The Impact of Health on Academic Performance: New Evidence Using Genetic Markers

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Abstract

The causal effect of health on education outcomes remains an open question in economics since unobserved factors may explain the correlation between health and education. Using a very rich data set that tracks 900 students through high school and an instrumental variables strategy we estimate the causal impact of obesity, ADHD and depression on adolescent academic performance by exploiting variation in genetic markers. Our data contains direct genetic measures of the DRD2, DAT and CYP2B6 loci, which are well known in the scientific literature to affect the health outcomes, considered in our study. Further, we account for the endogeneity of smoking which may serve as an antidepressant and appetite suppressor. We present evidence that these genetic markers and their interactions are valid instruments with good statistical properties. Second, we find there is substantial heterogeneity in the effects of health on academic performance. The academic performance of female students is strongly negatively affected by poor physical and mental health outcomes.

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Comments Welcome

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1 Introduction

Research on the relationship between health and education has primarily been conducted in the psychology, education and public health literature which generally report that students who are obese or depressed perform poorly relative to their classmates. Since factors other than health are responsible for this repeatedly observed, but potentially spurious association, one can not credibly claim that obesity and depression have a deleterious effect on student performance in schools. Empirical researchers in this area must overcome the inherent endogeneity when considering health and education. Put in more general terms, how does one know whether smarter kids are better equipped to learn how to keep themselves healthy, or healthier kids are able to concentrate better in school.

This paper exploits recent findings from the neuroscientific literature, which have identified candidate genes, or genetic markers that are associated with specific diseases and health behaviors to help identify the causal impact of health on academic outcomes. The importance of genetic factors to behavioral characteristics and health outcomes has been noted throughout recorded history and the passage of physical and disease traits from parents to offspring was first explicitly studied and modeled by Gregor Mendel in the 18th century. Since this work more sophisticated studies of laboratory animals as well as comparisons between monozygotic and dizygotic twins demonstrated that behavioral characteristics and economic as well as health outcomes were in part linked to genetic inheritance. The importance of genetics as a predictor of health behaviors and outcomes is substantial. Cutler and Glaeser (2005) present evidence that the association between genetic factors and health is substantial. By comparing the correlation of health behaviors between monozygotic and dizygotic twins they conclude that 72% of the variation in obesity and 30% of the variation in cigarette smoking is due to genetic factors.

While there are a number of theoretical models that link education and health in the economics

literature, there is a lack of empirical evidence in this area on the impacts of health on education.[1] The most popular method is the use of instrumental variables to mitigate the correlation between unobservables and education (or health) variables. For example, Behrman and Lavy (1998) use prices to instrument for health and find the impact of child health on cognitive achievement varies as a function of the assumptions made concerning parental choices. Glewwe and Jacoby (1995) use similar market instruments and find evidence that much of the impact of child health on school enrolment proxies for unobserved variables. However, the statistical properties including the weak instrument problem employed in these studies are of debate. With data from developed countries a large literature has examined the impacts of early health status on subsequent academic outcomes,[2] but to the best of our knowledge there does not exist a study which attempts to estimate the causal impact of adolescent health on contemporaneous academic performance.

We examine the experience of a cohort of adolescents that are followed from grade 10 to grade 12. We are provided with clinical measures of obesity and psychiatric diagnoses of ADHD, AD (inattention by itself), HD (hyperactivity or impulsivity by itself) and depression. In addition we account for the endogeneity of adolescent smoking decisions since the nicotine in cigarettes may be used as a form of medication against mental illnesses or serve as an appetite suppressor affecting health outcomes. To identify the impacts of these health behaviors and outcomes, we focus on genes that are located in regions of brain responsible for reward and pleasure. This region is distinct from those that are known to process and retain knowledge. Evidence that different parts of the brain are associated with economic decisions has been found using functional magnetic resonance imaging. McClure, Laibson, Loewenstein and Cohen (2004) have shown in making intertemporal choices, different regions of the brain are activated (or correlate) with different activities. In this paper,

[1] The majority of research focuses on the effect of education on health. See Grossman and Kaestner (1997) for a recent review of that literature.

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

we present strong evidence from first stage regressions and overidentification tests that a set of candidate genes are both strongly associated with the respective health behaviors and disorders and independent of unobserved factors that affect academic achievement.

It is worth stating explicitly that the goal of this analysis is not to report a causal link between genes and health broadly defined. We exploit the strong neural correlations between a set of genetic markers and certain behaviors and 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 that are often quickly refuted by medical authorities. As we outline in greater detail in the next section, our identification is based on a large body of evidence in several fields that explain the operation of a neurological reward system. The abundance of evidence in the biomedical literature that presents a significant association between certain genes in this system with particular health behaviors and health status such as smoking, alcohol usage, obesity, ADHD, depression, schizophrenia can not be denied.

Since these genetic markers are assigned at conception, they are predetermined to any outcome including those that occur during pregnancy and at birth. Genetic markers are truly what is meant by nature. Using this "nature filter", the health variables being instrumented will be isolated from the nurture influences or choice-based inputs such as schools parents choose for their kids, neighbourhoods 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 variation in health variables is due only to the exogenous variation due to differences in genetic code it is much less likely to be correlated with the environment. We are also implicitly making an assumption that the genetic markers we employ as instruments are independent from genetic factors that associate with either innate ability or the development of intelligence. While this assumption may appear strong, it is worth pointing out that even after decades of research in the scientific community the biomedical literature continues to support the validity of this assumption.

Our analysis leads to four major conclusions:[3]

1) Genetic markers show a great deal of promise as a set of instrumental variables. The genetic markers and the two by two polygenic interactions that we consider are strongly associated with each health behavior and status in the study. In addition, statistical tests demonstrate that the markers only affect academic performance through the health outcomes.

2) We find that the impact of poor health behaviors on academic achievement to be substantial. Depression and inattention both lead to a 0.5 point decrease on GPA which is roughly a one standard deviation reduction. However, there is substantial heterogeneity in the impact of health on academic performance across gender. Male students do not suffer any adverse impact from any of the four health outcomes. In contrast, the academic performance of female students is strongly negatively affected by poor physical and mental health outcomes.

3) In explaining 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 larger impacts of health on education. Cigarette smoking is endogenous and we find that accounting for this choice reduces the negative impact of inattention and ADHD. This is consistent with the hypothesis that for individuals with limited attention spans there is an immediate academic benefit or compensation from cigarette smoking.

4) The presence of high comorbidity of health outcomes such as medical disorders is striking. 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 AD/HD have one or more comorbid conditions such as depression. Unless the exogenous genetic and 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. In our analysis, we estimate a large and significant positive impact for obesity in males and inattention for the full sample when

[3]It is important to note that the population from which our sample was drawn is not representative of the United States and may only extend to schools in that particular county.

we do not account for the full health vector. Further, the significant impact of hyperactivity changes signs when one controls for the full vector of health states. Since many individuals suffer from more than one disorder ignoring related illnesses may lead to some misleading conclusions.

