friends and Spouses," *Addiction*, 98, 923–931.

Table 1: Summary Characteristics of the Sample

Time Invariant Variables N=893						
Variable		Mean		Standard Deviation		
Male		0.469		0.499		
African American		0.073		0.260		
Hispanic		0.093		0.291		
Asian		0.106		0.308		
Caucasian		0.667		0.471		
Body Mass Index		23.426		4.410		
Obese (BMI>=30)		0.081		0.272		
School 1		0.176		0.381		
School 2		0.249		0.432		
School 3		0.214		0.410		
School 4		0.138		0.345		
School 5		0.227		0.419		
AD diagnosis		0.043		0.202		
HD diagnosis		0.040		0.197		
ADHD diagnosis		0.063		0.243		
Responding Parent is Female		0.749		0.431		
Age of Responding Parent		45.613		5.734		
Responding Parent is High School Dropout		0.062		0.234		
Responding Parent is High School Graduate		0.092		0.282		
Responding Parent has Some College		0.207		0.398		
Responding Parent is College Graduate		0.641		0.487		
Responding parent is Biological Parent		0.966		0.180		
Responding Parent Currently Smokes		0.103		0.299		
Responding Parent was a Regular Smoker		0.354		0.472		
A Biological Parent was regular smoker in lifetime		0.449		0.498		
Time Varying Variables						
	Grade 10 Mean	Grade 10 Standard Deviation	Grade 11 Mean	Grade 11 Standard Deviation	Grade 12 Mean	Grade 12 Standard Deviation
Tried Smoking	0.433	0.495	0.483	0.500	0.533	0.499
Current Smoker	0.091	0.288	0.152	0.359	0.178	0.382
Years as a Regular Smoker	0.116	0.398	0.245	0.680	0.399	0.968
Currently depressed	0.161	0.368	0.117	0.322	N/A	N/A
Smoker in Household	0.241	0.428	0.246	0.431	0.231	0.422
Grade Point Average (GPA)	3.184	0.567	3.148	0.598	3.176	0.571
Age	16.032	0.399	17.030	0.396	18.034	0.400
Depressed last period	0.168	0.374	0.169	0.375	0.122	0.327
Smoker last period	0.088	0.283	0.095	0.293	0.147	0.354
Lagged number of years smoking	0.071	0.278	0.120	0.406	0.235	0.662
Number of observations	834		863		879	

Table 2: Summary Information on Genetic Markers in the Sample

Gene	Marker	Total Number of People	Number of people also have AA	Number of people also have TT	Number of people also have A1A1	Number of people also have DAT0
GenoTPH	AA	120 [0.135]	****	4 (0.033)	5 (0.042)	16 (0.133)
	AC	393 [0.440]	****	15 (0.038)	20 (0.051)	39 (0.099)
	CC	380 [0.426]	****	12 (0.032)	27 (0.071)	65 (0.171)
GenoCYP	TT	31 [0.035]	4 (0.129)	****	2 (0.065)	3 (0.097)
	CT	191 [0.214]	24 (0.126)	****	9 (0.047)	19 (0.099)
	CC	671 [0.751]	92 (0.137)	****	41 (0.061)	56 (0.083)
DRD2	A1A1	52 [0.058]	5 (0.096)	2 (0.038)	****	3 (0.058)
	A1A2	286 [0.320]	34 (0.119)	9 (0.031)	****	19 (0.066)
	A2A2	555 [0.622]	81 (0.146)	20 (0.036)	****	56 (0.101)
DAT	DAT0	72 [0.081]	16 (0.222)	3 (0.042)	3 (0.042)	****
	DAT1	317 [0.355]	38 (0.120)	13 (0.041)	17 (0.054)	****
	DAT2	498 [0.558]	65 (0.131)	15 (0.030)	32 (0.064)	****

Note: Each cell contains the number of individuals that possess the respective row and column combination of genetic markers. The conditional frequency of having the dual markers is presented in round parentheses. The marginal frequency of possessing a marker is presented in square parentheses.

Table 3: Relationship Between Genetic Markers with Health Behaviors and Health Outcomes During Adolescence

