Are we substituting between heart disease risks?

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Abstract

I speculate that the rises in overweight and obesity rates in the United States during the 1980s were related to the arrival and increasing use of highly effective and well-tolerated drugs to lower blood pressure and cholesterol. This hypothesis is constructed from a simple economic model in which people regard heart disease risk factors as substitutes. The hypothesis is tested empirically by using insurance status as an instrument for access to the new drugs and employing a difference-in-difference approach to individual level data. The results provide little evidence for the hypothesis, but reveal a striking pattern for adult males in the relationship between insurance and health measures related to heart disease risk. Between the early 1970s and the early 1990s, insured men with high school or above education experienced significant improvements in these health measures, while insured men with less than a high school education experienced the opposite.

Keywords: heart disease risk factors; overweight; high blood pressure; high cholesterol; substitution effect

I: Introduction

For over fifty years heart disease has been the number one cause of death for Americans, but we can take courage in the fact that we have a large amount of control over our individual vulnerabilities. Through our behavior and medical treatment we can modify several of the most important predisposing factors. The National Heart, Lung, and Blood Institute identifies six major "controllable" risk factors for heart disease: high blood pressure, high cholesterol level, overweight, smoking, diabetes, and physical inactivity[1] (NHLBI 1999). These controllable risk factors have changed significantly in the American population in the last two decades. The prevalences of high blood pressure, high cholesterol, and smoking have declined in the American adult population, while the prevalences of overweight and diabetes have increased (see Table 1). Physical inactivity is difficult to quantify reliably given available data.

This study focuses on the contrast between the recent sharp declines in proportion of people with high blood pressure or high cholesterol, and the recent sharp increase in proportion of people who are overweight. Why did these trends diverge? First, increased awareness of both the presence and consequences of risk factors has probably played a large role in reducing the rates of high blood pressure and high cholesterol. The federal government launched the National High Blood Pressure Education Program in 1973 and the National Cholesterol Education Program in 1985. Between the late 1970s and late 1980s, the percentages of Americans with high blood pressure who were aware they had this condition increased from 51 to 73. Treatment of people with high blood pressure increased from 31 to 55 (Sixth Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure, 1997). New attention to high blood pressure and high cholesterol in turn helped

contribute to a change in eating habits by the American people. For example, according to various surveys and food data, during the 1970s Americans significantly reduced their consumption of fat, cholesterol, sugar, and salt (Stamler 1985).

Meanwhile, increased awareness of heart disease risk has clearly not been sufficient to prevent the increase in the proportion of overweight people. The source of this upward trend is still not well understood. Some researchers have attributed the trend to decreases in calories expended, while others have attributed it to increases in calories consumed. Heini and Weinsier (1997) find in USDA food consumption data that average caloric intake fell by about five percent between 1977 and 1988, and therefore conclude that calories expended must have decreased. Cutler et al (2003) look at more recent USDA data and find that average caloric intake actually rose by over ten percent between 1977 and 1994; furthermore they find evidence suggesting that this increase is due to decreased food preparation time and costs. In contrast, Philipson and Posner (1999) make the case that the decreasingly physical nature of jobs is a significant factor, and they also argue that there is little evidence in support of alternative explanations such as decreases in smoking rates and shifts from home-cooked to fast food meals.

While the above explanations probably represent pieces of the puzzle, this study proposes an additional explanation involving recent innovations in medical technology, which could help explain *simultaneously* the increase in overweight and the declines in high blood pressure and cholesterol prevalence. In the last two decades, effective, relatively well-tolerated drug treatments for high blood pressure and high cholesterol have gained prominence, while comparably effective treatments for obesity have not. This study poses and tests the following hypothesis: economic utility maximization suggests that consumers, with the availability of more cost-effective blood pressure and cholesterol

reducing treatments, would begin to lead riskier lifestyles with respect to potential heart disease, in particular by controlling their weights less diligently[2].

In order to examine this hypothesis empirically a difference-in-difference approach is employed. Differentials in health outcomes (e.g. obesity) across drug availability (proxied by insurance status) and time (before versus after the influx of better drugs) are examined. The hypothesis under study would be consistent with an increase over time in the overweight differential for the insured versus the uninsured. This result would be strengthened if such a differential does not exist for other health outcomes that are not specific to heart disease risk. For this purpose, a measure of self-reported general health is also included in the analysis.

II: Background: trends in risk factors and drugs to control them

The hypothesis of this study is motivated by the fact that certain trends in high blood pressure, high cholesterol, and overweight rates coincided with the introduction and expanded popularity of effective treatments for high blood pressure and high cholesterol. In this section these trends are examined in more detail.

Trends in heart disease risk factors

Table 1 shows the proportions of U.S. adults aged 25-64 with the listed heart disease risk factors at different points in time. The percentages were computed using data from four different surveys published by the National Center for Health Statistics. The first is the National Health Examination Survey (NHES), conducted from 1960 to 1962, and the three succeeding surveys are versions of the National Health and Nutrition Examination Survey (NHANES I, II, and III), which were conducted in

1971-75, 1976-80, and 1988-94 respectively. The surveys contain nationally representative samples of individuals, who responded to surveys and underwent medical examinations.

High blood pressure, high cholesterol, and smoking declined between the first and last surveys, while overweight, obesity, and diabetes increased. The definitions for these measures are noted below Table 1. The changes in prevalences occurred largely between the last two surveys, i.e. during the 1980s. High blood pressure, high cholesterol, and smoking rates fell by 64 percent, 43 percent, and 19 percent, respectively, while overweight, obesity, and diabetes increased by 21 percent, 38 percent, and 50 percent, respectively. Cross-tabulations by sex and age groups (not shown) reveal that the basic trends above hold for each sex, as well as for each age group. It should be noted that the diabetes data are less reliable due to the fact that, unlike the other conditions, diabetes is self-reported. Improvements in detection rates could have accounted in part for the rise in diabetes prevalence. According to the Centers for Disease Control, currently almost 16 million Americans have diabetes, but about one-third of them are not aware of their condition (www.cdc.gov).