The rest of the paper is organized as follows. In Section 2, 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 3. We describe a model that guides our understanding of how education and health interact in adolescence in section 4. We note that restrictions must be placed on how parents' evaluate the trade-off between inputs that will improve children's health and education, which is often ignored in the literature. Our identification strategy and estimating equations are also presented in this section. Our results are presented and discussed in Section 5. A concluding section summarizes our findings and discusses directions for future research

2 Primer on Scientific Evidence Relating to Genes to Health Behavior

Empirical researchers in the social sciences have traditionally chosen to ignore or, at best, consider fixed either over time or across siblings including twins the unobserved heterogeneity conferred by variation in genetic inheritance. In the past, approaching inherited predispositions in this manner was necessary since data that correlated genetic markers with specific behaviors or outcomes did not exist. Due to recent advances in fields of molecular and behavioral genetics, these perceived limitations no longer hold.

The discovery of the structure of DNA (Watson and Crick (1953) enabled researchers to make great strides in understanding the biological underpinnings of health outcomes and behaviors most notably through the decoding of the human genome (Venter et al., 2002). While these advances have allowed researchers to find the genetic code for a number of inherited traits and diseases such as eye

color, cystic fibrosis, and Huntingdon's disease,[4] most products of inheritance have been found to be caused by the interaction of numerous genetic markers, or polygenic. The health outcomes and behaviors we consider are thought to be polygenic with researchers associating approximately 160 and 42 genes with obesity (Perusse, et al. 2005) and ADHD (Comings et al. (2000) respectively.

The decoding of the human genome has led behavioral geneticists to elucidate how differences in the genetic code add up to differences in behaviors across individuals. One of the first areas that behavioral geneticists focused on was the genes involved in a pathway in the brain that regulates basic drive and reward among higher mammals including humans. This pathway is referred to as the reward pathway or pleasure center because it is closely linked to primal drives such as feeding and sex, and has been shown to have a powerful affect on decision making. 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 lessen the stimulation of their pleasure center.[5]

Since the reward system of the brain has been closely linked to a number of problematic traits in humans such as addiction and depression, behavioral geneticists have focused their attention on how addictive processes work as well as how variation in different components of the pathway might make an individual more predisposed to addiction. In general, a region of the brain known as the ventral tegmental area (VTA) is activated when activities such as feeding or sex are undertaken. The neurons (brain cells) in the VTA release signaling molecules known as neurotransmitters (in this case dopamine[6]) to another area of the brain known as the nucleus accumbens (NA). This signal is passed between neurons through junctions called synapses until it eventually reaches the frontal cortex, where most "decisions" are made. Increases in the synapse of either neurotransmitters or

[4]HD Collaborative Research Group, 1993.

[5]Kandel and Schwarz (2004) provide an excellent background to these and other topics related to the neurobiological underpinnings of behavior

[6]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.

receptor neurons for them allow for a much stronger signal to be sent.

For humans, certain food and drugs can have an especially powerful effect on the reward center of the brain. These drugs, such as nicotine or caffeine, can mimic or potentiate the effects of neurotransmitters that occur there naturally. For example, nicotine strengthens the excitatory connections between the neurons that make dopamine in the VTA resulting in the release of more dopamine into the reward center.[7] This process has been described as a molecular "hijacking" of the reward pathway, since it appears to serve no adaptive purpose and only brings about pleasure. Since the response of these neurons to nicotine 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.

Based on data from the human genome project, behavioral geneticists have targeted markers that code for various steps along the dopaminergic reward pathway. Differences in DNA sequences, also called polymorphisms, have been closely studied to assess whether heterogeneity in polymorphism inheritance across subjects was correlated with variation in levels of pleasure or reward they received from various stimuli, as exhibited by addiction to tobacco, etc.[8]

In order to conceptualize the structure and implications of our estimation strategy, it is important to explain some of the syntax relevant to genetic marker inheritance. Each person inherits 2 copies of the same marker, known as an allele. Alleles are found in highly specific and small areas of the genome called loci (which, for our purposes, can be considered interchangeable from the term 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. There can be 2 (or more) alleles for the same marker. If you inherit 2 of the same allele, you are considered to be homozygous

[7] Nicotine has been shown to increase levels of synaptic dopamine by stimulating dopamine release (Di Chiara and Imperato, 1988) and inhibiting dopamine reuptake (Carr et al., 1992).

[8] Because the VTA-NA pathway is important in regulating pleasure and, therefore, emotion, a number of behavioral traits including depression and ADHD have been linked to this pathway.

for that marker. If you inherit 2 different alleles, you are considered to be homozygous for that particular marker. Therefore, if the alleles A and B exist for a given marker, you can be homozygous by being AA or BB or heterozygous by being AB or BA.[9]

The particular genetic markers included in this study were chosen based upon a large and growing body of research showing a correlation between their variation and traits such as smoking behavior and depression, controlling for other relevant factors. These markers include the, i) Dopamine Receptor D2 locus (DRD2), ii) SLC6A3 locus (DAT), iii) Tryptophan hydroxylase locus (TPH) and iv) CYP2B6 locus (CYP).

The DRD2 gene is believed to code for the number of D2 dopamine receptors on neurons in the brain, including those in the VTA. 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.[10] Since individuals have fewer D2 receptors it is postulated that, relative to individuals with two DRD2-A2 alleles, those with DRD2-A1 alleles (A1/A1 or A1/A2) are associated with compulsive and addictive behaviors including smoking, depression and obesity.[11]

The SLC6A3 gene encodes a protein that regulates synaptic levels of dopamine in the brain by coding for a reuptake protein called the dopamine transporter, DAT.[12] The SLC6A3 gene has been implicated in Parkinson's disease , attention deficit disorder, and Tourette's syndrome.[13] Variability in the length of the DAT gene is believed to influence levels of DAT protein in the brain. The

[9]Note that being AB or BA is meaningless, as one is still heterozygous regardless. Therefore, three groups can be compared in estimation, homozygous AA, homozygous BB, and heterozygous.

[10]This finding was first reported in Blum et al. (1991). See Sibley and Monsma (1992) for a detailed discussion of dopamine receptors.

[11]See Audrain-McGovern (2004) and Epstein et al. (2002) and the refererences within for evidence on these associations.

[12]Bannon, Granneman, & Kapatos, 1995

[13]See Seeman & Niznik (1990), Cook et al. (1995) and Comings et al. (1996) for evidence of each of these associations.

majority of individuals have SLC6A3 alleles 9 or 10 base pairs long of which the length is positively associated with levels of DAT protein.[14] For example, individuals with at least one copy of the 9-repeat allele (variant) of SLC6A3 have diminished dopamine reuptake and greater availability of synaptic dopamine. In contrast, individuals with SLC6A3-10 genotypes are associated with lower levels of endogenous synaptic dopamine due to greater reuptake. These individuals receive greater benefits from exogenous substances such as nicotine that stimulate dopamine transmission.

The tryptophan hydroxylase gene (TPH), a member of the serotoninergic 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.[15]

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 the CYP2A6 gene in particular have been linked to across population differences to smoking, alcoholism, and response to anti-depression medications . Population studies find high numbers of individuals with the CYP2B6 CC genotype (homozygous for the wild-type allele) relative to the number of people with either the CT or TT genotype.[16]

The descriptions above not only provide an understanding of how the individual genetic markers might influence behavior, they also allow the reader to understand how different allelic combinations, when interacted, can have powerful effects. For example, consider two individuals with the SLC6A3

[14]The length is associated with the number of vtr. Note the SLC6A3 loci may also take the form of 7- repeat, 8-repeat, 11-repeat or 12-repeat which are extermely rare in both the population and our sample.