Gene	Marker	Depression	Smoking	Obesity	BMI	ADHD	AD	HD
TPH	AA	0.149 (0.357) [0.176]	0.158 (0.365) [0.188]***	0.108 (0.312) [0.122]	23.939 (4.516)	0.067 (0.250) [0.071]	0.033 (0.180) [0.035]	0.033 (0.180) [0.035]
	AC	0.150 (0.357) [0.178]	0.105 (0.306) [0.117]	0.074 (0.262) [0.080]	23.291 (4.140)	0.074 (0.262) [0.080]	0.048 (0.215) [0.051]	0.043 (0.204) [0.045]
	CC	0.156 (0.363) [0.185]	0.101 (0.301) [0.112]	0.079 (0.270) [0.086]	23.403 (4.640)	0.050 (0.218) [0.053]	0.039 (0.195) [0.041]	0.039 (0.195) [0.041]
CYP	TT	0.165 (0.373) [0.197]	0.121 (0.328) [0.138]	0.032 (0.180) [0.033]	22.536 (3.283)	0.129 (0.341) [0.148]	0.129 (0.341) [.148]***	0.097 (0.301) [0.104]*
	CT	0.159 (0.366) [0.189]	0.111 (0.315) [0.125]	0.058 (0.234) [0.061]	23.082 (4.195)	0.031 (0.175) [0.032]**	0.010 (0.102) [0.011]***	0.026 (0.160) [0.027]
	CC	0.150 (0.357) [0.177]	0.109 (0.312) [0.123]	0.089 (0.286) [0.098]	23.565 (4.508)	0.069 (0.253) [0.074]	0.048 (0.213) [0.050]	0.042 (0.200) [0.044]
DRD2	A1A1	0.189 (0.393) [0.233]	0.122 (0.328) [0.138]	0.096 (0.298) [0.106]	23.562 (5.998)	0.058 (0.235) [0.061]	0.038 (0.194) [0.040]	0.038 (0.194) [0.040]
	A1A2	0.174 (0.380) [0.211]	0.100 (0.301) [0.112]	0.115 (0.320) [0.130]***	23.860 (4.651)	0.049 (0.216) [0.051]	0.021 (0.144) [0.021]	0.035 (0.184) [0.036]
	A2A2	0.138 (0.345) [0.160]***	0.114 (0.318) [0.129]	0.061 (0.240) [0.065]***	23.189 (4.088)	0.070 (0.256) [0.076]	0.054 (0.226) [0.057]***	0.043 (0.204) [0.045]
DAT	DAT0	0.155 (0.363) [0.183]	0.155 (0.363) [0.183]	0.077 (0.268) [0.083]	23.685 (5.310)	0.064 (0.247) [0.069]	0.038 (0.194) [0.040]	0.051 (0.222) [0.054]
	DAT1	0.109 (0.311) [0.139]***	0.122 (0.327) [0.122]	0.095 (0.293) [0.105]	23.775 (4.749)	0.091 (0.289) [0.101]***	0.063 (0.244) [0.067]***	0.060 (0.238) [0.064]***
	DAT2	0.172 (0.378) [0.207]***	0.104 (0.306) [0.116]	0.072 (0.259) [0.078]	23.161 (4.004)	0.044 (0.206) [0.046]***	0.030 (0.171) [0.031]***	0.026 (0.160) [0.027]***

Note: Each cell presents the conditional mean, the standard deviation in round parentheses and the odds ratio for outcomes (excluding BMI) in square parentheses. ***,**, * denote the Null of homogeneity of odds across markers by genotype from a chi-squared test is rejected at the 1%, 5%, 10% level respectively. The tests were conducted with the same sample used to construct Table 1.

Table 4: Relationship between Health Behaviors and Health Outcomes During Adolescence

Behavior	Total Number	Nothing Else[1]	Also Smokes	Also AD	Also HD	Also ADHD	Also Obese	Also Depressed
Wave 3, N=834								
Nothing	471 [0.565]	***	***	***	***	***	***	***
Smokes	73 [0.088]	36 (0.493)	***	7 (0.096)	4 (0.055)	8 (0.110)	7 (0.096)	16 (0.219)
AD	33 [0.040]	5 (0.152)	7 (0.212)	***	14 (0.424)	33 (1.000)	3 (0.091)	15 (0.455)
HD	30 [0.036]	8 (0.267)	4 (0.133)	14 (0.467)	***	30 (1.000)	2 (0.067)	10 (0.333)
ADHD	49 [0.059]	25 (0.510)	8 (0.163)	33 (0.673)	29 (0.592)	***	4 (0.082)	19 (0.388)
Obese	68 [0.082]	39 (0.574)	7 (0.103)	3 (0.044)	2 (0.029)	4 (0.059)	***	17 (0.250)
Depression	140 [0.168}	93 (0.664)	16 (0.114)	15 (0.107)	10 (0.071)	19 (0.136)	17 (0.121)	***
Wave 4, N=863								
Nothing	477 [0.553]	***	***	***	***	***	***	***
Smokes	82 [0.095]	42 (0.512)	***	9 (0.110)	5 (0.061)	10 (0.122)	10 (0.122)	21 (0.256)
AD	37 [0.043]	7 (0.189)	9 (0.243)	***	17 (0.459)	37 (1.000)	4 (0.108)	15 (0.405)
HD	34 [0.039]	9 (0.265)	5 (0.147)	17 (0.5)	***	34 (1.000)	3 (0.088)	9 (0.265)
ADHD	54 [0.063]	25 (0.463)	10 (0.185)	37 (0.685)	33 (0.611)	***	5 (0.093)	19 (0.352)
Obese	70 [0.081]	34 (0.486)	10 (0.143)	4 (0.057)	3 (0.043)	5 (0.071)	***	17 (0.243)
Depression	146 [0.169]	96 (0.656)	21 (0.144)	15 (0.103)	9 (0.062)	19 (0.130)	17 (0.116)	***
Wave 5, N=879								
Nothing	483 [0.595]	***	***	***	***	***	***	***
Smokes	129 [0.147]	60 (0.465)	***	15 (0.116)	11 (0.085)	18 (0.14)	15 (0.116)	20 (0.155)
AD	38 [0.043]	8 (0.211)	15 (0.395)	***	18 (0.474)	38 (1.000)	4 (0.105)	10 (0.263)
HD	36 [0.041]	8 (0.222)	11 (0.306)	18 (0.500)	***	36 (1.000)	3 (0.083)	9 (0.250)
ADHD	56 [0.064]	30 (0.536)	18 (0.321)	38 (0.679)	36 (0.643)	***	5 (0.089)	15 (0.268)
Obese	67 [0.076]	28 (0.418)	15 (0.224)	4 (0.06)	3 (0.045)	5 (0.075)	***	10 (0.149)
Depression	107 [0.122]	66 (0.617)	20 (0.187)	10 (0.093)	9 (0.084)	15 (0.140)	10 (0.093)	***