Further investigation of the increasing trend for overweight prevalence reveals that the rise in the 1980s was even steeper for more severe cases of overweight. Flegal, Carroll, Kuczmarski, and Johnson (1998) classified obesity as class I if a person's body mass index[3] was between 30.0 and 34.9, class II if between 35.0 and 39.9, and class III if over 40.0. Their results showed dramatic increases in the proportion of American falling into each category between NHANES II and NHANES III: 43 percent for class I, 68 for class 2, and 123 for class 3.

Trends in drugs for heart disease risk factors

In the NHES and NHANES surveys, data regarding drugs used to control risk factors are much less detailed than the data regarding the risk factors themselves. The surveys contain information on whether an individual uses a prescribed drug to lower blood pressure, but this information is only available for relatively small subsets of the surveys. NHANES III also contains similar information on whether an individual uses any *cholesterol*-lowering drugs.

The National Ambulatory Medical Care Survey (NAMC), on the other hand, offers a better picture of trends in the use of the drugs of interest. The prescription numbers in Table 2 come from three different publications from the National Center for Health Statistics' *Advance Data from Vital and Health Statistics*, each of which uses the NAMC as its data source (Koch 1982, Koch and Knapp 1987, Nelson 1993). The NAMC conducts annual, year-long surveys of office-based medical care providers and projects its findings to national figures.

The *Advance Data* publications provide numbers on national "drug mentions" (defined in Table 2 notes) to treat hypertension and obesity, but no data on drugs to treat high cholesterol, diabetes, or smoking. The data show markedly divergent trends for hypertension and obesity drugs during the 1980s. Antihypertensive drug mentions increased significantly, particularly during the second half of the decade, while anti-obesity drug mentions plummeted. The increase in antihypertensive drugs was propelled largely by the emergences of calcium channel blockers and angiotensin converting enzyme (ACE) inhibitors, which both increased five to six fold in number of prescriptions between 1986 and 1995. During that same time, prescriptions for diuretics, which had been a popular treatment for high blood pressure for many years, fell by about 20 percent (Mulrow 1998).

The increasing acceptance of new blood pressure medications and such medications in general during the 1980s and early 1990s was reflected not only in the number of drug mentions but also in the recommendations of the 1993 Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure. In his *JAMA* review of the report, Alderman (1993) writes "Perhaps the most significant new departure in this fifth edition of the Joint National Committee Reports is reflected in the therapeutic algorithm. The committee continues to recommend that initial efforts to control blood pressure should be through lifestyle modification. However, for that large majority of hypertensive patients in whom behavioral change does not produce a sustained and significant drop in pressure, antihypertensive drugs are indicated." Later in his review, Alderman notes "It was also agreed that side effects, while different for the classes, were, for most patients, not a persistent problem." Furthermore, the popularity of blood pressure lowering drugs was bolstered by studies which validated their effectiveness. For example, a 1993 meta-analysis of clinical trials and observational studies estimated that drug therapy for mild to moderate hypertension decreases the risk of coronary heart disease by 16 percent (95 percent CI: 8 to 23 percent) (Hebert et al 1993).

Cholesterol-reducing drugs also progressed rapidly during the 1980s. An article appearing in *JAMA* by Wysowski et al. (1990), which derives its numbers from IMS America Ltd.'s *National Prescription Audit*, details the rising use of cholesterol lowering drugs, as seen in Table 3. The number of prescriptions fell between 1978 and 1983, but then exploded between 1983 and 1988. The article also highlights the dramatic shift in *which* cholesterol lowering drugs were being used. In 1978, clofibrate dominated the cholesterol lowering drug market with 80.9 percent of prescriptions, and dextrothyroxine was second at 8.9 percent. By 1988, these two drugs only accounted for 3.5 and 0.4 percent of prescriptions respectively, while the two leaders were lovastatin at 29.7 percent and

gemfibrozil at 29.4 percent. Lovastatin was introduced to the market in 1987 and shot to the top in only one year, while gemfibrozil was introduced in 1982.

These newer drugs have generally been shown to be both effective and free of major adverse side effects. According to the 1993 Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults, statins are highly effective in lowering LDL cholesterol ("bad cholesterol") levels, and are relatively safe. A meta-analysis (Hebert et al 1997) of all published trials testing statin drugs between 1985 and 1995 found the following: an average of 22 percent decrease in total cholesterol level and 30 percent decrease in LDL level; a 29 percent reduction in risk of stroke (95 percent CI: 14 - 41 percent) and 22 percent reduction in mortality (95 percent CI: 12 - 31 percent). The study found no evidence of any increased risk in non-CVD mortality. The other leading new drug, gemfibrozil, is deemed to be effective at lowering triglyceride levels but not so much in lowering LDL cholesterol and raising HDL ("good cholesterol"). In contrast to gemfibrozil and the statins, nicotinic acid, an older cholesterol lowering drug, has several side effects that limit its use in some patients, according to the 1993 Expert Panel. As in the case of antihypertensive drugs, the emergence of new cholesterol lowering drugs clearly suggest that the 1980s was a decade of great innovation and progress for this class of therapies.

More recent data suggest that prescriptions for blood pressure lowering drugs and cholesterol drugs have continued to increase steadily in the 1990s, while anti-obesity drug prescriptions remained stable during the early 1990s and then rose precipitously in 1995 and 1996[4]. These data, obtained from National Data Corporation (NDC) Health Information Services, are presented in Table 4. NDC collects data from participating pharmacies and then projects figures to the known U.S. pharmacy universe. The data in Table 3 and 4 are not directly comparable to the data in Table 2, since

prescriptions are compiled directly from the pharmacies whereas drug mentions are compiled from doctors' responses to surveys. Also, some drugs that are sometimes but not always used to lower blood pressure may have been counted in one survey as blood pressuring lowering, but not in the other. Due to these inconsistencies, it is more instructive to examine the trends within each data source rather than trying to construct trends by bridging them.