[15]Lucki (1998) presents evidence of these associations. It is worth noting that recent research (Walther, et al 2003) has indicated that the locus included in our dataset codes for an isoform of Tryptophan hydroxylase (TPH1) that is located primarily outside of the CNS. It is likely that the second isoform of TPH (TPH2) which is expressed in the CNS would have an even stronger association with the conditions we are considering.

[16]See Lerman et al. (2001) or Lerman et al. (2003) for a discussion.

9-length repeat that differ in their DRD2 geneotype (A1/A1 vs. A2/A2). Individuals that have both the DRD2 A1 allele and the SLC6A3 9-length repeat could have both more dopamine to excite the neurons of the reward center as well as more receptors to bind to it, thus increasing neuronal activation exponentially. 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 questionnaires given over four years of high school (1999-2003) along with the four genetic markers described in the preceding section. The primary aim of the study was to evaluate prospectively the contributions of specific genetic factors to the adoption of smoking.

The study began in 1999 when researchers selected five high schools from the same county in Northern Virginia.[17] The county contains over 950,000 residents and is one of the most affluent in the US with a median household income of $70,000 in 1995.[18] School administrators provided the names and mailing addresses of the complete 9th grade class roster of students for each of these schools. To recruit study participants project information packets which included an explanatory cover letter from the school principal, consent forms, and a brief demographic/response form were mailed to 2120 students' homes.[19] To increase participation rates, up to three waves of mailings

[17] A total of 21 high schools existed in these counties. Using data from the NCES CCD we did not find any significant differences between particpating and non participating schools in student demographics at grade level or standard school input measures.

[18] The average household income that is twice that of the nation and only 8.7% of households had incomes below $25,000 in 1995.

[19] 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

were sent and telephone calls were placed to encourage parents to respond. While seventy-two percent of the parents/guardians (1533 of 2120) responded to the mailings, 75% (1151) provided written consent for their adolescent to participate in the study. Of the 1151 adolescents who had parental consent to participate, 99% provided assent.[20]

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. 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, full genetic information was obtained for 1032 subjects.

The GATOR study also contains basic information on demographic characteristics (i.e. race, gender, etc.), academic performance as measured by GPA (waves 3-5 only), reports on physical

total population were excluded.

[20] Audrain et al. (2002) report that caucasian parents were more likely to give consent than other races and among Caucasian parents higher levels of educationw ere associated with greater consent. Finally, we compared the percentage of students who consented with the remaining school population using data from the NCES Common Core of Data and found no significant differences in race or gender.

[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 μM; Pharmacia, Piscataway, NJ) in 50-μl total volume. The reaction conditions included an initial melting step (94^oC; 4 min) followed by 35 cycles of melting (94^oC; 1 min), annealing (65^oC; 1 min), and extending (72^oC; 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 of these assays have been validated by confirming a polymorphic inheritance pattern in seven human family lines that encompassed three generations.

activity and information on smoking activity by family and residence members. In the initial survey this information was collected during mandatory grade 9 health and physical education classes. These surveys were administered by a GATOR staff member to students who provided assent. Participants received $5 gift certificates to media stores to acknowledge their time and participation in this study.

The participants were resurveyed in the fall and spring of the 10th grade and in the spring of the 11th and 12th grade, for a total of five data collection waves. The rates of participation at the follow-ups from baseline were about 95%, 96%, 93% and 89% respectively. Similar to the initial data collection, surveys were completed during a classroom common to all students in the presence of a member of the research team.[22]

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 sent to the homes of students who had either switched schools or dropped out of school.

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

[22] 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.

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 symptoms. 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 providing 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 females, > 22 for males) 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 used to assess symptoms of Attention-deficit/hyperactivity disorder (ADHD) from DSM-IV (Barkley and Murphy, 1998) in the second wave survey.[23] 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 >=6 symptoms on either the inattention or hyperactivity-impulsivity subscale) was used to determine the likely presence or absence of clinically significant ADHD symptoms.[24] 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).

In total we have information on academic performance as measured by GPA (collected in waves

[23]The American Psychiatric Association defines ADHD as a heterogeneous neurobehavioral syndrome that begins in childhood and is applied to individuals who display developmentally inappropriate levels of attention problems or hyperactivity-impulsivity, along with impairments in functioning at home, school, or in social settings.

[24]This scoring algorithm is described in Barkley and Murphy (1998).

3-5 only), 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 information on 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% and12.20% and 5.54% to 19.3% respectively. The overall sample's subscale averages fell within normal limits (inattention mean = 5.9; hyperactivity-impulsivity mean = 6.6) 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.

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 statistics over these three years. In contrast, the number of individuals who currently smoke and have tried smoking rises rapidly during this period. Yet, the percentage of daily smokers in the 10th grade and 12th grade is similar to national averages calculated using the NELS88 (Miller, 2005). The percentage of depressed adolescent in our sample is slightly higher than estimates of 12.5% of the adolescent population being diagnosed as clinically depressed from the U.S. Department of Health and Human Services. In our empirical analysis, we will make use of one year information on depression and smoking so that our measures are predetermined and summary statistics on lagged smoking and depression are included. Similar to our ADHD measure, we need to use predetermined variables since once could postulate that the answers from the psychological questionnaires used to diagnose these conditions could be influenced by current academic performance.

Summary information on the genetic markers in our data are provided in Table 2.The SLC6A3 genotype was classified as the number of 10-repeat alleles (zero, one, or two), DRD2 is classified as A1/A1 or A1/A2 or A2/A2. In our analysis we include indicator variables for the available AA AC and CC genotypes of the TPH gene. Finally, we include indicator variables for the available CC,

CT and TT genotypes of the CYP gene. The first column provides the raw number of individuals who possess each particular marker. The entries in the remaining columns indicate the number of people in each row that also possess one of . Notice that each cell contains a minimum of two people. Further, there does not appear to exist any systematic relationship between the different genes. thus, having a rare polymorphism for one gene does not make it appear more likely that you would have a rare polymorphism. This suggests that coding for polygenic combinations is likely to be extremely important in our empirical analysis.

A major limitation of this data is a lack of information on the students' family, neighbourhood and school. Data at the school level can be obtained from the CCD and we could recover some neighbourhood information from US census records at the zip code level. In terms of the family we only have information on smoking patterns and smoking history within the household and across a complete set of family members. 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

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 (from three distinct fields) that attempt to 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. Psychologists claim that the heterogeneous health behaviors arise from the different environment or situational factors that different individuals encounter. Natural scientists hypothe-

size 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 regular smoking, drinking alcohol or using narcotic drugs in the first stage given their demographics, risk preference, genetic markers and home and school environments as well as their current health status. This model could also flexibly include the selection of health enhancing behaviors such as proper diet and regular exercise. Child 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 costs to child i. The immediate satisfaction that child i derives from k is affected by child i's current health status (i.e. kids with ADHD may find that smoking helps them concentrate) and their genetic predispositions (some kids receive more neurological rewards from smoking than others). The current cost of k is the immediate cost of taking action k; this cost includes both pecuniary (price) and nonpecuniary parts. For example, the cost includes the difficulty level (time spent could be one indicator) of taking action k (acquiring cigarettes as a teenager or narcotic drugs is not straightforward), which is affected by the neighbourhood / school / family environment / input to child i. For example, increased parental monitoring might make cigarette smoking more costly / difficult; a drug infested neighbourhood might make drug usage less costly / difficult. The perceived future costs usually depend on the discount factors and value of life, which may be influenced by current health status (healthy people are more patient) and/or genetic predispositions. Notice that here we assume a non-binding monetary budget constraint for ease of exposition. As a result child 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 family / school / neighbourhood environments which 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_{iT}^{k} captures an independent random shock. We can easily generalize this model to account for a set of risk/health-enhancing behaviors by treating k as a vector of those behaviors.