Note: Each cell contains the number of individuals diagnosed with the respective row and column combination. The conditional frequency of dual diagnoses is presented in round parentheses. The marginal probability of being diagnosed with each outcome is presented in square parentheses.

[1] For ADHD nothing else excludes AD and HD.

Table 5: Ordinary Least Squares Estimates of the Achievement Equation

	Full Sample	Females Only	Males Only	Full Sample	Females Only	Males Only
ADHD	-0.218*** (0.071)	-0.216** (0.106)	-0.230** (0.098)	N/A	N/A	N/A
AD	N/A	N/A	N/A	-0.408*** (0.099)	-0.358 (0.185)	-0.464*** (0.125)
HD	N/A	N/A	N/A	0.111 (0.084)	0.032 (0.107)	0.164 (0.124)
Depression	-0.130*** (0.029)	-0.059* (0.033)	-0.221*** (0.050)	-0.125*** (0.029)	-0.056* (0.033)	-0.214*** (0.049)
Obesity	-0.341*** (0.071)	-0.469*** (0.088)	-0.191 (0.103)	-0.340*** (0.071)	-0.474*** (0.087)	-0.185* (0.101)
Smoker in Home	-0.159*** (0.037)	-0.110** (0.045)	-0.215*** (0.057)	-0.158*** (0.036)	-0.111** (0.045)	-0.207*** (0.056)
Age	0.743 (0.455)	0.570 (0.566)	0.716 (0.709)	0.748* (0.456)	0.559 (0.567)	0.725 (0.711)
Age Squared	-0.023* (0.013)	-0.016 (0.016)	-0.024 (0.021)	-0.024* (0.013)	-0.016 (0.016)	-0.024 (0.021)
African American	-0.278*** (0.056)	-0.219*** (0.065)	-0.315*** (0.098)	-0.283*** (0.056)	-0.226*** (0.065)	-0.314*** (0.098)
Hispanic	-0.185*** (0.064)	-0.147 (0.101)	-0.201** (0.087)	-0.176*** (0.064)	-0.136 (0.102)	-0.192** (0.087)
Asian	0.149*** (0.051)	0.221*** (0.067)	0.026 (0.077)	0.147*** (0.051)	0.212*** (0.067)	0.037 (0.077)
Male	-0.267** (0.032)	N/A	N/A	-0.261*** (0.032)	N/A	N/A
Parent is HS Dropout	-0.179** (0.088)	-0.161 (0.112)	-0.198 (0.140)	-0.172* (0.088)	-0.145 (0.113)	-0.211 (0.140)
Parent is HS Grad	-0.238*** (0.067)	-0.245*** (0.082)	-0.210** (0.105)	-0.232*** (0.066)	-0.238*** (0.081)	-0.210** (0.104)
Parent some College	-0.147*** (0.044)	-0.205*** (0.057)	-0.062 (0.070)	-0.143*** (0.043)	-0.198*** (0.056)	-0.064 (0.068)
Biological Parent	0.376*** (0.106)	0.272* (0.144)	0.423*** (0.139)	0.358*** (0.102)	0.271* (0.145)	0.387*** (0.133)
Constant	-3.576 (3.939)	-2.534 (4.936)	-3.517 (6.181)	-3.614 (3.950)	-2.470 (4.941)	-3.588 (6.205)
N	2576	1366	1210	2576	1366	1210
R squared	0.24	0.26	0.21	0.25	0.26	0.22

Note: Corrected standard errors in parentheses. Regressions include school and time period indicators. ***,**, * denote statistical ignificance at 1%, 5%, 10% level respectively.