The NDC data also breaks down the blood pressure lowering and cholesterol lowering drugs into sub-classes (not shown here). The leading blood pressure lowering drugs changed little from 1991 to 1997, with diuretics, calcium channel blockers, ACE inhibitors, and beta blockers being the top four in each of the years. Meanwhile, the cholesterol lowering drugs are classified as statins and "others." Statins continued their rising dominance from the 1980s, with approximately 50 percent of prescriptions in 1991 and over 80 percent of prescriptions in 1997.

Do risk factor trends and drug trends coincide?

Given the data discussed above on heart disease risk factors and related drugs, can we note any temporal connections? Although the data points are spaced at intervals of several years, with the exception of the annual drug prescription data in Table 4, we can at least note that a great emergence of blood pressure lowering and cholesterol lowering drugs coincided roughly with considerable decreases in the proportion of people with high blood pressure and high cholesterol, as well as an increase in the proportion of overweight people. Each of these events occurred during the early and mid 1980s. Furthermore, there was no sustained emergence of effective weight lowering drugs. These observations conform to the basic hypothesis posited by this study, but of course cannot be considered evidence.

The next section elaborates on the theory behind the study's hypothesis, and then the following sections present empirical tests.

III: Economic Theory

How we manage our heart disease risks is not only a biological issue but also a distinctly economic one. We can devote various limited resources, such as our time, energy, and money, to controlling or lowering our risks. Every day we make choices, whether they are conscious or not, about how much of these resources to allocate and where to allocate them. Do we take half an hour to go for a jog? Do we take the time and spend the money to see the doctor for a checkup? Do we avoid tasty but fatty foods?

Certainly not every action aimed at controlling heart disease risk involves only costs. Many people enjoy exercise, for example, and derive benefits besides the lowering of their heart disease risk. However, for the purpose of the model outlined below, the important point is that most choices related to heart disease risk involve definite tradeoffs, and to some extent these tradeoffs can be understood and analyzed in an economic framework.

The model used here focuses on the tradeoff between the probability of avoiding heart disease and a risky lifestyle (Figure 1). Both concepts are "goods" that enter positively into the consumer's utility function. A "risky lifestyle" would include eating high-fat and high-cholesterol foods, smoking, and avoiding exercise, for example. The straight line AB represents the initial budget constraint. At point A the person has the least risky lifestyle possible, and thus the highest probability of avoiding heart disease. By contrast, at point B the person has the riskiest lifestyle possible, and thus the lowest probability of

avoiding heart disease. In this example, the person maximizes utility, given the budget constraint, at point 1.

Such a model is analogous to the model outlined by Sam Peltzman in his 1975 *Journal of Political Economy* article, "The Effects of Automobile Safety Regulation." Peltzman argues that for automobile drivers, "driving intensity" is a good and "probability of death" is a bad, and there is a positive correlation between the two that represents a budget constraint. If a driver wants to consume more driving intensity (which refers to speed, most notably), then he or she must bear an increased risk of death. In the heart disease risk model in Figure 1, a risky lifestyle is analogous to driving intensity in Peltzman's model, and the probability of avoiding heart disease is analogous to the probability of death except that the former has been transformed from a bad to a good by inserting the word "avoiding." This small departure from Peltzman's model seems preferable for the simple reason that the two-good framework is a more familiar one than the one good-one bad framework, and can be understood more easily in terms of tradeoffs.

In Figure 1 I consider what happens when there is an increase in the availability of effective and well-tolerated drugs that lower blood pressure and cholesterol. With the new drugs, the person's tradeoff between the probability of avoiding heart disease and riskiness of lifestyle becomes less steep, and the person faces the new budget constraint AC. This constraint applies to any person for whom the new drugs are available[5], and reflects the fact that for any amount of riskiness in lifestyle, the person's probability of avoiding heart disease has increased. In essence the price of a risky lifestyle has fallen, because adding a given amount of lifestyle riskiness (e.g. consuming more fatty foods) results in less additional heart disease risk than before.

In order to analyze the move to the new optimum in Figure 1, I divide the net effect of the price change into a substitution and income effect. First, the substitution effect moves the person from point 1 to point 2. Unambiguously, the substitution effect results in a lower probability of avoiding heart disease and a riskier lifestyle. The risky lifestyle has essentially become cheaper, so the person "consumes" more of it. Next, the nature of the income effect depends on whether the goods in the model are normal goods. If they are normal goods, then the income effect will cause the person to consume more of both goods, and the person will end up on a point within the dark segment on AC. Thus, under the normal good assumption, we can determine in this model that the use of new, more cost-effective drugs increases the riskiness of lifestyle, and whether or not the probability of avoiding heart disease rises or falls depends upon the relative size of substitution and income effects.

One could certainly criticize the model for being too simplified. For example, it does not consider explicitly how the person's management of heart disease risk through drugs fits into his overall budget constraint and consumption of other goods. However, for the purposes of this model, the important point is that the person's consumption of heart disease risk management through drugs increases with the introduction of more cost-effective drugs. What happens to consumption of other goods does not influence this relationship.

A more serious simplification in the model is that it does not consider what happens if someone does contract heart disease. Heart disease ranges widely in terms of its gravity; persistent uncomfortable chest pain and a fatal heart attack are both considered heart disease, for example. In the context of the model presented in Figure 1, one can see how this issue could affect the analysis by comparing points 3 and 4. The two points represent the same probability of avoiding heart disease, but the probabilities result from different behaviors. Point 3 involves a less risky lifestyle and less devotion

of resources to heart disease risk management through drugs, than point 4, which involves a very risky lifestyle and more resources to control risk. One might guess that the person at point 4 would generally experience more serious consequences, given that heart disease occurs, than the person at point 3, because at point 4 he is relying on medications rather than a healthy lifestyle to control the risk. For now, however, this idea remains speculation until empirical medical evidence passes judgment. In any case, it should not affect the qualitative predictions based on the model. With the availability of better drugs we still should expect the change in riskiness of lifestyle to be positive and the change in probability of avoiding heart disease to be ambiguous,.