Stage 2, at time T_1, altruistic parents select a level of health input l (i.e. health insurance coverage) for child i, given the observed child i's health behaviors $\tilde{K}_T$and revealed health status H_{iT-1}that provides the highest indirect utility for their household V_{il}^{*},

$$V_{iT}^{l} \equiv V_{iT}(X_{2i}, C_{lT}, H_{iT-1}, \tilde{K}_T, G_i^H), \text{ for each } l \text{ available to child } i \tag{2}$$

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.

The set of genetic and home and school environments that impact health outcomes are not identical from those that determine health behaviors. Given the history of health behaviors chosen by child i and the health inputs chosen by child i's parents, health production functions translate these elements into a vector of health outputs as follows

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

where $X_{2iT}...X_{2i0}, k_{iT}...k_{i0}$ and $\epsilon_{iT}^{H}...\epsilon_{i0}^{H}$ are the full history of individual specific, health behaviors 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 the extreme cases that parents have to choose between putting enough food on the table and paying the kid's medical/insurance bill. Since our data has

little health inputs information, this assumption puts few constraint on the estimation equations. Under these two assumptions, parents will always choose l* that leads to the highest possible level of Hit.

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 knowledge as measured by a score on an achievement test or report card:

$$A_{ijT} = f(X_{iT}^e ... X_{i0}^e, Q_{j_T} ... Q_{j_o}, H_{iT,} I_{i,} \epsilon_{iT} ... \epsilon_{i0}) \tag{6}$$

where $(\epsilon_{iT} ... \epsilon_{i0})$ are the full history of independent random shocks. Notice the full history of school and personal inputs in addition to the current health status affects current academic achievement.

There are three popular explanations put forth in the health economics literature for the 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 as both health status and

education are directly related to an unobserved variable such as time discounting (Fuchs (1982)) or one's upbringing (Rosenzweig and Schultz, 1983). Our model allows for the latter two explanations. Since we have data on adolescents of the same grade, the timeline does not support the first model. However, parental education level may enhance the production of child health at this stage. We do not consider this possibility due to lack of data on parents.

There are several scenarios under which health status ($H_{iT,}$) can serve as an educational input. First, it affects the physical energy level that a child has 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 may cause low self esteem which leads to classroom disengagement that may reduce academic performance. Other health status such as being diagnosed with ADD 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 the health input (both physical and mental) in the education process while the last scenario influences a child's education outcome through other input 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 being obese on GPA from that which is due to the impact of the environment on GPA resulting from obesity. lily's three pages. 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 if the response of these individuals 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 investment exploring a structural model of parental investment in to education and health based on their child's quality is beyond the scope of this paper.

4.2 The Estimating Equations

Linearizing the above achievement relationship (equation 6) yields

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

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 in its contemporaneous version as

$$A_{ijT}=\beta_0+\beta_1X_{iT}+\beta_2H_{iT}+\beta_3Q_{jT}+\tilde{\epsilon}_{iT} \qquad (8)$$

where the vector X contains individual characteristics (gender, race, residential smoking status), the vector H is a vector of variables that captures current predetermined health measures. The implied restrictive assumption for this type of model is that the effect of all previous observed and unobserved influences are zero in the current period.[25]

Similarly we linearize and generate contemporaneous equations for both the health production function in equation (3) and the decision to engage in health behavior equation (1) as follows:

$$H_{iT} = \gamma_0+\gamma_1X_{iT}+\gamma_2k_{jT}+\gamma_3G^H_i+\tilde{\epsilon}^H_{iT} \qquad (9)$$

$$k_{iT} = \delta_0+\delta_1X_{iT}+\delta_2H_{iT}+\delta_3G^k_i+\tilde{\epsilon}^S_{iT} \qquad (10)$$

[25]Due to the limitation of almost all education data sets, this assumption is adopted for practical reasons and only recently has been tested (Ding and Lehrer (2005, Todd and Wolpin (2005) who present evidence that the assumption may not hold for family inputs.

Instrumental variable methods are used to estimate the above system of equations ((8) - (10)) to generate estimates of the causal impact of health on education (β_2). This provides consistent estimates of β_2, γ_2 and δ_2. Our identification relies on the assumption that the vectors of genetic markers that impact health behaviors (G_i^S) are unrelated to unobserved components of equation (9). In addition, the markers that affect health outcomes (G_i^H) are unrelated to unobserved components in equations (8) and (10). While there is absolutely no evidence for the former assumption that the markers considered in this study have any impact on intelligence, it remains possible. However, it is doubtful that the markers in either (G_i^H) and (G_i^S) proxy for the genetic determinants of intelligence included in the vector I_i. Recall, from Table 2 there was no evidence for a systematic relationship between the four different genes considered in this study that are involved in the reward mechanism.

5 Results

To justify our four sets of genetic markers and two by two polygenic actions to explain health behavior and status we begin by examining whether are there differences in academic and health measures between individuals with different genetic markers. Table 3 presents some summary information on the educational performance and health outcomes by genetic marker. Notice there are no substantial differences in academic performance between each marker for all four genes. However, there are substantial differences in health outcomes and behaviors by genetic markers. Individuals with the AA polymorphism of the TPH gene have higher propensities for smoking and obesity. Among the CYP gene, those with the rare TT polymorphism are more likely to be diagnosed with inattention (AD) and hyperactivity (HD). While those with the more common CC marker are more likely to be obese. Among the DRD2 gene, individuals with the common A2A2 are substantially less likely to be diagnosed as depressed or obese relative to the other DRD2 markers. Finally, among the DAT gene, individuals with only one 10-repeat (DAT1) are more likely to be diagnosed with ADHD and less likely to be diagnosed with depression relative to the other DAT

markers. Individuals that have no 10- repeats (DAT0) are associated with slightly higher smoking rates. These results suggest that the four sets of genetic markers are likely to have some correlations with the health measures that we consider and will not directly impact academic performance.

We next examine whether are there strong associations between educational performance and our health measures. Our data is consistent with literature in psychology, education and public health in that on average individuals who engage in negative health activities or have been diagnosed with a negative health outcome have a significantly lower GPA (one sided t-tests). On average, individuals with ADHD, depression and obesity respectively have GPA scores that are 0.26, 0.18 and 0.43 lower than their counterparts. The raw GPA gap of individuals with ADHD and obesity increases between grades 10 to 12 by approximately 20%. While the gap of depression children does not vary across 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 numerous individuals start smoking over time. These new smokers have substantially higher GPAs than long-term smokers. Between grade 10 and grade 12 long-term smokers constantly have GPAs that are approximately one half point lower relative to their classmates.

A major challenge in medically treating these health behaviors and conditions is the presence of comorbid conditions. Comorbid conditions, or comorbidities, are conditions that happen to occur at the same time. For example, seventy percent of adults with AD/HD are treated for depression at some point in their life. Table 4 presents some summary information on the presence of comorbordities in our sample. Column 1 of Table 4 presents the number of individuals 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 who also engage in other health behaviors or suffer other poor health outcomes. Notice that many individuals who are diagnosed with ADHD also choose to smoke and suffer from depression. On average individuals with ADHD are more likely to smoke, be diagnosed as either clinically depressed or obese than their schoolmates (one sided t-tests). The significant differences exist for each health outcome and behavior.