Table 6: Summary Information on the Performance of the Instruments

	Full Sample	Females Only	Males Only	Full Sample	Females Only	Males Only
First Stage F statistics						
Full System	19.01	12.03	9.25	19.14	12.84	9.33
ADHD Equation	10.61	8.13	5.37	N/A	N/A	N/A
AD Equation	N/A	N/A	N/A	14.37	8.19	10.20
HD Equation	N/A	N/A	N/A	8.66	11.83	6.70
Depression Equation	12.20	5.18	10.41	12.20	5.18	10.41
Obesity Equation	10.16	11.32	11.39	10.16	11.32	11.39
Smoking Equation	7.33	7.27	6.30	7.33	7.27	6.30
P-values from Overidentification Tests						
Full System	0.611	0.278	0.386	0.217	0.236	0.486
ADHD Equation	0.553	0.420	0.236	N/A	N/A	N/A
AD Equation	N/A	N/A	N/A	0.842	0982	0.440
HD Equation	N/A	N/A	N/A	0.845	0.812	0.266
Depression Equation	0.773	0.822	0.465	0.773	0.822	0.465
Obesity Equation	0.216	0.232	0.817	0.216	0.232	0.817
Smoking Equation	0.267	0.874	0.421	0.524	0.617	0.293

Note: First stage F statistics is computed from a joint test of significance of the full set of genetic instruments from individual first stage regressions that also include the full set of control variables included in the second stage. In each case, the Null is rejected at the 1% level. P-values are computed from Sargan tests of the joint null hypothesis that the excluded instruments are valid instruments for the health variables in the achievement equation

Table 7: Two Stage Least Squares Estimates of the Achievement Equation

	Full Sample	Females Only	Males Only	Full Sample	Females Only	Males Only
ADHD	0.218	-0.053	0.503	N/A	N/A	N/A
	(0.288)	(0.274)	(0.330)			
AD	N/A	N/A	N/A	-0.513	-0.455	-0.111
				(0.364)	(0.395)	(0.400)
HD	N/A	N/A	N/A	0.822	0.032	0.204
				(0.512)	(0.374)	(0.569)
Depression	-0.452**	-0.186	-0.322	-0.322**	-0.353**	-0.273
	(0.198)	(0.192)	(0.240)	(0.161)	(0.167)	(0.197)
Obesity	-0.450**	-0.500***	0.096	-0.460**	-0.470**	0.023
	(0.222)	(0.190)	(0.300)	(0.229)	(0.199)	(0.295)
Smoker in Home	-0.161***	-0.111***	-0.253***	-0.157***	-0.099***	-0.224***
	(0.032)	(0.033)	(0.050)	(0.030)	(0.033)	(0.046)
Age	0.495	0.632	0.315	0.567	0.678	0.541
	(0.517)	(0.690)	(0.769)	(0.510)	(0.713)	(0.719)
Age Squared	-0.016	-0.018	-0.012	-0.019	-0.020	-0.019
	(0.015)	(0.020)	(0.023)	(0.015)	(0.021)	(0.021)
Black	-0.279***	-0.232***	-0.285***	-0.288***	-0.259***	-0.308***
	(0.046)	(0.054)	(0.074)	(0.045)	(0.057)	(0.069)
Hispanic	-0.194***	-0.144**	-0.191***	-0.176***	-0.103	-0.191***
	(0.043)	(0.063)	(0.069)	(0.044)	(0.070)	(0.065)
Asian	0.173***	0.225***	0.050	0.166***	0.227***	0.041
	(0.040)	(0.045)	(0.067)	(0.039)	(0.048)	(0.068)
Parent is HS Dropout	-0.145**	-0.153**	-0.204**	-0.147**	-0.124*	-0.227**
	(0.059)	(0.065)	(0.103)	(0.060)	(0.072)	(0.096)
Parent is HS Grad	-0.215***	-0.234***	-0.169**	-0.211***	-0.247***	-0.195***
	(0.045)	(0.052)	(0.077)	(0.043)	(0.053)	(0.073)
Parent some College	-0.108***	-0.184***	-0.039	-0.104***	-0.163***	-0.050
	(0.033)	(0.044)	(0.049)	(0.034)	(0.049)	(0.045)
Biological Parent	0.428***	0.277***	0.512***	0.372***	0.312***	0.430***
	(0.073)	(0.107)	(0.115)	(0.074)	(0.109)	(0.108)
Constant	-1.259	-2.944	-0.120	-1.826	-3.217	-2.218
	(4.446)	(5.890)	(6.576)	(4.381)	(6.093)	(6.169)
N	2576	1366	1210	2576	1366	1210

Note: Corrected standard errors in parentheses. Regressions include parental age, parental age squared, parental gender, school and time period indicators. ***,**, * denote statistical ignificance at 1%, 5%, 10% level respectively.

