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Production of and the demand for coronary health

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PRODUCTION OF AND THE DEMAND FOR CORONARY HEALTH

by

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INTRODUCTION

Since the early 1970's many published reports have linked moderate alcohol consumption and lower mortality due to coronary heart disease. Yet the biological explanations for this association have not gained general acceptance from the medical community. Because of the lack of an unaccepted biological mechanism that would relate moderate alcohol consumption to lower mortality due to coronary heart disease, certain medical researchers have speculated that moderate alcohol consumption is related to some unknown third variable which is responsible for the observed lower mortality due to coronary heart disease. One possible confounding variable is moderate lifestyle. Individuals who eat, drink, and exercise moderately and avoid stress may be said to live a lifestyle that promotes cardiovascular health. This total lifestyle may be the cause of lower mortality due to coronary heart disease, not just the alcohol consumption.

Another explanation of the link between moderate drinking and lower mortality due to coronary heart disease involves the idea that healthier people consume alcohol and unhealthy individuals refrain from drinking alcoholic beverages for fear that consumption will worsen health. If this is the case, then there would be a positive link between alcohol consumption and lower mortality due to

coronary heart disease, but it would not be causal from alcohol to lower mortality.

The above concerns about the moderate drinking - lower mortality due to coronary heart disease relationship are important in light of the recent notoriety of alcohol abuse and especially drunk driving. In today's society there is great sentiment running against alcohol abuse and many studies have suggested that an alcohol excise tax policy may be effective in curbing this problem.[1] Another reason for considering an alcohol excise tax policy is the current need by government to enhance revenues to offset a considerable budget deficit. Any alcohol excise tax policy should consider any benefits that alcohol consumption may have on cardiovascular health.

The research done here investigates the moderate alcohol consumption - lower mortality due to coronary heart disease relationship using data from the National Health and Nutrition Examination Survey and the ten year follow-up, plus additional sources. A two stage recursive model is fit to estimate the impact of moderate alcohol consumption on mortality from several causes. With a two stage approach the questions of causality and confounding variables will be addressed.

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[1] For a discussion of alcohol consumers sensitivity to price see D. Coate and M. Grossman, "Effects of Alcoholic Beverage Prices and Legal Drinking Ages on Youth Alcohol Use", Journal of Law and Economics, XXXI (April 1988).

CHAPTER I

The evidence reporting a negative association between "moderate" alcohol consumption and mortality due to coronary heart disease is extensive. This evidence comes in the form of a variety of published analyses, including autopsy, ecological, case control and cohort studies.

In a thorough review of the literature on this topic, Moore and Pearson (1986) report:

> The evidence indicates that a potentially protective level of alcohol consumption exists between the extremes of abstention and drinking in excess of 3 to 4 drinks per day. The strength of existing evidence makes new and expensive population based studies of the association of alcohol consumption and CAD unnecessary.[2]

One of the more influential studies establishing the "moderate" drinking, coronary heart disease negative relationship is a cohort study performed by Yano et al. (1977). This study, which centered on Japanese men born between 1900 and 1919 who were residing in Hawaii at the time of the study, found coronary heart disease and mortality due to coronary heart disease to be lowest in a group of individuals comprised of the heaviest drinkers. This group was made up of men who consumed three or more

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[2] Moore, R. D. and Pearson, T. A. "Moderate Alcohol Consumption and Coronary Heart Disease." Medicine, 65, p. 263

drinks of alcohol per day. Mortality due to coronary heart disease in the heaviest drinking group was one half the mortality due to the same cause of death in the grouping of those that abstained from alcoholic beverage consumption. These mortality rates were age adjusted. This study was especially interesting in that it addressed the question of causality in the relationship involving alcohol consumption - mortality due to coronary heart disease. Most studies indicate that "moderate" drinking causes lower mortality rates due to coronary heart disease rates. However, the possibility exists that the relationship between alcohol consumption and lower mortality due to coronary heart disease is that causality runs from good health to drinking. In other words, it is possible that individuals who have some existing medical problem will not consume alcohol. Yano et al. tested for the possibility that current non-drinkers were one time drinkers who stopped consuming alcohol because of latent coronary heart disease. The authors computed age adjusted incidence rates of coronary heart disease for lifetime abstainers, former drinkers, and current drinkers. They found current drinkers to have a statistically significant difference in rates of coronary heart disease than abstainers or former drinkers. The two latter groups were not found to have statistically different rates. From this they concluded that the higher incidence of coronary heart disease in non-drinkers could not be attributed to the

possible existence of latent coronary heart disease in non-drinkers. Since former drinkers and lifetime abstainers had similar mortality rates.