Another possible concern about this model relates to people's awareness of heart disease risk factors. If, for example, people do not understand that both high blood pressure and being overweight are risk factors, then it is unlikely that they will behave in response to the availability of effective blood pressure lowering drugs in the manner predicted in the model. However there is some empirical evidence that shows that at least a significant minority of the adult population is aware of the major "controllable" risk factors that are prominent in this study. Kirkland et al. (1999) analyzed a survey of Canadian adults aged 55-74 who were asked to name risk factors for heart disease in 1986 and 1992 and found the following: 23 percent listed high blood cholesterol; 16percent listed hypertension; 42 percent listed smoking; and 31 percent listed overweight. Also, Mosca et al. (2000) conducted a similar survey of American women over the age of 25 in 1997, and found that about 30 percent listed high blood cholesterol, smoking, and overweight, and about 15 percent listed high blood pressure. While these numbers are not overwhelming, they suggest that the behavioral response hypothesized in this study could occur for at least a nontrivial fraction of the population. Furthermore, even without

patient awareness, the response could occur to the extent that doctors act as if heart disease risks are substitutes when they treat and make recommendations to patients.

The key prediction in this study is that riskiness of lifestyle with respect to heart disease should increase with the introduction of more cost-effective drugs to lower heart disease risk. More specifically, the hypothesis is that the introductions during the 1980s of new drugs to lower blood pressure and cholesterol were partly responsible for the increasing proportion of overweight people in the American adult population. In addition we would expect that this substitution effect was responsible for keeping smoking rates from falling more than they actually did. As for blood pressure and cholesterol, the theory presented above does not make a clear prediction regarding the effect of the new drugs; the benefits of the drugs and the substitution towards riskier behaviors oppose each other, and the net effect is ambiguous.

IV: Empirical strategy

A straightforward way to start examining the relationship between the availability of effective blood pressure and cholesterol medications and "riskiness of lifestyle" would be to look at the correlation between the use of these medications and outcomes such as overweight status or whether a person smokes. A regression might take the following form:

(1) $overweight_i = B_0 + B_1 X_i + B_2 (med_i) + e_i$

However, one cannot interpret results from this regression as evidence that medications cause people to control their weight less. The direction of causality undoubtedly runs the other way as well; people who are more overweight tend to use the medications more frequently, because the lowering of their blood pressure and cholesterol is more urgent.

In the theory of this study, the force which drives the substitution towards riskier lifestyles is the *availability* of effective medications, as opposed to the actual use of them. The use of medications is highly correlated with availability, but as a RHS variable it suffers from obvious endogeneity problems, as mentioned above. A better proxy for availability of medications is insurance status. It represents a reasonable measure of the availability of medications for a person, and furthermore it is less likely than medication use to be correlated with unobservable factors related to health outcomes such as overweight.

Health insurance information for respondents is available for one survey before the influx of effective blood pressure and cholesterol medications (NHANES 1, 1971-75) and for one survey after this influx (NHANES 3, 1988-94). Thus, one can compare the impact of drug availability, proxied by insurance status, on health outcomes before and after the drugs arrived. The empirical strategy can be summarized in the following statement: if the availability of the drugs causes people on average to be more overweight (smoke more), then we should observe an increase over time in the overweight (smoking) differential across insurance status. Essentially this is a difference-in-difference analysis, with the two dimensions of comparison being insurance status (yes or no) and time (1971-75 versus 1988-94). Using this method of analysis, other health outcomes are also examined (high blood pressure, high cholesterol, and general self-reported health) in addition to overweight and smoking, in order to provide points of comparison.

In the regression framework the NHANES 1 and the NHANES 3 samples are combined, and the following equation is estimated (with overweight as the example health measure):

(2) $overweight_i = B_0 + B_1 X_i + B_2 (NHANES3_i) + B_3 (W_i * NHANES3_i) + B_4 (ins_i) + B_5 (ins_i * NHANES3_i) + e_i$

X_i is a set of demographic and socioeconomic control variables. NHANES3 is a dummy variable which is equal to one for observations from the NHANES 3 survey (post-influx of more effective drugs), and zero for observations from NHANES 1 (pre-influx of more effective drugs). W_i is a subset of X_i, including just education and income. These variables are interacted with the NHANES3 dummy because they are highly correlated with insurance status and might have a changing relationship with overweight over time.

The variable "ins" is a dummy for whether the person has any health insurance[6]. In this equation, the coefficient B_4 represents the change over time in the health differential across insurance status. The analysis is conducted separately for males and females. The data used are summarized in Table 5. The age group is limited to 25 – 64, for two reasons. First, persons under 25 are not included in the NHANES I sample. Second, there is very little variation in insurance status for people over 64, as they are all eligible for Medicare and therefore are almost all insured.

One potential concern with the empirical strategy is that the line of causation could run in both directions. Being insured when better blood pressure and cholesterol drugs are available could lead to increased probability of being overweight, but also being overweight could increase people's motivation to be insured when the better drugs are available. This concern should be kept in mind when interpreting the results. It should bias the estimated B_4 coefficient upwards.

A related concern is that the insured population, relative to the uninsured population, might have changed over time in significant ways that confound the relationship estimated in equation 2. To address

this issue, some basic characteristics of the insured and the uninsured are examined separately for NHANES I and NHANES III (Table 6). The table shows that, at least in terms of observable characteristics, insured versus uninsured differentials did not change much. The insured population, relative to the uninsured, became slightly more female, young, and nonwhite. The uninsured rate remained nearly constant (12.3 versus 12.9 percent).

V: Results

Probit regression results for equation 2 are shown separately for males and females in Tables 7 and 8, respectively. The results for the key variable for the hypothesis of this study, the interaction between insurance status and the time dummy (NHANES 3), are highlighted in grey in both tables. For this variable the estimated coefficients for overweight (column 1) and obesity (column 2) have the hypothesized positive sign for both males and females, but they are not statistically significant at the 95 percent confidence level. The coefficient for male smoking, on the other hand, is both positive and significant (Table 7, column 5). This result, taken together with the coefficient for the insurance status variable, suggests that insured males are 23.8 percent less likely to smoke than uninsured males in the early 1970s, but they are about equally likely to smoke in the late 1980s and early 1990s. The result for female smoking (Table 8, column 5) is not statistically significant.