Table 8: Two Stage Least Squares Estimates of the Achievement Equation where Years of Smoking is Treated as Exogenous

	Full Sample	Females Only	Males Only	Full Sample	Females Only	Males Only
ADHD	-0.646*	-0.672**	0.010	N/A	N/A	N/A
	(0.343)	(0.287)	(0.345)			
AD	N/A	N/A	N/A	-1.180***	-1.456***	-0.414
				(0.408)	(0.393)	(0.428)
HD	N/A	N/A	N/A	0.611	0.220	0.108
				(0.588)	(0.420)	(0.613)
Depression	-1.115***	-0.474**	-0.938***	-0.756***	-0.495***	-0.753***
	(0.230)	(0.208)	(0.229)	(0.176)	(0.186)	(0.192)
Obesity	-0.501*	-0.659***	0.290	-0.627**	-0.724***	0.192
	(0.287)	(0.209)	(0.324)	(0.263)	(0.219)	(0.317)
Smoker in Home	-0.082**	-0.073**	-0.192***	-0.100***	-0.069*	-0.182***
	(0.040)	(0.036)	(0.053)	(0.033)	(0.037)	(0.049)
Age	0.450	0.863	-0.162	0.590	0.862	0.137
	(0.668)	(0.767)	(0.831)	(0.586)	(0.803)	(0.771)
Age Squared	-0.015	-0.026	0.003	-0.019	-0.026	-0.006
	(0.020)	(0.022)	(0.024)	(0.017)	(0.024)	(0.023)
Black	-0.338***	-0.261***	-0.300***	-0.329***	-0.295***	-0.312***
	(0.059)	(0.060)	(0.080)	(0.051)	(0.063)	(0.074)
Hispanic	-0.173***	-0.057	-0.222***	-0.146***	0.008	-0.207***
	(0.056)	(0.068)	(0.075)	(0.050)	(0.076)	(0.070)
Asian	0.209***	0.231***	0.098	0.178***	0.195***	0.083
	(0.051)	(0.050)	(0.072)	(0.045)	(0.054)	(0.072)
Male	-0.386***	N/A	N/A	-0.328***	N/A	N/A
	(0.041)			(0.035)		
Parent is HS Dropout	-0.115	-0.119	-0.257**	-0.098	-0.045	-0.265*
	(0.077)	(0.072)	(0.112)	(0.068)	(0.080)	(0.103)
Parent is HS Grad	-0.270***	-0.281***	-0.183**	-0.236***	-0.261***	-0.194**
	(0.058)	(0.057)	(0.084)	(0.050)	(0.059)	(0.079)
Parent some College	-0.059	-0.153***	0.001	-0.067*	-0.118**	-0.018
	(0.042)	(0.049)	(0.052)	(0.039)	(0.055)	(0.049)
Biological Parent	0.415***	0.354***	0.411***	0.350***	0.362***	0.356***
	(0.095)	(0.119)	(0.123)	(0.085)	(0.123)	(0.115)
Constant	-0.690	-4.589	3.189	-1.864	-4.663	0.753
	(5.744)	(6.550)	(7.129)	(5.036)	(6.869)	(6.630)
N	2576	1366	1210	2576	1366	1210

Note: Corrected standard errors in parentheses. Regressions include parental age, parental age squared, parental gender, school and time period indicators. ***.**, * denote statistical ignificance at 1%, 5%, 10% level respectively.

Table 9: Two Stage Least Squares Estimates of the Achievement Equation Including A Subset of Health Outcomes

Included health states	Full Sample	Girls	Boys
ADHD	-0.054 (0.253)	-0.441* (0.250)	0.145 (0.256)
AD	-0.213 (0.396)	-0.085 (0.370)	-0.010 (0.378)
HD	0.515 (0.464)	-0.426 (0.332)	0.195 (0.552)
AD	0.026 (0.256)	-0.287 (0.324)	0.089 (0.254)
HD	0.327 (0.357)	-0.459 (0.301)	0.184 (0.366)
Depression	-0.484*** (0.191)	-0.788*** (0.252)	-0.270 (0.188)
Obesity	-0.311* (0.188)	-0.394** (0.168)	0.064 (0.283)
Observations	2576	1366	1210

Note: Corrected standard errors in parentheses. Each cell of the table corresponds to a separate regression. The dependent variable of the regression differs by row. Columns reflect different samples. Regressions include the non-health inputs in Table 7, school and time period indicators. ***,**, * denote statistical significance at 1%, 5%, 10% level respectively.