Dyer, Stamler et al. (1980) in a study of 1832 white males who participated in the Chicago Western Electric Company Study, examined the effect of alcohol consumption on coronary heart disease mortality. The individual participants were aged between 40 and 55 years at the time of the baseline interview in 1957. These men were then followed for seventeen years. Age adjusted mortality rates were lower for individuals who consumed approximately two to five drinks per day. The mortality due to coronary heart disease for this group was about eighty five percent of the mortality due to the same cause for those participants who consumed less than one drink per day. The heaviest drinkers, those who consumed more than six drinks per day were found to have coronary heart disease mortality rates double those who drank two to five drinks a day and approximately eighty seven percent of the coronary heart disease mortality rates of non-drinkers. Dyer et al. also calculated age adjusted death rates due to all causes and found that those who consumed six or more drinks per day had mortality rates two to three times the mortality rates of those who drank less.

In another study involving 2015 white men and women from the San Fransico area, Klatsky et al. also investigated the coronary heart disease mortality - alcohol consumption

negative relation. Individuals were selected to participate from 182,357 persons of all races who were members of the Kaiser Foundation Health Plan. The 2015 subjects were divided into four categories based on their alcohol consumption per day. The four groups consisted of those who drank zero drinks per day, those who consumed greater than zero but less than or equal to two drinks daily, those who consumed three to five drinks per day and those who consumed six or more drinks daily. This study followed the sample from 1966 to 1976. Ten year mortality rates for the groups were computed. These group mortality rates, interpreted as percent that died, were calculated by the authors. They found mortality attributed to cardiovascular diseases for the total of men and women to be 4.4, 3.2, 4.1, and 3.8 across the drinking categories. Results reported for men and women separately also found lower death rates for the moderate drinking groups (greater than zero to two, three to five). The authors pointed to alcohol's purported stimulation of high-density lipoprotein (HDL) levels. Accepted medical thought considers a larger ratio of HDL cholesterol to LDL cholesterol to have beneficial effects on cardiovascular health. Klatsky et al. also reported that the group with the lowest mortality due to coronary heart disease (more than zero but less than or equal to two) had the highest education levels. This last finding is of interest in the sense that it provides an

explanation for a beneficial effect on cardiovascular health that is not caused by alcohol. Moderate alcohol consumption, in this explanation, may be a correlate of education or some other variable which in fact causes the observed lower mortality due to coronary heart disease.

Yet another report citing the beneficial effects of alcohol on cardiovascular health was done by Kozarevic et al. (1982). This study investigated the incidence of myocardial infarction or non-sudden heart disease deaths in a sample of Yugoslav males who resided in urban settings and were drawn from the Yugoslavia Cardiovascular Disease Study. The authors reported that death rates due to myocardial infarction were twice as high in those who rarely or never drank as in drinkers who consumed at least one drink per day.

Kittner et al. (1983) in studying Puerto Rican males reported that the rate of non-sudden coronary heart disease mortality among drinkers who consumed one to three drinks per day was approximately seventy percent of the rate of abstainers and heavy drinkers. This study also found that total mortality rates (due to all causes) of the moderate drinkers were about ninety percent of the non drinkers and heavy drinkers. Mortality rates were age adjusted and were controlled for smoking and physical activity. Kittner et al. also reported that for individuals over the age of sixty or below the median income, the negative association between

alcohol intake and mortality due to coronary heart disease was not seen. The authors could not explain these findings by differences in smoking habits, diet, or by urban versus rural dwellings. They suggested that unmeasured health care differences were responsible for their findings on income and age.