The control variables in Tables 7 and 8 provide some interesting results in their own right. As people age, on average they gain weight from their 20s to their 50s, and then lose weight in their 60s.[7] Blood pressure and cholesterol rise with age, and smoking falls with age with the exception that it is flat for males from their 20s until their 40s. Self-reported general health falls with age. Nonwhites are more likely to be overweight or to have high blood pressure. Female nonwhites are less likely to have high

cholesterol than female whites, but male nonwhites are equally likely as male whites. Male nonwhites are more likely to smoke, but female nonwhites are equally likely. Female nonwhites are less likely to report having good or better health, and male nonwhites are equally likely.

Education and income are associated with better health outcomes for the most part. A notable exception is that more educated and higher-income males do not have lower cholesterol than other males. Similarly, higher income females do not have lower cholesterol than other females. In addition, the interactions of time with education and income reveal an interesting pattern for females. Overweight and obesity rates increased over time more for more educated and richer females than they did for other females. Finally, the time trend variable (NHANES3) for the most part shows expected trends: more overweight, obesity, and less high blood pressure and cholesterol. It also shows that smoking declined for men much more than it did for women, and self-reported health also improved much more for men than for women (for whom it actually became worse).

On the whole the results in Tables 7 and 8 provide very limited evidence for the hypothesis of this study. As noted above, only male smoking shows the hypothesized shift to riskier behavior for people who have access to the new drugs that control blood pressure and cholesterol. Also, it is interesting to note that high blood pressure and high cholesterol do not improve significantly for insured people over time. This fact might actually be explained by the hypothesis of this study, as people might offset their access to effective drugs with lifestyles that increase blood pressure and cholesterol. This interpretation would be more convincing if the behavioral changes showed up more in the overweight and obesity regressions.

A closer inspection of the relationship between insurance status and these health measures over time adds some possible clues. Tables 9 and 10 repeat the regressions of Tables 7 and 8, except that

the insurance-time interaction is now broken down by education level (less than high school, high school grad, and college grad). The tables show a pattern that is especially pronounced for males. For insured people, health measures (overweight, obesity, high blood pressure, high cholesterol, and smoking) become worse over time for the less educated, and better over time for the more educated. For overweight and obesity, one might interpret this result as suggesting that less educated people fall prey to the substitution of risks hypothesized of this study while more educated people do not. However, the results suggest that less educated insured people experience greatly increased rates of high blood pressure and high cholesterol, not just overweight and obesity. One possibility is that less educated people are unable to be their own advocates in a health care system that increasingly rationed resources with the rise of managed care. Another possibility is that government provided insurance, which is disproportionately consumed by the less educated and less affluent, declined in quality with respect to the care of these conditions.

V: Conclusion

In Section II, a simple theory was developed for how people respond to the availability of new, more cost-effective and well-tolerated drugs to lower blood pressure and cholesterol. The theory suggests that people should have shifted to lifestyles that are riskier with respect to heart disease, by controlling their weight less and smoking more, for example. This theory could help explain why the prevalences of high blood pressure and high cholesterol fell substantially starting in the late 1970s, while the prevalences of overweight and obesity rose substantially. The aggregate data in Section II demonstrate that these trends were tightly connected temporally.

However, individual level data analyzed in a difference-in-difference framework provides very limited evidence for the hypothesis. It is possible that insurance status is too crude a measure of access to the new, effective drugs. While not necessarily supportive of the study's hypothesis, an additional result that is notable is that insured people with less than high school education experienced worsening over time of health measures related to heart disease risk, whereas insured people with at least a high school education experienced improvements in these measures.

Although the evidence for the hypothesis of this study is minimal, it is worth discussing the implications if it were shown to be true, as might happen with better data or other methods. If people substitute between heart disease risks, should we really be concerned? People are merely maximizing their own utilities by choosing to engage in riskier lifestyles, so why should we worry how they respond to the new drugs? One source of concern is that patients, and even doctors, do not understand enough about how risk factors combine to result in a particular heart disease outcome. For example, perhaps, as suggested in Section III, having an equal probability of heart disease as someone else does not mean that the expected outcomes will be of the same gravity. That is, person A, who is overweight and using medication to control blood pressure, might have the same probability of heart disease as person B, who is not overweight and uses no medication, but person A could be at risk for more serious cases of heart disease. People tend to focus on numbers such as blood pressure level, but a certain blood pressure level achieved through a healthy diet is perhaps better than that same level achieved through an unhealthy diet and medication to lower blood pressure. This issues deserves attention from medical researchers, because there is a good possibility that people are making tradeoffs at "exchange rates" that are suboptimal for their bodies. Until we gain further understanding of heart disease risk, physicians

should emphasize, as they undoubtedly do in most cases, that lowering blood pressure or cholesterol through medications is not a perfect substitute for a healthy lifestyle.

Another implication would be that we should regard a drug's estimated efficacy in lowering heart disease risk in a clinical trial or other medical study with a certain amount of caution. We should consider the fact that many patients might increase their riskiness of lifestyle with respect to heart disease in response to the availability of the drug, much like people started to drive more recklessly as automobile safety equipment proliferated in the early 1970s, as shown by Peltzman. In cases where clinical trials carefully prescribe healthy lifestyles and diets, the real impact on health of a new wonder drug on heart disease risk will likely not be as great as the trial suggests. On the flip side of this issue, in cases where clinical trials do not prescribe healthy lifestyles and diets, we might not observe much improvement in the heart disease risks even though people might be enjoying substantial *utility,* if not health, benefits from the drugs (through substitution to riskier, higher utility lifestyles).