Since health disorders or negative health behaviors are more common among individuals diagnosed with one particular disorder than among the remaining population one would need to control for comorbidities to ensure that one disorder is not proxying for another. In our analysis we consider two different health state vectors when estimating equation (8). The first health vector includes depression, obesity and ADHD. In the second health vector we decompose the diagnosis of ADHD into being clinically inattentive or clinically diagnosed as hyperactive or impulsive and also include depression and obesity.

In order to determine whether there is a sizeable association between genetic markers and our health measures it remains open as to how to select an appropriate set of markers for each outcome/behavior. The scientific literature provides some (arguably weak) guidance but much of the evidence is conflicting primarily due to the use of small unrepresentative clinical samples.[26] In preparing this draft of the paper we considered eight different instrument sets for the equations. One set involved the use of the complete set of the markers in our study,[27] another set was constructed based on our reading of the neuroscientific literature up to May 23, 2005 and the remaining six sets were constructed from stepwise estimation using alternative selection criteria. We present and report results from instruments selected by forward stepwise estimation for each health outcome and behavior at the 5% level. We selected this set not since it will have good first stage properties for the full sample by design but rather since it is more parsimonious than the other instrument sets we used to verify the robustness of our findings. It is crucial to stress that whichever of these eight sets that we use the findings for the full sample and sub sample of females does not vary substantially either qualitatively or quantitatively.[28] We do not vary the instrument set across race

[26] 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 behavior.

[27] We used all the markers with the exception of A1/A1 for the health outcomes and used A1/A1 to identify health behavior.

[28] For the subsample of males there were some minor differences with several of the other instrument sets and numerous failures of overidentification tests with two of the other instrument sets.

or gender so that any observed difference in terms of health effects is not the result of the selection of different instrument sets.

Ordinary least squares estimates of equations (8) are presented in the top panel of Table 5.[29] The first column presents the results for the full sample where we include ADHD as a health outcome. Notice that the impact of each health behavior is negatively and significantly associated with academic performance. The negative impact of obesity is nearly twice the size of the other health behaviors and indicates that on average obese individuals have a GPA 0.37 points lower. The magnitude exceeds that of any race or family variable. Results that correspond to the sample of females and males alone are presented in columns two and three respectively. Notice that the impact of obesity on GPA for females is large quantitatively and highly significant. Obese females are associated with a half a point decrease in their GPA, an effect that is five times as large as being depressed. In addition, ADHD does not impact female's academic outcomes. In contrast, the impact of health behaviors were all negative and significantly associated with GPA for boys but the impacts did not vary across the health measures. Finally, the negative impact of the family environment variable is nearly twice as large for boys.

In columns four to six we present OLS estimates using the second health vector. Notice that the negative impact of ADHD was driven by AD and HD is positively associated with academic performance for the full sample. Boys have a strong negative association with AD which is approximately 50% larger than the impact for girls. Similarly, the positive impact of HD is 50% larger for boys but is statistically insignificant for both genders. Interestingly Asian females performed significantly higher than their Caucasian classmates while there were no differences for Asian boys. Finally, the results suggest that home environment had nearly twice as large an impact on GPA for boys than girls.

We next consider two stage least squares (2SLS) estimation of the full system of equations ((8)

[29] Due to space limitations estimates of equations (9) and (10) are available from the authors by request.

- (10)) for the two health vectors. Identification of the parameters arise from the use of genetric markers and two by two polygenic actions as instruments. To serve as an instrument, these markers must possess two properties. First, they must have a substantial correlation with the potentially endogenous health variables. Second, they must be unrelated to unobserved determinants of the other equations in the sysyem. Table 6 presents estimates of their performance relating to each criteria.

In the top panel we present estimates of the F-statistics of the instruments joint significance in the first stage regressions. For each health outcome and health behavior with each sample, the specification of the instrument set is highly statistically significant and above current cutoffs for weak instruments. Since our estimates are over-identified, we use a J-test to formally test the overidentifying restrictions. 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. However, these tests are known to have poor power properties.

Two stage least squares estimates of the achievement equation from the full system are presented in Table 7. The first three columns consider health vector 1 and the second three columns health vector two. For the full sample, only depression is significantly related to academic performance when ADHD is included. The impact of depression is approximately four times larger than the OLS estimate presented in Table 5. When ADHD is broken in to components both obesity and HD become statistically significant. Hyperactivity and impulsiveness is positively related to academic performance and the magnitude is extremely large. In contrast, the portion attributable to AD is no longer statistically significant once we correct for endogeneity.

The results for the subsample of females in columns 2 and 5 are most striking. The quantitative impact of each health behavior is substantial. Both depression and obesity lead to decreases in GPA. However, the impact of depression is nearly three times as large as that of obesity in health vector one. With health vector two, both depression and obesity lead to 0.8 GPA point decrease

representing increases of 0.3 and decreases of magnitude of 0.4 GPA points respectively. While the total impact of ADHD is close to zero, the separate effects of AD and HD are both large in magnitude and statisticlly significant. The impacts of these condition account for approximately 40% of mean GPA but offset each other. Similar to the other health outcomes inattention leads to lower GPA. As indicated in Appendix Table 3, which presents comorbidities by gender there are substantially fewer girls diagnosed with both AD and HD relative to boys. Further, there are many more depressed females particularly in the early waves. However, unlike males, girls that suffer from depression have fewer comorbid conditions.

Whereas health outcomes significantly affect academic achievement for girls, there are no statistically significant estimates for boys. Obesity has a large positive but insignificant impact with health vector one. The impact of home environment remains significant and the magnitude is slightly larger. in contrast, the effect of home environment diminished by approximately 40% for girls. We must emphasize that our variable indicating whether a smoker resides in the household is a proxy for family environment variables that we lack direct information. 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 the proxy for home environment and the magnitude as well as the statistical significance of the estimates was unchanged for each sample and health vector.

Appendix Table 2 presents results for the male and female subsample that correspond to their preffered instruments sets using stepwise estimation on those subsamples. While the first stage properties for these samples 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 from taking a quick glance. For females with health vector 2, the positive impact of HD and negative impact of AD shrinks by approximately 25% with this inst. However, the impact of depression 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 diagnosed with AD, depression or as obese; whereas no significant relationships exist for the males.

We test for the exogeniety of health status by testing the null hypothesis that the OLS and 2SLS estimates are equal using a Hausman-Wu test. We can reject the Null of exogeneity of health outcomes for each health vector with each sample. We next examine whether health behavior such as years of cigarette smoking can be treated as exogenous. We estimate a modified system including only equations (8) and (9) by 2SLS. Estimates of the parameters of the achievement equation are presented in Table 8.

There are substantial differences between the estimates in Table 7 and Table 8. The magnitude of all health outcomes increases markedly. In addition depression, HD and obesity significantly impact the performance of male students. The results suggest that obese leads to boys scoring 0.8 points higher on their GPA. The impact of depression nearly doubles in magnitude for the full sample and girls. 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.