Gordon and Kannel (1983,1984) using the Framingham heart study data also investigated the relationship between alcohol consumption and mortality. The famous Framingham study amassed data on 5209 participants who have been tracked since 1950. This study is considered by many health scientist to be among the most useful sources of information involving cardiovascular health in the United States. The 5209 persons who originally participated in the study represented about half the population (men and women) aged 30 to 62 of this Massachusetts community. Almost all of those who participated were in good health at the start of the study. Gordon and Kannel found that individuals who consumed up to two drinks per day had lower mortality due to coronary heart disease. They reported that this consumption level of alcohol was also associated with lower mortality from causes other than coronary heart disease. Mortality due to causes other than coronary heart disease is dominated by stroke and cancer deaths. There is strong medical evidence that links alcohol consumption positively with certain cancers. This interesting result again brings up the

question of direction of causality or the notion that moderate drinking is a correlate of some third unknown variable which is responsible for the lower mortality rates.

These thought provoking questions involving a non causal or reverse causal explanation of the observed link between moderate alcohol consumption and lower mortality due to coronary heart disease are especially profound in light of Moore's and Pearson's (1986) conclusion that in none of the studies that they reviewed could the authors find a precise biological mechanism to explain the well documented negative relationship between moderate alcohol consumption and lower mortality due to coronary heart disease. Although most studies that they reviewed point to alcohol's ability to affect blood serum cholesterol ratios, Moore and Pearson found no convincing evidence of this. Kittner et al. (1983) also concluded that it is questionable whether or not alcohol raises the HDL-subfraction which is considered to have a beneficial effect on cardiovascular health.

The study which this researcher undertakes to investigate the alcohol - coronary heart disease mortality relationship, makes use of the household production function approach to consumer behavior developed by Becker (1965). Before this enlightening work, consumer demand was based on the idea that every consumer made choices in the market place based on the level of satisfaction that they enjoyed through the consumption of different goods and services.

This theory put forth the idea that everyone has a utility function which allows them to choose between alternative purchases. Every good and service consumed gives a certain utility or satisfaction. Therefore, consumers choose that combination of goods and services that yields the greatest utility. Of course, market choices made by consumers do not depend completely on the levels of utility derived from consuming different goods and services. Consumers are constrained by the resources they have to purchase these goods and services and by the prices they must pay. To account for these constraints, traditional demand theory proposes that consumers maximize a utility function subject to constraints imposed by resources and prices. While this notion of consumer behavior explains satisfactorily the demand for many goods and services, it falls short in explaining the demand for certain things that individuals consume and demand such as recreation and good health. For example, one does not just purchase recreation and consume it. It must be produced by the consumer using market purchases plus his own time inputs. Becker theorized that what ultimately affects consumers' utility is not purchases of market goods and services, but what he called "commodities". These "commodities" produced by the consumer, with inputs of time and market goods and services, are the objects of choice that enter the utility function. This is analogous to the notion of the firm taking inputs and

producing outputs. Therefore, in the Becker framework it is not the fishing pole, bait, and boat rental that give utility, but the afternoon spent fishing which is produced using the items along with the fisherman's own time.

This useful theory was used by Grossman (1972a,b) in developing a theory of production and demand for health. It is easily seen how the household production function approach to demand theory is useful in studying the economics of health. The object that consumers desire is not CAT scans or visits to their doctor, which are purchased in the market, but good health. Good health is produced using these inputs, so demand for doctor visits is demand for an input into the production of good health. Grossman's work was the first to make use of this insight. In Grossman's research, health was produced using inputs of medical care, diet, exercise, cigarette smoking, alcoholic beverage consumption and a measure of one's own ability or the ability of a spouse to produce health. Grossman noted that certain individuals were more efficient at producing health with given inputs, just as certain technologies used by the firm to produce output have differing levels of efficiency. Factors that affect health production efficiency, as argued by Grossman, include personal characteristics such as age, race, sex, education, and genetically endowed health.