Appendix Table 1: Ordinary Least Squares Estimates of the Cigarette Smoker Equation

	Full Sample	Females Only	Males Only	Full Sample	Females Only	Males Only
ADHD	0.083 (0.065)	0.172 (0.097)	0.036 (0.087)	N/A	N/A	N/A
AD	N/A	N/A	N/A	0.143 (0.082)	0.299** (0.131)	0.106 (0.104)
HD	N/A	N/A	N/A	0.018 (0.074)	0.065 (0.103)	-0.026 (0.106)
Depression	0.051* (0.026)	0.006 (0.026)	0.116** (0.051)	0.048 (0.026)	0.001 (0.025)	0.113** (0.051)
Obesity	0.018 (0.049)	0.092 (0.081)	-0.093* (0.052)	0.018 (0.048)	0.094 (0.081)	-0.094* (0.051)
Smoker in Home	0.117*** (0.039)	0.183*** (0.059)	0.041 (0.047)	0.116*** (0.039)	0.181*** (0.059)	0.038 (0.047)
Age	-0.779* (0.415)	-0.193 (0.497)	-0.849 (0.633)	-0.789* (0.415)	-0.183 (0.497)	-0.862 (0.632)
Age Squared	0.024 (0.013)	0.006 (0.016)	0.026 (0.020)	0.024* (0.013)	0.006 (0.016)	0.027 (0.019)
Black	-0.032 (0.054)	0.002 (0.084)	-0.096 (0.071)	-0.030 (0.054)	0.006 (0.083)	-0.095 (0.072)
Hispanic	-0.082* (0.050)	-0.003 (0.093)	-0.174*** (0.053)	-0.086* (0.050)	-0.014 (0.094)	-0.176*** (0.053)
Asian	-0.101** (0.042)	-0.099* (0.056)	-0.106* (0.072)	-0.099** (0.042)	-0.093* (0.056)	-0.107* (0.072)
Male	0.043 (0.032)	N/A	N/A	0.041 (0.032)	N/A	N/A
Parent is HS Dropout	-0.015 (0.061)	0.001 (0.080)	0.021 (0.082)	-0.018 (0.061)	-0.011 (0.080)	0.024 (0.082)
Parent is HS Grad	0.140** (0.064)	0.146* (0.088)	0.141 (0.095)	0.139** (0.064)	0.146* (0.088)	0.141 (0.094)
Parent some College	-0.032 (0.035)	0.023 (0.051)	-0.096** (0.048)	-0.033 (0.035)	0.022 (0.050)	-0.095** (0.048)
Biological Parent	-0.283** (0.131)	-0.671** (0.288)	-0.127 (0.125)	-0.277** (0.131)	-0.674** (0.289)	-0.116 (0.124)
Constant	6.075* (3.295)	1.135 (3.911)	6.971 (5.077)	6.173* (3.294)	1.084 (3.909)	7.089 (5.070)
N	2576	1366	1210	2576	1366	1210
R squared	0.07	0.12	0.09	0.08	0.13	0.09

Note: Corrected standard errors in parentheses. Regressions include parental age, parental age squared, parental gender, school and time period indicators. ***.**, * denote statistical ignificance at 1%, 5%, 10% level respectively.

Appendix Table 2: Summary Statistics on GPA Performance by Health Disorder and Health Behavior

	Grade 10	**Grade 11**	**Grade 12**
Smokers	2.673 (0.661)	2.626 (0.715)	2.847 (0.688)
Non Smokers	3.233 (0.532)	3.202 (0.557)	3.232 (0.529)
T-statistic for Differences in Mean GPA by Smoking Status	8.388***	8.662***	7.278***
Depression Diagnosis	3.035 (0.617)	3.003 (0.647)	3.025 (0.665)
No depression Diagnosis	3.213 (0.552)	3.177 (0.583)	3.197 (0.554)
T-statistic for Differences in Mean GPA by Depression Status	3.416***	3.224***	2.921***
Obese	2.830 (0.620)	2.699 (0.729)	2.788 (0.623)
Non Obese (BMI <30)	3.215 (0.552)	3.187 (0.568)	3.208 (0.555)
T-statistic for Differences in Mean GPA by Obesity Status	5.453***	6.713***	5.883***
ADHD Diagnosis	2.929 (0.694)	2.919 (0.685)	2.919 (0.697)
No ADHD Diagnosis	3.200 (0.555)	3.163 (0.589)	3.193 (0.558)
T-statistic for Differences in Mean GPA by ADHD Diagnosis	3.263***	2.911***	3.492***
AD Diagnosis	2.714 (0.703)	2.733 (0.718)	2.754 (0.742)
No AD Diagnosis	3.203 (0.553)	3.166 (0.585)	3.195 (0.555)
T-statistic for Differences in Mean GPA by AD Diagnosis	4.921***	4.357***	4.713***
HD Diagnosis	3.155 (0.527)	3.054 (0.587)	3.047 (0.630)
No HD Diagnosis	3.185 (0.569)	3.151 (0.598)	3.181 (0.568)
T-statistic for Differences in Mean GPA by HD Diagnosis	0.285	0.937	1.379

Note: Most cells present the mean GPA and standard deviations in parentheses for individuals by health category ***,**, * denote statistically significant differences in mean GPA by health outcome at the 1%, 5%, 10% level respectively.