We conducted a Hausman test of each health status equation for each vector in Table 7 by comparing it to the corresponding equation in Table 7. We can reject the Null of exogeneity for years of cigarette smoking. In addition, years of cigarette smoking serves as an exclusion restriction in Table 7. J- tests consistently reject the overidentifying restrictions underlying Table 8 suggesting that smoking may affect achievement through channels other than health.

Since health behaviors such as smoking are important explanatory variables in explaining health outcomes, one must account for the fact that these reflect individual choices. Despite the use of genes as instruments the inclusion of smoking leads to wildly different results. We believe the reason is that these health status and health behavior are strongly correlated to each other. Further, the

differences between genders may be a result in part of different smoking patterns. In particular, boys with AD have a greater association with smoking than girls. This is consistent with the hypothesis that for individuals with limited attention spans there is an immediate academic benefit or compensation from cigarette smoking. The results suggest that there may be compensation benefits from the smoking behavior.

At this point, our analysis indicates that health outcomes have a significant negative effect on academic achievement for females. We now consider what, if any, effect it would have on our estimates of the other coefficients if we followed the usual policy of ignoring comorbid conditions and only including one health outcome. The results from 2SLS of achievement equations that include only one health variable are presented in Table 9. Each entry refers to the point estimate of that health behavior from a system of equation which includes the achievement equation and that health behavior alone. Notice that for the full sample, we would conclude that inattention is positively related and HD is negatively related to GPA. In addition, the impact of depression is approximately 40% larger as it may be capturing a portion of the negative impact of obesity or ADHD. As indicated in Table 4, the majority of people with AD are comorbid. Similarly, the results for the subsample of boys vary greatly 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. Further, the negative impact of depression becomes substantially smaller when the other health states are excluded. Taken together the results of Table 8 and Table 9 illustrate the importance of accounting for a full set of health outcomes and behaviors in any analysis. Even with exogenous instruments such as genes to correct for the endogeneity of health status the omission of comorbid conditions may present a misleading picture of the causal relation between particular health states and academic performance among other outcomes.

6 Conclusions

This paper considers the use of information on genetic markers as a predetermined and exogenous source of information to identify the causal impact of health on education. The interactions and dynamics between health behavior and health status together with the information on genes might really be important in a line of research that tries to assess the impact of health as a form of a human capital on many outcomes of interest to economists such as labor market activity, marriage and educational attainment. In this paper, we find that the genetic markers used in our study exhibit properties to serve as a good instrument. These strong correlations between genes and health permit us to shed light on a long standing open question in empirical microeconomics: What is the impact of health status on academic outcomes? Our evidence suggests substantial heterogeneity in these effects and the academic performance of females is strongly affected by negative physical and mental health outcomes. Unfortunately, the results also lead to more questions in understanding why females and not males are so adversely affected by poor health outcomes.

Future work will attempt to understand why health states only negatively impact females. We will examine responses to a variety of psychological questionnaires to shed light on possible differences with males in their self-perception. We also plan to take a closer look at understanding how the inclusion of different sets of genetic markers affects the sensitivity of our results. Finally, it is well known that 2SLS is an inefficient estimation procedure when the error terms across the same individual are correlated. In subsequent drafts we will present evidence from the more efficient three stage least squares estimator where we will construct a random effects structure for the residual. In conclusion, recent years have witnessed an explosion of findings on the causes and correlates of health outcomes and behaviors in neurobiology which may provide a source of predetermined exogenous variation to help identify the impact of health on a set of outcomes of great interest to economists.

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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		
Biological Parent smoked		0.449		0.498		
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		
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: Number of Individuals with Each Genetic Marker

Gene	Marker	Total Number of People	Interacted with AA	Interacted with TT	Interacted with A1A1	Interacted with DAT0
GenoTPH	AA	120	****	4	5	16
	AC	393	****	15	20	39
	CC	380	****	12	27	65
GenoCYP	TT	31	4	****	2	3
	CT	191	24	****	9	19
	CC	671	92	****	41	56
DRD2	A1A1	52	5	2	****	3
	A1A2	286	34	9	****	19
	A2A2	555	81	20	****	56
DAT	DAT0	72	16	3	3	****
	DAT1	317	38	13	17	****
	DAT2	498	65	15	32	****

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

Gene	Marker	GPA	Depression	Smoking	Obesity	BMI	ADHD	AD	HD
GenoTPH	AA	3.124	0.149	0.158	0.108	23.939	0.067	0.033	0.033
		(0.642)	(0.357)	(0.365)	(0.312)	(4.516)	(0.250)	(0.180)	(0.180)
	AC	3.154	0.150	0.105	0.074	23.291	0.074	0.048	0.043
		(0.567)	(0.357)	(0.306)	(0.262)	(4.140)	(0.262)	(0.215)	(0.204)
	CC	3.198	0.156	0.101	0.079	23.403	0.050	0.039	0.039
		(0.569)	(0.363)	(0.301)	(0.270)	(4.640)	(0.218)	(0.195)	(0.195)
GenoCYP	TT	3.271	0.165	0.121	0.032	22.536	0.129	0.129	0.097
		(0.460)	(0.373)	(0.328)	(0.180)	(3.283)	(0.341)	(0.341)	(0.301)
	CT	3.177	0.159	0.111	0.058	23.082	0.031	0.010	0.026
		(0.565)	(0.366)	(0.315)	(0.234)	(4.195)	(0.175)	(0.102)	(0.160)
	CC	3.162	0.150	0.109	0.089	23.565	0.069	0.048	0.042
		(0.587)	(0.357)	(0.312)	(0.286)	(4.508)	(0.253)	(0.213)	(0.200)
DRD2	A1A1	3.133	0.189	0.122	0.096	23.562	0.058	0.038	0.038
		(0.564)	(0.393)	(0.328)	(0.298)	(5.998)	(0.235)	(0.194)	(0.194)
	A1A2	3.144	0.174	0.100	0.115	23.860	0.049	0.021	0.035
		(0.582)	(0.380)	(0.301)	(0.320)	(4.651)	(0.216)	(0.144)	(0.184)
	A2A2	3.185	0.138	0.114	0.061	23.189	0.070	0.054	0.043
		(0.578)	(0.345)	(0.318)	(0.240)	(4.088)	(0.256)	(0.226)	(0.204)
DAT	DAT0	3.163	0.155	0.155	0.077	23.685	0.064	0.038	0.051
		(0.528)	(0.363)	(0.363)	(0.268)	(5.310)	(0.247)	(0.194)	(0.222)
	DAT1	3.161	0.109	0.122	0.095	23.775	0.091	0.063	0.060
		(0.601)	(0.311)	(0.327)	(0.293)	(4.749)	(0.289)	(0.244)	(0.238)
	DAT2	3.175	0.172	0.104	0.072	23.161	0.044	0.030	0.026
		(0.572)	(0.378)	(0.306)	(0.259)	(4.004)	(0.206)	(0.171)	(0.160)

Note: We include information on the full sample used in our analysis for GPA, depression and smoking. The remaining health outcomes present summary information where each only one observation per individual is included.