Grossman maximized a utility function containing health subject to resource constraints and obtained demand

functions for the inputs used in the production of health. Demand for the inputs is a function of income, prices, taste (certain individuals are more willing than others to give up activities such as smoking in order to obtain greater levels of health), health endowment, (the greater the endowment of health, the less demand for other inputs such as medical care), and efficiency of production of health. Since Grossman's significant contribution to the study of health economics, his model has been widely used and is the basis of the model used in this study.

Education, as an input in the production of health, has received much attention. Grossman's initial work on the demand and production of health (1972) suggested that more years of schooling made an individual a more efficient producer of health. The efficiency may act through making the individual, who obtains more schooling, more knowledgeable about choices that affect health (medical care, smoking, etc.).

Grossman (1976), in another investigation of the relationship between schooling and health production, studied schooling's affect on health of middle aged men. He found a statistically significant affect of schooling on health after controlling for income, high school health status, family background, and scores on physical and mental test that participants took when they were in their twenties. Grossman concluded from this study that more

schooling does make an individual a more efficient producer of health. This finding is of tremendous importance when considering national health policy. If more schooling makes individuals better producers of health than a clear way to improve the health of the citizens of a country is through increasing schooling levels.

Fuchs (1982) argues that education's well documented correlation with health may result from the fact that schooling and health may be correlated to some third variable which has yet to be identified. In this case, more schooling would not make an individual necessarily a more efficient producer of health. Fuchs considered that the cofactor related to both schooling and health was an individual's time preference. In this explanation, if an individual is more future orientated, that is, more interested in future consumption than another person, then this individual is expected to invest more heavily in things that provide greater benefits in the future. Greater schooling levels provide greater income opportunities in the future; greater health investments, everything else equal, provide the opportunity for better health in the future. Therefore, as suggested by Fuchs, differences in time preferences account for the correlation of education and health.

Fuchs tested differences in time preferences' effects on education and health, using various psychological

measures. He reported results showing time preference to be related to both schooling and health investment. His evidence was not strong and Fuchs questioned whether the results would have been stronger had more accurate measures of variables been used. While Fuchs presents an interesting argument, this researcher assumes education to have an affect on health. However, following Rosenzweig and Schultz (1982), who argue that education effects health production through schooling's ability to allow the producer of health to make choices involving health inputs, the work done by this researcher will include education levels in the demand for health input equations only and will not include education directly in the health production function.

Following Grossman's health production model, Coate and Grossman (1989) investigated the alcohol - mortality due to coronary heart disease negative relationship. Using data from the National Health and Nutrition Examination Survey (NHANES1) (1971-1974) and the follow-up (1982-1984) (which tracked the original participants) they fit a multinomial logit mortality production function for white males and females. The study involved four mortality outcomes: (1) the subject died from myocardial infarction in the approximately ten years from NHANES1 to the follow-up, or (2) the subject died from stroke during this period, or (3) the subject died from another cause during this period, or (4) the subject survived. Right hand side variables used in this study

included number of cigarettes smoked, blood pressure measurements, age, body mass, and alcohol consumption variables which were represented by dummy variables indicating heavy drinking and moderate drinking. The dummy variable construction, indicating alcohol consumption, was an interesting way for the authors to study the so called U-shaped relationship between alcohol consumption and mortality due to coronary heart disease. As mentioned in most studies reported here, the beneficial effect of alcohol intake on cardiovascular health exists between the extremes of abstention and heavy consumption levels. Another interesting aspect of Coate's and Grossman's work was the inclusion of stroke deaths to the mortality outcomes. Most other studies looked at the relationship between alcohol and ischemic heart disease mortality versus mortality due to all other causes. Coate and Grossman argue that there exists substantial evidence that indicates alcohol raises blood pressure, which not only has a negative effect on heart health but is also positively associated with higher incidence of stroke. In their study they tried to identify alcohol's relationship with deaths due to stroke and alcohol's possible effects upon blood pressure.