An important step for future study will be to quantify in a more precise and direct way the extent to which the hypothesized behavioral response to new drugs exists. A panel data set which extends for a period of several years would be ideal for this purpose. One could essentially compare how people behave after beginning to take medications to lower heart disease risk, versus how similar people who do not take the medications behave. Assuming that the drug interventions are not part of a randomized trial, some instruments, such as prescription drug insurance coverage, would be necessary to account for the non-random selection into drug treatment. Differences in government insurance coverage for drugs across time and geographic areas would probably address endogeneity concerns even more effectively. Another possibility would be to model carefully people's behaviors related to heart disease risk, choose reasonable preference parameters, and obtain quantitative data on the effectiveness and

patient's cost (including side effects) of drugs, and then run a simulation to predict behavioral responses. While this approach is less grounded in real data than an analysis using a panel data set, it is perhaps more feasible in the near future given the limited amount of detailed data available on individual health behaviors.

Notes:

[1] Most significant non-controllable factors are being male, age, and genetic background, according to the NHLBI.

[2] It should be acknowledged that Philipson and Posner (1999) mention a hypothesis of this sort in their paper.

[3] BMI is defined as height (meters) divided by the square of weight (kilograms)

[4] This rise might have been due to the fen-phen craze in 1995.

[5] In this simplified analysis, consumers are assumed to have access to drugs at very low cost. This is a reasonable assumption for people in many health plans, but of course it is not necessarily accurate for people in health plans with high medication co-payments or people with no health plan at all.

[6] Unfortunately the NHANES surveys do not contain sufficient information on prescription drug coverage to construct a corresponding dummy variable for both surveys.

[7] Of course, cohort effects might also be responsible for any health trends by age.

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Table 1: Trends in "controllable" heart disease risk factors for adults age 25-64, 1960-1994

	NHES (1960-62)	NHANES I (1971-75)	NHANES II (1976-80)	NHANES III (1988-1994)
overweight	0.281	0.271	0.273	0.330
obese	0.151	0.155	0.154	0.212
high blood pressure	0.292	0.375	0.371	0.135
high cholesterol	0.332	0.284	0.283	0.162
smokes	n/a	0.425	0.390	0.314
diabetes	0.0166	0.022	0.030	0.045

Notes: 1) variable definitions: overweight = 1 if bmi>27.8 for males or >27.3 for females; obese = 1 if bmi > 30; high bp = 1 if systolic bp > 140 mmHg or diastolic bp > 90; high chol = 1 if total cholesterol > 240 mg/dL.

Table 2: Drug mentions (in thousands) from office visits in the U.S.

	1980	1985	1990
Drugs to treat hypertension	38,463	39,011	47,309
Drugs to treat obesity	13,554	3,470	2,926

Source: National Center for Health Statistics.

Note: A "drug mention" is defined by the ordering or providing of a drug by a physician for a patient as a result of an office visit. Non-prescription drugs are included.

Table 3: Dispensed prescriptions (in thousands) in the U.S.

	1978	1983	1988
Drugs to lower cholesterol	4,396	2,549	12,900

Source: Wysowski et al, "Prescribed Use of Cholesterol Lowering Drugs in the United States." JAMA. April 25, 1990; Volume 263(16): 2185-2188.

Table 4: Dispensed prescriptions (in thousands) in the United States

Type of drug	1991	1992	1993	1994	1995	1996	1997
Blood pressure lowering	241,749	247,781	253,326	270,261	288,957	308,638	326,495
Cholesterol lowering	21,844	24,254	26,353	30,316	35,355	42,968	54,980
Anti-obesity	4,546	4,329	4,380	4,616	7,939	22,639	19,454

Source: National Data Corporation Health Information Services.

Table 5: Variable means, adults aged 25-64

	NHANES I, 1971-75 (N = 5,689)		NHANES III, 1988-94 (N = 10,725)	
variable	mean	S.D.	mean	S.D.
overweight	**0.273**	0.445	**0.364**	0.481
obese	**0.156**	0.363	**0.234**	0.423
high blood pressure	**0.387**	0.487	**0.149**	0.356
high cholesterol	**0.291**	0.454	**0.179**	0.383
smokes currently	**0.425**	0.494	**0.313**	0.464
self-reported health -- good or better	**0.828**	0.377	**0.861**	0.345
age	**43.774**	11.650	**41.289**	10.869
female	**1.499**	0.500	**1.516**	0.500
nonwhite	**1.109**	0.312	**1.164**	0.370
HS (but not college) grad	**0.525**	0.499	**0.553**	0.497
college grad	**0.157**	0.363	**0.235**	0.424
family income	**1.100**	0.601	**1.050**	0.619
insured	**0.877**	0.328	**0.871**	0.336

Notes: a) family income -- proportion of contemporary mean.

Table 6: Comparing the insured to the uninsured, adults aged 25-64

	NHANES I (1971-1975) N = 5,615				NHANES III (1988-1994) N = 10,189			
	Uninsured		Insured		Uninsured		Insured	
% of sample	12.3		87.7		12.9		87.1	
variable	mean	S.D.	mean	S.D.	mean	S.D.	mean	S.D.
female	**0.538**	0.499	**0.494**	0.500	**0.487**	0.500	**0.521**	0.500
age	**43.481**	12.291	**43.804**	11.570	**38.237**	10.397	**41.828**	10.869
nonwhite	**0.185**	0.389	**0.098**	0.297	**0.209**	0.407	**0.156**	0.363
education	**10.357**	3.474	**12.033**	2.963	**10.775**	3.625	**12.967**	2.912
family income	**0.690**	0.537	**1.159**	0.585	**0.581**	0.447	**1.135**	0.608

Notes: a) education -- highest grade level attained; b) family income -- proportion of the contemporary mean; c) % of sample does not correspond to N's, because it is calculated using appropriate sample weights.