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	570	***	***	***	***	***	***	***
Smokes	73	46	***	7	4		7	16
AD	33	6	7	***	14		3	15
HD	30	9	4	14	***		2	10
ADHD	49	25	8	33	29	***	4	19
Obese	68	42	7	3	2		***	17
Depression	140	93	16	15	10		17	***
Wave 4, N=863								
Nothing	584	***	***	***	***	***	***	***
Smokes	82	47	***	9	5		10	21
AD	37	7	9	***	17		4	15
HD	34	10	5	17	***		3	9
ADHD	54	25	10	37	33	***	5	19
Obese	70	43	10	4	3		***	17
Depression	146	96	21	15	9		17	***
Wave 5, N=879								
Nothing	596	***	***	***	***	***	***	***
Smokes	129	80	***	15	11		15	20
AD	38	8	15	***	18		4	10
HD	36	8	11	18	***		3	9
ADHD	56	30	18	38	35	***	5	15
Obese	67	40	15	4	3		***	10
Depression	107	66	20	10	9		10	***

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

Table 5: Ordinary Least Squares Estimates of the Achievement Equation

	Full Sample	Females Only	Male Only	Full Sample	Females Only	Male Only
ADHD	-0.198 (0.079)	-0.154 (0.115)	-0.241 (0.110)	N/A	N/A	N/A
AD	N/A	N/A	N/A	-0.431 (0.108)	-0.350 (0.200)	-0.493 (0.138)
HD	N/A	N/A	N/A	0.158 (0.090)	0.120 (0.114)	0.177 (0.135)
Depression	-0.143 (0.040)	-0.097 (0.046)	-0.191 (0.069)	-0.135 (0.040)	-0.094 (0.046)	-0.180 (0.068)
Obesity	-0.371 (0.071)	-0.529 (0.086)	-0.204 (0.106)	-0.370 (0.070)	-0.533 (0.085)	-0.199 (0.104)
Smoker in Home	-0.199 (0.038)	-0.135 (0.046)	-0.274 (0.058)	-0.195 (0.037)	-0.135 (0.045)	-0.265 (0.057)
Age	0.856 (0.466)	0.754 (0.556)	0.918 (0.753)	0.856 (0.466)	0.745 (0.557)	0.917 (0.753)
Age Squared	-0.027 (0.014)	-0.022 (0.016)	-0.030 (0.022)	-0.027 (0.014)	-0.022 (0.016)	-0.030 (0.022)
Black	-0.313 (0.057)	-0.276 (0.064)	-0.345 (0.097)	-0.318 (0.057)	-0.283 (0.064)	-0.346 (0.097)
Hispanic	-0.266 (0.060)	-0.253 (0.087)	-0.250 (0.086)	-0.255 (0.060)	-0.235 (0.089)	-0.244 (0.086)
Asian	0.095 (0.054)	0.170 (0.067)	-0.053 (0.084)	0.094 (0.054)	0.163 (0.067)	-0.041 (0.084)
Male	-0.255 (0.032)	N/A	N/A	-0.249 (0.032)	N/A	N/A
Constant	-3.234 (4.019)	-2.986 (4.846)	-3.636 (6.499)	-3.216 (4.020)	-2.912 (4.848)	-3.587 (6.498)
N	2576	1366	1210	2576	1366	1210
R squared	0.19	0.21	0.14	0.20	0.21	0.16

Note: Standard errors clustered at the individual level are in parentheses. Regressions include school and time period indicators.

Table 6: Summary Information on the Performance of the Instruments

	Full Sample	Females Only	Male Only	Full Sample	Females Only	Male Only
First Stage F statistics						
ADHD	9.51	8.12	7.25	N/A	N/A	N/A
AD	N/A	N/A	N/A	13.80	10.25	10.88
HD	N/A	N/A	N/A	9.37	12.83	7.32
Depression	6.95	5.78	6.55	6.95	5.78	6.55
Obesity	7.43	12.55	11.39	7.43	12.55	11.39
Smoking	6.38	9.83	8.81	6.38	9.83	8.81
P-values from Overidentification Tests						
ADHD	0.553	0.420	0.236	N/A	N/A	N/A
AD	N/A	N/A	0.817	0.842	0982	0.440
HD	N/A	N/A	N/A	0.845	0.812	0.266
Depression	0.773	0.822	0.465	0.773	0.822	0.465
Obesity	0.216	0.232	0.817	0.216	0.232	0.817
Achievement	0.267	0.874	0.421	0.524	0.617	0.293

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

	Full Sample	Females Only	Male Only	Full Sample	Females Only	Male Only
ADHD	0.017 (0.277)	-0.074 (0.316)	0.161 (0.305)	N/A	N/A	N/A
AD	N/A	N/A	N/A	-0.644 (0.458)	-1.410 (0.649)	-0.036 (0.413)
HD	N/A	N/A	N/A	1.297 (0.685)	1.306 (0.736)	0.753 (0.610)
Depression	-0.574 (0.249)	-1.112 (0.289)	-0.127 (0.275)	-0.520 (0.252)	-0.822 (0.265)	-0.237 (0.283)
Obesity	-0.288 (0.272)	-0.452 (0.249)	0.338 (0.296)	-0.634 (0.286)	-0.838 (0.327)	0.011 (0.302)
Smoker in Home	-0.194 (0.031)	-0.094 (0.042)	-0.306 (0.045)	-0.187 (0.033)	-0.095 (0.042)	-0.296 (0.047)
Age	0.691 (0.521)	0.378 (0.885)	0.761 (0.746)	0.663 (0.552)	0.611 (0.874)	0.626 (0.746)
Age Squared	-0.022 (0.015)	-0.011 (0.026)	-0.025 (0.022)	-0.021 (0.016)	-0.018 (0.026)	-0.021 (0.022)
Black	-0.323 (0.047)	-0.367 (0.070)	-0.342 (0.076)	-0.319 (0.049)	-0.372 (0.069)	-0.321 (0.074)
Hispanic	-0.259 (0.042)	-0.137 (0.079)	-0.234 (0.060)	-0.224 (0.049)	-0.021 (0.108)	-0.255 (0.061)
Asian	0.128 (0.041)	0.225 (0.057)	-0.067 (0.067)	0.127 (0.043)	0.150 (0.065)	-0.018 (0.075)
N	2576	1366	1210	2576	1366	1210
R squared	0.12	0.29	0.15	0.19	0.24	0.15

Note: Standard errors clustered at the individual level are in parentheses. Regressions include school and time period indicators.

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

	Full Sample	Females Only	Male Only	Full Sample	Females Only	Male Only
ADHD	-0.959 (0.375)	-0.630 (0.409)	-0.138 (0.392)	N/A	N/A	N/A
AD	N/A	N/A	N/A	-1.382 (0.647)	-3.760 (1.216)	-0.441 (0.605)
HD	N/A	N/A	N/A	1.101 (1.147)	3.782 (1.715)	1.689 (1.094)
Depression	-1.297 (0.328)	-1.251 (0.424)	-0.857 (0.337)	-1.304 (0.350)	-0.962 (0.619)	-1.456 (0.502)
Obesity	-0.158 (0.382)	-0.601 (0.303)	0.774 (0.420)	-0.912 (0.418)	-2.080 (0.755)	0.833 (0.588)
Smoker in Home	-0.123 (0.040)	-0.062 (0.049)	-0.280 (0.052)	-0.113 (0.040)	-0.045 (0.073)	-0.268 (0.064)
Age	0.787 (0.676)	0.458 (0.967)	0.774 (0.834)	0.734 (0.692)	1.051 (1.476)	0.319 (1.033)
Age Squared	-0.024 (0.020)	-0.014 (0.028)	-0.024 (0.024)	-0.023 (0.020)	-0.030 (0.043)	-0.010 (0.030)
Black	-0.389 (0.061)	-0.386 (0.081)	-0.348 (0.085)	-0.371 (0.061)	-0.429 (0.122)	-0.299 (0.102)
Hispanic	-0.228 (0.055)	-0.070 (0.093)	-0.270 (0.067)	-0.153 (0.064)	0.345 (0.215)	-0.315 (0.085)
Asian	0.164 (0.053)	0.219 (0.065)	-0.025 (0.076)	0.157 (0.055)	0.010 (0.124)	0.104 (0.109)
Male	-0.267 (0.031)	N/A	N/A	-0.260 (0.032)	N/A	N/A
Constant	-2.751 (5.770)	-0.184 (8.285)	-2.816 (7.111)	-2.153 (5.907)	-5.355 (12.649)	0.860 (8.788)
N	2576	1366	1210	2576	1366	1210
R squared	0.18	0.21	0.17	0.24	0.24	0.16

Note: Standard errors clustered at the individual level are in parentheses. Regressions include school and time period indicators.