Coate's and Grossman's results indicate a negative relationship does exist between moderate alcohol consumption and mortality due to coronary heart disease. They also found moderate and heavy drinking to be negatively related to

deaths from causes other than coronary heart disease and that this negative relationship also existed between moderate and heavy drinking and deaths due to strokes. It was this result that led the authors to question whether drinkers consisted of individuals who were healthier, and abstainers consisted of individuals who had health problems. It is from this point that the study undertaken here initiates.

CHAPTER II

Following the household production function approach to health economics in Grossman's study (1972), a mortality production function, similar to that used by Coate and Grossman (1989) is used to study mortality due to coronary heart disease. In the model used in this study, mortality is produced by inputs of alcoholic beverage consumption (A), number of cigarettes smoked (C), and a vector of exogenous socioeconomic and health measure variables (Z). Examples of the socioeconomic variables include income and age, while the health measures are represented by blood pressure measurements and levels of exercise. The functional form of this mortality production is given by the following:

$$M = m(A,C,Z) \qquad (1)$$

Alcohol and cigarette consumption have long been considered by the medical profession to have negative health impacts, although as mentioned earlier, "moderate" consumption of alcohol has been strongly linked to lower mortality due to coronary heart disease. Exercise is considered to be beneficial to cardiovascular well being, which therefore indicates that it should be negatively related to mortality. Income is an economic variable which indicates the ability to purchase medical services and other

goods and services which may have either a positive or negative impact on health, and age, in a Grossman (1972) health investment context, represents a measure of health depreciation. High blood pressure is a well documented risk factor in mortality due to strokes and ischemic heart disease (coronary heart disease).

It is readily seen that the estimation of (1) leads to problems, since alcohol consumption and cigarette smoking are not predetermined variables but are endogenous, and rely on other factors. Coate and Grossman (1989), in their work using a mortality production function, found certain interesting results involving alcoholic beverage consumption's effect on mortality outcomes. They found "moderate" and "heavy" alcoholic beverage intake to result in lower mortality due to coronary heart disease. This finding was not completely surprising, although most previous epidemiological studies show a beneficial affect on cardiovascular health from only "moderate" consumption of alcoholic beverages. More confounding however, was their reporting of "moderate" and "heavy" alcohol consumption to be associated with lower mortality due to stroke and all other causes of death besides coronary heart disease and stroke. Causes of death other than stroke and coronary heart disease are dominated by cancers. There is abundant medical literature that exists establishing a positive link between alcoholic beverage consumption and certain cancers. Cancer

of the esophagus and of the liver are examples of cancers positively associated with consumption of alcohol. With this result the authors questioned the direction of causality in the mortality production function. They speculated that certain individuals may have existing medical conditions such as high blood pressure, ulcers, gastrointestinal disorders and other diseases that, with or without medical advice, might lead the individual in question to abstain from alcohol intake. If this were the case, then alcohol consumption's negative impact on mortality outcomes would be overestimated since only a healthier sample undertook drinking of alcohol. A healthier sample should be expected to have lower mortality rates than a sample containing individuals who are healthy as well as in poor health. In essence Coate and Grossman questioned the direction of causality. Does causality run from drinking alcohol to good health or does it flow in "reverse" from good health to alcohol consumption?

Treating alcoholic beverage consumption as an endogenous variable, estimating a demand function for it and then using the predicted values of alcoholic beverage consumption generated by this demand equation in the mortality production function, is one way of eliminating the problem of unobserved, unhealthy individuals self selecting themselves into abstention from alcoholic beverage consumption based on their health status. Using this two

stage process, the question of reverse causality running from good health to drinking of alcohol can be addressed. Of course, if health status is correlated with any of the right hand side variables in the alcohol input demand equation, then the problem of individuals not drinking alcohol due to health conditions would not be solved by using the predicted value of alcoholic beverage consumption in the mortality production function.

As mentioned above, alcohol consumption is not the only endogenous variable to appear on the right hand side of the mortality production function. Number of cigarettes smoked should also be considered to be determined by other variables. Therefore, in this work, both alcoholic beverage intake and smoking of cigarettes are fit with the following demand functions:

$$A = a(Pa, Pc, X) \qquad (2)$$

$$C = c(Pc, Pa, X) \qquad (3)$$

The above equations show the demand for the inputs, alcohol and cigarette consumption, that are used in the production of mortality.