Appendix Table 3: Relationship Between Health Behaviors and Health Outcomes During Adolescence by Gender

FEMALES

Behavior	Total Number	Nothing Else	Also Smokes	Also AD	Also HD	Also Obese	Also Depressed
Wave 3, N=438							
Nothing	231	***	***	***	***	***	***
Smokes	33	13	***	4	3	6	7
AD	11	1	4	***	4	1	7
HD	13	3	3	4	***	1	6
Obese	34	19	6	1	1	***	9
Depression	81	59	7	7	6	9	***
Wave 4, N=453							
Nothing	237	***	***	***	***	***	***
Smokes	35	8	***	4	3	8	9
AD	13	2	4	***	4	2	7
HD	15	5	3	4	***	2	6
Obese	36	17	8	2	2	***	10
Depression	88	64	9	7	6	10	***
Wave 5, N=466							
Nothing	243	***	***	***	***	***	***
Smokes	64	30	***	7	6	10	7
AD	13	3	7	***	6	2	3
HD	15	4	6	6	***	2	4
Obese	35	11	10	2	2	***	5
Depression	56	41	7	3	4	5	***

MALES

Behavior	Total Number	Nothing Else	Also Smokes	Also AD	Also HD	Also Obese	Also Depressed
Wave 3, N=389							
Nothing	240	***	***	***	***	***	***
Smokes	39	23	***	3	1	1	8
AD	22	4	3	***	10	1	8
HD	16	5	1	10	***	1	4
Obese	34	22	1	2	1	***	8
Depression	58	34	8	8	4	8	***
Wave 4, N=402							
Nothing	240	***	***	***	***	***	***
Smokes	46	27	***	5	2	2	12
AD	24	5	5	***	13	2	7
HD	18	4	2	13	***	1	3
Obese	34	20	2	2	1	***	7
Depression	58	32	12	7	3	7	***
Wave 5, N=405							
Nothing	240	***	***	***	***	***	***
Smokes	62	30	***	8	5	5	10
AD	25	5	8	***	12	2	7
HD	20	4	5	12	***	1	5
Obese	32	17	5	2	1	***	5
Depression	51	25	10	7	5	5	***

Appendix Table 4: Two Stage Least Squares Estimates of the Achievement Equation by Subsample with Alternative Preferred instrument Sets

	Females Only	Males Only	Females Only	Males Only
ADHD	-0.201 (0.270)	-0.047 (0.274)	N/A	N/A
AD	N/A	N/A	-0.875* (0.447)	-0.024 (0.395)
HD	N/A	N/A	0.622 (0.402)	0.115 (0.550)
Depression	-0.446** (0.202)	-0.277 (0.199)	-0.461** (0.190)	-0.228 (0.197)
Obesity	-0.549*** (0.187)	0.380 (0.292)	-0.640*** (0.207)	0.235 (0.292)
Smoker in Home	-0.095*** (0.035)	-0.232*** (0.046)	-0.099*** (0.036)	-0.234*** (0.046)
Age	0.772 (0.735)	0.572 (0.741)	0.801 (0.759)	0.584 (0.734)
Age Squared	-0.023 (0.022)	-0.019 (0.022)	-0.024 (0.022)	-0.020 (0.022)
African American	-0.259*** (0.058)	-0.320*** (0.072)	-0.284*** (0.061)	-0.314*** (0.070)
Hispanic	-0.104 (0.066)	-0.167** (0.067)	-0.053 (0.075)	-0.174*** (0.066)
Asian	0.236*** (0.048)	0.023 (0.064)	0.207*** (0.052)	0.028 (0.068)
Parent is HS Dropout	-0.133* (0.069)	-0.283*** (0.101)	-0.078 (0.077)	-0.258** (0.097)
Parent is HS Grad	-0.249*** (0.054)	-0.176** (0.075)	-0.220*** (0.056)	-0.182** (0.074)
Parent some College	-0.153*** (0.046)	-0.039 (0.047)	-0.113** (0.051)	-0.046 (0.047)
Biological Parent	0.320*** (0.114)	0.405*** (0.110)	0.314*** (0.117)	0.425*** (0.109)
N	1366	1210	1366	1210

Note: Corrected standard errors in parentheses. Regressions include school and time period indicators. ***, **, * denote statistical significance at 1%, 5%, 10% level respectively.