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

Include health behaviors	Full Sample	Girls	Boys
ADHD	-0.351 (0.319)	-0.319 (0.359)	0.284 (0.452)
R-squared	0.15	0.13	0.07
AD	1.392 (0.669)	0.648 (0.633)	0.615 (0.546)
HD	-1.966 (1.183)	-1.040 (0.609)	0.237 (0.911)
R-squared	0.17	-0.02	-0.06
AD	0.529 (0.304)	-0.124 (0.400)	0.766 (0.383)
R-squared	-0.34	0.13	-0.08
HD	-0.144 (0.517)	-0.330 (0.445)	0.972 (0.766)
R-squared	0.06	0.12	-0.02
Depression	-0.713 (0.302)	-1.250 (0.455)	-0.032 (0.391)
R-squared	0.14	-0.48	0.11
Obesity	-0.331 (0.329)	-0.352 (0.235)	1.067 (0.738)
R-squared	0.17	0.19	-0.22
Observations	2576	1366	1210

Note: Standard errors clustered at the individual level are in parentheses. Regressions include school and time period indicators.

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

	Full Sample	Females Only	Male Only	Full Sample	Females Only	Male Only
ADHD	0.109 (0.051)	0.169 (0.080)	0.057 (0.064)	N/A	N/A	N/A
AD	N/A	N/A	N/A	0.165 (0.071)	0.274 (0.117)	0.117 (0.089)
HD	N/A	N/A	N/A	0.007 (0.061)	0.051 (0.088)	-0.032 (0.075)
Depression	0.030 (0.022)	-0.007 (0.024)	0.068 (0.038)	0.027 (0.021)	-0.013 (0.023)	0.065 (0.038)
Obesity	0.043 (0.040)	0.116 (0.065)	-0.032 (0.044)	0.042 (0.040)	0.117 (0.065)	-0.034 (0.043)
Smoker in Home	0.096 (0.025)	0.136 (0.034)	0.040 (0.037)	0.095 (0.025)	0.134 (0.034)	0.038 (0.037)
Age	-0.377 (0.224)	-0.146 (0.280)	-0.526 (0.308)	-0.379 (0.224)	-0.145 (0.279)	-0.525 (0.308)
Age Squared	0.012 (0.007)	0.005 (0.008)	0.018 (0.009)	0.012 (0.007)	0.005 (0.008)	0.018 (0.009)
Black	-0.027 (0.040)	-0.021 (0.053)	-0.035 (0.065)	-0.026 (0.040)	-0.016 (0.053)	-0.034 (0.065)
Hispanic	-0.050 (0.031)	0.004 (0.049)	-0.119 (0.039)	-0.055 (0.031)	-0.006 (0.050)	-0.120 (0.039)
Asian	-0.056 (0.027)	-0.059 (0.034)	-0.048 (0.047)	-0.056 (0.027)	-0.055 (0.034)	-0.050 (0.048)
Male	0.019 (0.019)	N/A	N/A	0.017 (0.019)	N/A	N/A
Constant	2.865 (1.880)	1.200 (2.467)	3.851 (2.531)	2.875 (1.883)	1.201 (2.458)	3.839 (2.530)
N	2576	1366	1210	2576	1366	1210
R squared	0.07	0.12	0.09	0.08	0.13	0.09

Note: Standard errors clustered at the individual level are in parentheses. Regressions include school and time period indicators.

Appendix Table 2: Two Stage Least Squares Estimates of the Achievement Equation by Subsample with Alternative Preferred instrument Sets

	Females Only	Male Only	Females Only	Male Only
ADHD	-0.222 (0.350)	0.255 (0.311)	N/A	N/A
AD	N/A	N/A	-1.092 (0.541)	-0.036 (0.438)
HD	N/A	N/A	0.580 (0.421)	0.835 (0.576)
Depression	-1.296 (0.349)	-0.207 (0.316)	-1.132 (0.324)	-0.199 (0.321)
Obesity	-0.385 (0.237)	0.166 (0.311)	-0.708 (0.257)	0.055 (0.351)
Smoker in Home	-0.057 (0.052)	-0.246 (0.048)	-0.052 (0.051)	-0.237 (0.048)
Age	0.291 (0.959)	0.634 (0.740)	0.490 (0.924)	0.587 (0.740)
Age Squared	-0.009 (0.028)	-0.021 (0.022)	-0.015 (0.027)	-0.020 (0.022)
Black	-0.397 (0.080)	-0.324 (0.075)	-0.383 (0.077)	-0.321 (0.074)
Hispanic	-0.123 (0.085)	-0.263 (0.060)	-0.028 (0.101)	-0.274 (0.060)
Asian	0.237 (0.062)	-0.054 (0.068)	0.183 (0.063)	-0.017 (0.073)
N	1366	1210	1366	1210
R squared	0.28	0.16	0.24	0.16

Note: Standard errors clustered at the individual level are in parentheses. Regressions include school and time period indicators.

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	305	***	***	***	***	***	***
Smokes	33	18	***	4	3	6	7
AD	11	2	4	***	4	1	7
HD	13	4	3	4	***	1	6
Obese	34	19	6	1	1	***	9
Depression	81	59	7	7	6	9	***
Wave 4, N=453							
Nothing	311	***	***	***	***	***	***
Smokes	35	17	***	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	324	***	***	***	***	***	***
Smokes	64	42	***	7	6	10	7
AD	13	3	7	***	6	2	3
HD	15	4	6	6	***	2	4
Obese	35	19	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	260	***	***	***	***	***	***
Smokes	39	28	***	3	1	1	8
AD	22	4	3	***	10	1	8
HD	16	5	1	10	***	1	4
Obese	34	23	1	1	1	***	8
Depression	58	34	8	8	4	8	***
Wave 4, N=402							
Nothing	267	***	***	***	***	***	***
Smokes	46	30	***	5	2	2	12
AD	24	5	5	***	13	2	7
HD	18	5	2	13	***	1	3
Obese	34	26	2	2	1	***	7
Depression	58	32	12	7	3	7	***
Wave 5, N=405							
Nothing	268	***	***	***	***	***	***
Smokes	62	38	***	8	5	5	10
AD	25	5	8	***	12	2	7
HD	20	4	5	12	***	1	5
Obese	32	21	5	2	1	***	5
Depression	51	25	10	7	5	5	***