In these equations, consumption of both inputs depend upon own prices, cross prices, and a vector of socioeconomic variables such as income, age, education, and drinking

sentiment. Education as considered by Rosenzweig and Schultz (1982) is assumed to enter the input demand equations for alcohol and cigarette use. It is assumed the more education an individual has, the greater the awareness of that individual to the hazards of consuming these products. The drinking sentiment variable is based on the probability of the individual's being a member of a certain religion. For example, Mormons and Southern Baptists have tenets in their religion prohibiting the use of alcohol. Therefore, states with high percentages of these two groups can be considered to have attitudes reflecting their religious beliefs.

Once equations 2 and 3 are estimated, one can obtain predicted values of alcoholic beverage consumption ($\hat{A}$) and cigarette smoking ($\hat{C}$). These predicted values are then used in the estimation of the mortality production function.

$$M = m(\hat{A}, \hat{C}, Z) \qquad (4)$$

This recursive system is used to study the impact of alcohol intake on mortality outcomes. The estimation procedure, which will be addressed in a future chapter must take account of the high probability that this recursive system contains correlated errors. It is likely that the error terms in the input demand equations are correlated with the error term in the mortality production equation. In the following system of estimated equations, the input

demand equations (5,6) are likely to have error terms which are correlated with the disturbance term from the mortality production equation (7):

$$A = a_0 + a_1 Pa + a_2 Pc + BX + E \qquad (5)$$

$$C = b_0 + b_1 Pa + b_2 Pc + BX + U \qquad (6)$$

$$M = c_0 + c_1 A + c_2 C + BZ + V \qquad (7),$$

In the above equations, a,b and c are parameters, B is a vector of parameters, and E,U and V are error terms for their respective equations.

The correlation of these disturbance terms arises because unobserved genetically endowed health would likely affect alcohol consumption and cigarette smoking, and it would definitely have an impact on mortality. That is, individuals who are healthier due to genetic endowment may be less inclined to stop consumption of alcohol and cigarette smoking for health reasons then an individual who has less of a genetic endowment of health; while at the same time, a greater initial health endowment is most certainly associated with lower mortality, holding all else constant. Since genetic health endowment is unobserved it is captured

in the error components of each equation. Therefore, an estimation procedure other than OLS will be used to estimate the mortality production function.

CHAPTER III

The data used in this study is derived from several sources, the bulk of which comes from the National Health and Nutrition Examination Survey, NHANES1 and the National Health and Nutrition Epidemiological Follow-up Study, NHEFS (this is a follow-up to NHANES1).

The former was a survey instituted by the National Center for Health Statistics (NCHS). It is a stratified probability sample of the non-institutionalized population of the contiguous United States, excluding persons living on Indian reservations. This survey contains data on over twenty eight thousand individuals, which includes 20,729 individuals between the ages of 25 and 74 years, collected from April of 1971 through June, 1974.

The NHANES1 survey includes data on medical examinations performed on a sub sample of participants (roughly 70 percent of participants aged 25-74) by physicians with the aid of nurses. It also includes medical histories, questionnaire responses containing dietary and socioeconomic information, and certain results from bio medical laboratory analysis of blood and urine. The responses to the questionnaires and the medical history information were obtained through interviews with the participants by a trained employee of the NCHS. The interviews were conducted at the participants' residences.

The interviewers were trained to encourage the interviewees to respond to all questions honestly.

The NHANES1 was designed so that certain segments of the U.S. population were over sampled. The targeted groups were thought to be at high risk for malnutrition and health problems. These high risk groups were made up of low income earners, women of childbearing age, and the elderly.

The data was weighted to adjust the sample to closely resemble the U.S. non-institutionalized population aged 1 through 74 years.

The data from NHANES1 relevant to this work includes information on alcohol consumption, blood pressure readings, age, race, sex, family income, education attainment, exercise and physical activity, and medication usage as well as information on certain health conditions.