Appendix Table 5: OLS and Two Stage Least Squares Estimates of the Impacts of Cigarette Smoking on Health Outcomes

	Full Sample	Females Only	Males Only	Full Sample	Females Only	Males Only
Two Stage Least Squares						
ADHD	-0.003 (0.302)	0.339 (0.361)	-0.151 (0.335)	N/A	N/A	N/A
AD	N/A	N/A	N/A	0.074 (0.366)	-0.401 (0.399)	1.133** (0.522)
HD	N/A	N/A	N/A	-0.069 (0.536)	0.628 (0.406)	-1.663** (0.768)
Depression	0.455* (0.240)	-0.540** (0.261)	0.824*** (0.297)	0.511*** (0.179)	-0.186 (0.188)	0.739*** (0.267)
Obesity	-0.338 (0.227)	-0.095 (0.243)	-0.353 (0.380)	-0.196 (0.237)	-0.050 (0.219)	-0.126 (0.405)
OLS						
ADHD	0.083 (0.065)	0.172* (0.097)	0.036 (0.087)	N/A	N/A	N/A
AD	N/A	N/A	N/A	0.143 (0.082)	0.299** (0.131)	0.106 (0.104)
HD	N/A	N/A	N/A	0.018 (0.074)	0.065 (0.103)	-0.026 (0.106)
Depression	0.051* (0.026)	0.006 (0.026)	0.116** (0.051)	0.048* (0.026)	0.001 (0.025)	0.113** (0.051)
Obesity	0.018 (0.049)	0.092 (0.081)	-0.093* (0.052)	0.018 (0.048)	0.094 (0.081)	-0.094* (0.051)

Note: Corrected standard errors in parentheses. Each cell contains information on the impact of smoking on a health outcome from a regression that also controls for all the factors listed in Table 7, genetic markers, school and time period indicators. *, **, *** denote significance at 1%, 5%, 10% level respectively.

Appendix Table 6: Two Stage Least Squares Estimates of the Cigarette Smoker Equation

	Full Sample	Females Only	Males Only	Full Sample	Females Only	Males Only
ADHD	-0.003 (0.302)	0.339 (0.361)	-0.151 (0.335)	N/A	N/A	N/A
AD	N/A	N/A	N/A	0.074 (0.366)	-0.401 (0.399)	1.133** (0.522)
HD	N/A	N/A	N/A	-0.069 (0.536)	0.628 (0.406)	-1.663** (0.768)
Depression	0.455* (0.240)	-0.540** (0.261)	0.824*** (0.297)	0.511*** (0.179)	-0.186 (0.188)	0.739*** (0.267)
Obesity	-0.338 (0.227)	-0.095 (0.243)	-0.353 (0.380)	-0.196 (0.237)	-0.050 (0.219)	-0.126 (0.405)
Smoker in Home	0.100*** (0.033)	0.198*** (0.037)	0.005 (0.051)	0.093*** (0.029)	0.188*** (0.032)	-0.037 (0.055)
Age	-0.548 (0.492)	0.092 (0.751)	-0.041 (0.796)	-0.556 (0.495)	-0.065 (0.673)	-0.013 (0.869)
Age Squared	0.018 (0.014)	-0.003 (0.022)	0.002 (0.023)	0.018 (0.014)	0.002 (0.020)	0.002 (0.026)
Black	-0.006 (0.046)	-0.052 (0.061)	-0.091 (0.075)	-0.003 (0.044)	-0.032 (0.055)	-0.087 (0.081)
Hispanic	-0.058 (0.041)	0.055 (0.074)	-0.126 (0.072)	-0.064 (0.043)	0.058 (0.071)	-0.134 (0.078)
Asian	-0.137** (0.038)	-0.095 (0.050)	-0.164* (0.068)	-0.136** (0.038)	-0.122** (0.046)	-0.224** (0.082)
Male	0.087* (0.035)	0.000 (0.000)	0.000 (0.000)	0.092** (0.031)	0.000 (0.000)	0.000 (0.000)
Parent is HS Dropout	-0.004 (0.058)	0.048 (0.072)	0.048 (0.110)	-0.024 (0.059)	0.057 (0.071)	0.092 (0.120)
Parent is HS Grad	0.147** (0.044)	0.151** (0.058)	0.145 (0.078)	0.145** (0.042)	0.157** (0.051)	0.196* (0.087)
Parent some College	-0.061 (0.035)	0.102 (0.053)	-0.132* (0.053)	-0.071* (0.036)	0.078 (0.053)	-0.116* (0.056)
Biological Parent	-0.289** (0.070)	-0.619** (0.118)	-0.085 (0.117)	-0.291** (0.072)	-0.653** (0.103)	0.042 (0.130)
Constant	4.170 (4.237)	-0.795 (6.397)	0.653 (6.745)	4.138 (4.266)	0.235 (5.738)	0.044 (7.419)
N	2576	1366	1210	2576	1366	1210
R squared	5	5	5	5	5	5

Note: Corrected standard errors in parentheses. Regressions include school and time period indicators. *, **, *** denote statistical significance at 1%, 5%, 10% level respectively.