Table 7: Males 25-64: impact of insurance status on health, "before" ('71-'75) & "after" ('88-'94)

model:	1	2	3	4	5	6
method:	probit	probit	probit	probit	probit	probit
# obs :	6,783	6,783	6,783	6,783	6,783	6,781
dep var:	Overweight?	Obese?	High BP?	High cholesterol?	Smokes currently?	In "good" or better health?
mean of dep var:	0.306	0.167	0.315	0.238	0.401	0.862
age in 20s	-0.2730*	-0.1968*	-1.0705*	-0.9028*	0.5715*	0.9697*
	(0.0714)	(0.0829)	(0.0758)	(0.0783)	(0.0723)	(0.0892)
age in 30s	-0.1992*	-0.0758	-0.6054*	-0.4456*	0.5917*	0.8220*
	(0.0650)	(0.0748)	(0.0649)	(0.0661)	(0.0673)	(0.0773)
age in 40s	0.0684	0.1410*	-0.3146*	-0.1670*	0.5517*	0.4092*
	(0.0645)	(0.0740)	(0.0641)	(0.0650)	(0.0674)	(0.0729)
age in 50s	0.1362*	0.2723*	-0.1662*	-0.0381*	0.3400*	0.1061
	(0.0659)	(0.0750)	(0.0651)	(0.0659)	(0.0688)	(0.0722)
nonwhite	-0.0146	0.0204	0.2563*	0.031	0.1264*	0.0081
	(0.0493)	(0.0549)	(0.0515)	(0.0536)	(0.0483)	(0.0613)
high school grad	-0.0180	0.0643	-0.0817	0.0487	-0.1228*	0.3763*
	(0.0570)	(0.0655)	(0.0551)	(0.0577)	(0.0546)	(0.0657)
HS grad * NHANES 3	0.0187*	-0.0856	0.0766	0.015	-0.049	-0.0998
	(0.0809)	(0.0908)	(0.0858)	(0.0877)	(0.0790)	(0.0955)
college grad	-0.2004*	-0.1361	-0.3070*	0.106	-0.6150*	0.4811*
	(0.0783)	(0.0943)	(0.0754)	(0.0774)	(0.0743)	(0.1047)
college grad * NHANES 3	-0.1241	-0.1291	0.1766	-0.1131	-0.1731	0.2533
	(0.1051)	(0.1222)	(0.1097)	(0.1109)	(0.1040)	(0.1479)
family income	0.2389~	-0.0145	-0.0899	0.191	-0.4260*	1.2625*
	(0.1061)	(0.1187)	(0.1080)	(0.1145)	(0.1021)	(0.1347)
family income * NHANES 3	-0.0581	0.1344	0.101	-0.0100	-0.0683	-0.0146
	(0.0622)	(0.0717)	(0.0644)	(0.0662)	(0.0608)	(0.0846)
family income squared	-0.0882*	-0.0556	0.0272	-0.0805*	0.1355*	-0.2678*
	(0.0378)	(0.0426)	(0.0389)	(0.0414)	(0.0366)	(0.0516)
NHANES 3	0.3216*	0.1317	-0.9331*	-0.3604*	-0.3955*	0.2954*
	(0.1058)	(0.1194)	(0.1139)	(0.1152)	(0.1030)	(0.1191)
insured	-0.0527*	-0.0434*	0.0194	-0.0932	-0.2379*	0.1723*
	(0.0789)	(0.0900)	(0.0766)	(0.0797)	(0.0751)	(0.0876)
insured * NHANES 3	0.0310	0.0959	0.0379	0.0815	0.2171*	-0.1498
	(0.1033)	(0.1173)	(0.1116)	(0.1126)	(0.1003)	(0.1208)
R-squared	0.023	0.026	0.112	0.049	0.064	0.178

Notes: 1) the reported coefficients are scaled to represent changes in probability w.r.t. one unit changes in the RHS variables; 2) * denotes coefficient is significant at 95% confidence; 3) dependent variables defined as in Table 1; 4) for age, the omitted category is the 60-64 age group; 5) for education, the omitted category is less than a HS education.

Table 8: Females 25-64: impact of insurance status on health, "before" ('71-'75) & "after" ('88-'94)

model:	1	2	3	4	5	6
method:	probit	probit	probit	probit	probit	probit
# obs :	7,979	7,980	7,980	7,980	7,980	7,979
dep var:	Overweight?	Obese?	High BP?	High cholesterol?	Smokes currently?	In "good" or better health?
mean of dep var:	0.323	0.215	0.227	0.231	0.327	0.832
age in 20s	-0.5786*	-0.5417*	-1.4819*	-1.2658*	0.5790*	0.6861*
	(0.0633)	(0.0684)	(0.0771)	(0.0700)	(0.0637)	(0.0758)
age in 30s	-0.3201*	-0.2395*	-1.1353*	-1.2394*	0.4563*	0.5579*
	(0.0554)	(0.0585)	(0.0612)	(0.0598)	(0.0584)	(0.0643)
age in 40s	-0.1715*	-0.2373*	-0.7569*	-0.620*	0.3791*	0.1637*
	(0.0560)	(0.0598)	(0.0593)	(0.0563)	(0.0595)	(0.0627)
age in 50s	0.0368	0.0347	-0.2518*	-0.2502*	0.2166*	0.0792
	(0.0564)	(0.0593)	(0.0578)	(0.0558)	(0.0603)	(0.0623)
nonwhite	0.2831*	0.2764*	0.3716*	-0.1155*	-0.0236	-0.1833*
	(0.0433)	(0.0452)	(0.0505)	(0.0515)	(0.0443)	(0.0497)
high school grad	-0.1159*	-0.0913	-0.0368	-0.0805	-0.1152*	0.4658*
	(0.0531)	(0.0576)	(0.0537)	(0.0548)	(0.0513)	(0.0576)
HS grad * NHANES 3	0.1598~	0.0967	0.0431	0.0402	-0.1583*	0.1474
	(0.0758)	(0.0805)	(0.0883)	(0.0842)	(0.0749)	(0.0834)
college grad	-0.4982*	-0.4961*	-0.2251*	-0.2272*	-0.4823*	1.0387*
	(0.0963)	(0.1135)	(0.0903)	(0.0900)	(0.0827)	(0.1320)
college grad * NHANES 3	0.1824	0.1154	-0.0066	-0.0316	-0.2025	-0.263
	(0.1199)	(0.1372)	(0.1340)	(0.1238)	(0.1113)	(0.1616)
family income	-0.2995*	-0.3846*	-0.3374*	0.0073	-0.2229*	1.0639*
	(0.0928)	(0.0992)	(0.1070)	(0.1017)	(0.0937)	(0.1093)
family income * NHANES 3	0.1323*	0.1372*	0.1477*	0.1134	-0.098	0.1524*
	(0.0583)	(0.0642)	(0.0665)	(0.0626)	(0.0564)	(0.0704)
family income squared	-0.0054	0.0335	0.0402	-0.0343	0.0609	-0.3153*
	(0.0357)	(0.0384)	(0.0420)	(0.0391)	(0.0358)	(0.0437)
NHANES 3	0.0855	0.0425	-0.9217*	-0.3790*	-0.0313	-0.1852~
	(0.0927)	(0.0970)	(0.1083)	(0.1043)	(0.0918)	(0.0988)
insured	-0.1764*	-0.1892*	-0.0611	-0.0027	-0.0395	0.1446*
	(0.0682)	(0.0725)	(0.0702)	(0.0715)	(0.0659)	(0.0729)
insured * NHANES 3	0.0282	0.1282	-0.0631	-0.0379	-0.0391	0.0215
	(0.0930)	(0.0982)	(0.1094)	(0.1056)	(0.0921)	(0.1023)
R-squared	0.063	0.06	0.196	0.124	0.044	0.154