The NHEFS, the follow-up study contains data on approximately thirteen thousand individuals who were aged 25 to 74 years at the time of NHANES1 and who took part in the medical examination. The follow-up survey took place during the period 1982-1984. These thirteen thousand individuals represented approximately ninety five percent the 14,407 persons who underwent the medical examination as part of NHANES1. Of the thirteen thousand eligible NHANES1 participants who were traced as part of the follow-up, 12,220 or ninety one percent completed the follow-up survey.

The data in the follow-up was collected mostly through

interviews with NCHS employees and whenever possible at the subjects' home. In some cases interviews were conducted by telephone.

Approximately two thousand of the eligible NHANES1 participants had died in the roughly ten year period between NHANES1 and NHEFS. In these cases proxy respondents were used to obtain information on the deceased subject. The proxies were usually spouses or other relatives but in some cases neighbors of the deceased were queried. Death certificates were used to obtain the cause or causes of death. The NHEFS also contained blood pressure and pulse readings. Important data for this work includes information obtained from NHEFS on cigarette smoking and mortality. Cigarette smoking data was also available from NHANES1 but was only asked of approximately five thousand respondents who went through a more rigorous medical examination. Smoking data was available for almost all of NHEFS participants.

The follow-up to the NHANES1 contains data on 4336 white males and 6,041 white females. Since race is considered to be a factor in mortality due to coronary heart disease, non whites are excluded in this study because they represent a relatively small number of participants to provide statistically significant results. The difference in sample size of white females to white males is explained by the fact that NHANES1 over sampled the older non-

institutionalized population. Females have a longer life expectancy than males so samples skewed to the older population would naturally contain more females. Another explanation for this discrepancy lies in an over sampling of lower income individuals. Evidence abounds indicating a larger proportion of females to males populating the lower income groups relative to what is found nationally.

Causes of death that were coded in NHEFS and are used in this study come from the National Center for Health Statistics' ninth revision of the 72 causes of death (ICD72).

Other data sources used in this research are alcoholic beverage prices from the Liquor Handbook published by Gavin-Jobsin. These prices include state excise taxes on distilled spirits, and prices are the manufactures' suggested retail prices. Prices inclusive of state excise taxes are used for distilled spirits because certain so called "monopoly" states only permit sales of distilled spirits, and in some cases wine, through state operated stores. These states obtain distilled spirits and wine sales revenue not from excise taxes but through a mark up on price. Distilled spirits sales data by state were obtained through the Annual Statistical Review of the Distilled Spirits Council of the U.S..

State specific cigarette prices and cigarette sales by year were obtained from the Tobacco Tax Council (1986).

Cigarette prices are a weighted average of a pack, accounting for the types of cigarette (regular, king, 100mm) and the type of purchase (carton, single pack, machine). These prices include state and municipal excise taxes.

Data on religious affiliation was obtained from a county specific survey and pertains to 1980. This survey was conducted by the National Council of Churches of Christ and the Glenmary Research Center.

CHAPTER IV

The econometric implementation of the model used in this study follows a two stage process. The first stage fits demand equations for the inputs alcoholic beverage consumption and cigarette smoking that are used in the mortality production function. These equations take the following form:

$$C = c_0 + c_1 Pa + c_2 Pc + B_1 X \qquad (8)$$

$$A = a_0 + a_1 Pa + a_2 Pc + B_2 X \qquad (9)$$

In the above equations a_1 a_2 c_1 and c_2 are parameters, B_1 and B_2 are vectors of parameters and a_0 and c_0 represent intercept terms.

In equation (8) C represents lifetime number of cigarettes smoked for individual j derived from NHEFS. Cigarette prices (Pc), which were discussed earlier, are state specific and by year. Alcohol prices (Pa), also mentioned earlier, are state and beverage specific by year. A vector of exogenous variables (X) includes a measure of community tolerance towards consumption of alcohol and smoking. This measure of sentiment is indicated by the probability of being a member of a certain religion. In the

NHANES I data there is information naming the site where each individual was examined by a physician. The medical examinations took place at one hundred NHANES I test sites. Subjects went to the site closest to their residence. Religious membership data was available by county. Test site counties and surrounding counties were grouped into a test site geographical unit, which was matched to religious information for the same area. The religion data took the form of church membership. Probability of being a member of a specific religious denomination was calculated by taking the number of adherents to a particular religion in a given NHANES I site area and dividing by the total population for the site area. Church membership data was available for 1980 only. It is likely that from the years of NHANES I to 1980 religious affiliation did not change appreciably.