Notes: 1) the reported coefficients are scaled to represent changes in probability w.r.t. one unit changes in the RHS variables; 2) * denotes coefficient is significant at 95% confidence; 3) dependent variables defined as in Table 1; 4) for age, the omitted category is the 60-64 age group; 5) for education, the omitted category is less than a HS education.

Table 9: Males 25-64: impact of insurance status on health, "before" ('71-'75) & "after" ('88-'94)

model:	1	2	3	4	5	6
method:	probit	probit	probit	probit	probit	probit
# obs :	6,783	6,783	6,783	6,783	6,783	6,781
dep var:	Overweight?	Obese?	High BP?	High cholesterol?	Smokes currently?	In "good" or better health?
mean of dep var:	0.306	0.167	0.315	0.238	0.401	0.862
other controls in Table X	yes	yes	yes	yes	yes	yes
NHANES 3	0.1962	0.0101	-1.2457*	-0.5665*	-0.4273*	0.2574
	(0.1218)	(0.1389)	(0.1482)	(0.1433)	(0.1174)	(0.1321)
insured	-0.0517	-0.0422	0.0200	-0.0919	-0.2376*	0.1725*
	(0.0789)	(0.0900)	(0.0766)	(0.0797)	(0.0751)	(0.0876)
insured * NHANES 3	0.1981	0.2558	0.4271*	0.3462~	0.2600*	-0.0993
	(0.1314)	(0.1492)	(0.1587)	(0.1546)	(0.1268)	(0.1436)
insured * NHANES * HS grad	-0.1319	-0.1612	-0.5630*	-0.2846	-0.0331	-0.0821
	(0.1420)	(0.1601)	(0.1775)	(0.1736)	(0.1390)	(0.1687)
insured * NHANES * college grad	-0.7629*	-0.6090*	-0.7663*	-0.8151*	-0.2309	-0.2882
	(0.2008)	(0.2225)	(0.2401)	(0.2284)	(0.2100)	(0.3756)
R-squared	0.0250	0.0270	0.1140	0.0510	0.0640	0.1780

Notes: 1) the reported coefficients are scaled to represent changes in probability w.r.t. one unit changes in the RHS variables; 2) * denotes coefficient is significant at 95% confidence; 3) dependent variables defined as in Table 1; 4) for education, the omitted category is less than a HS education.

Table 10: Females 25-64: impact of insurance status on health, "before" ('71-'75) & "after" ('88-'94)

model:	1	2	3	4	5	6
method:	probit	probit	probit	probit	probit	probit
# obs :	7,979	7,980	7,980	7,980	7,980	7,979
dep var:	Overweight?	Obese?	High BP?	High cholesterol?	Smokes currently?	In "good" or better health?
mean of dep var:	0.323	0.215	0.227	0.231	0.327	0.832
other controls in Table X	yes	yes	yes	yes	yes	yes
NHANES 3	0.0866	0.0224	-1.0488*	-0.4268*	-0.1649*	-0.1487
	(0.1093)	(0.1137)	(0.1338)	(0.1251)	(0.1085)	(0.1125)
insured	-0.1763*	-0.1889*	-0.0606	-0.0026	-0.0395*	0.1448*
	(0.0682)	(0.0725)	(0.0702)	(0.0715)	(0.0659)	(0.0729)
insured * NHANES 3	0.0264	0.1541	0.1044	0.0252	0.1422	-0.0305
	(0.1217)	(0.1267)	(0.1494)	(0.1396)	(0.1206)	(0.1259)
insured * NHANES * HS grad	0.0108	0.0108	-0.2245	-0.0841	-0.3017*	0.1294
	(0.1310)	(0.1365)	(0.1731)	(0.1590)	(0.1318)	(0.1432)
insured * NHANES * college grad	-0.0456	-0.3740	-0.5786*	-0.2443	-0.2816	-0.1615
	(0.2306)	(0.2383)	(0.2845)	(0.2793)	(0.2452)	(0.3183)
R-squared	0.063	0.061	0.197	0.124	0.045	0.154

Notes: 1) the reported coefficients are scaled to represent changes in probability w.r.t. one unit changes in the RHS variables; 2) * denotes coefficient is significant at 95% confidence; 3) dependent variables defined as in Table 1; 4) for education, the omitted category is less than a HS education.

Figure 1: Simple model of response to availability of new, effective drugs