Other variables considered to be exogenous and to have an effect on smoking and cigarette consumption that are used in this study are divorce and unemployment rates. These variables are included to capture the effects that stressful social situations may have on cigarette smoking and alcohol consumption. It is assumed that the greater the divorce and unemployment rates, the greater the per capita consumption of alcohol and the greater the number of cigarettes smoked.

In this study divorce rates are available by state and by year and represent the percentage of women aged 25 through 34 years who are divorced. These values were

obtained from State Census Volumes in census years. During intercensal years the figures were obtained using an exponential growth rate, and adjusted so that a weighted average of intercensal years was equal to the observed national rate during this time. State specific unemployment rates were obtained by year from the Manpower Report of the President.

Certain state specific per capita sales data for cigarettes and alcohol (by type) as described earlier were also used in some specifications of the input demand equations.

The cigarette equation was estimated several times using data from various time frames. As indicated earlier, number of cigarettes smoked was obtained from NHEFS because only a small sub sample of NHANES I subjects were questioned about cigarette smoking. Except, of course, for mortality outcomes and causes of death, the follow-up study is a less desirable source of data due to the fact that proxies were used to gather information on deceased NHANES I participants. This complicated the estimation process by bringing into question different time frames for estimating the mortality production input demand equations. To resolve this, two different time frame estimations of the cigarette demand equation were attempted. One was tried using right hand side variables (prices, per capita sales figures, unemployment and divorce rates) with 1982 values. The other

was performed using right hand side variables from the 1972 time frame. Since religion data was only available for the year 1980, probability of being affiliated with a certain religious group was unaffected in the two time frame specifications.

In using the 1982 cigarette demand specification, an attempt was made to control for the possibility that a NHANES I subject had moved out of state and was therefore no longer affected by variables that exist in his or her former state of residence. If a subject moved out of state, he or she was simply eliminated from the sample. Roughly nine percent of the males and 10 percent of the females were eliminated in this fashion from the 1982 cigarette demand specification. This procedure was of course not necessary for the 1972 specification.

Ordinary least square estimates were obtained from the cigarette demand equations.

The dependant variable in the demand for alcoholic beverage equation (9) is based on data pertaining to NHANES I. In the NHANES I interview subjects were asked to report on the weekly number of drinks of their favorite beverage (beer, wine, spirits). Following Coate and Grossman (1989), the alcohol variable is designed to capture the non-linear relationship involving alcohol consumption and mortality due to coronary heart disease. Since moderate consumption of alcohol, not heavy consumption, is reported to have

beneficial effects on cardiovascular health, an attempt is made to construct a dependant variable that would allow for this result. One way, as suggested by Coate and Grossman, is the construction of a polychotomous variable. This variable would take on a value of 1 if the subject was a moderate drinker, 2 if the individual was a heavy drinker and 0 if the subject was an abstainer. Construction of this variable involved grouping subjects into drinking categories. Weekly alcohol consumption levels of NHANES I participants were divided by the subjects' weight to account for the fact that 20 drinks per week would effect health differently for a 200 pound subject and a 120 pound subject. The heavy drinking category was designed to capture ten percent of the males, the moderate category captures the next seventy percent of males with the remainder falling into the abstainer grouping. For females, who generally drink less than males, the representation in the heavy drinking category was proportionally smaller than the males. Only approximately three percent of the females belonged to the heavy drinking category, sixty six percent to the moderate drinking category with the rest being abstainers.

The polychotomous constriction of the alcohol variable is useful for another reason. Coate and Grossman (1989) calculated that the average weekly number of drinks of a favorite alcoholic beverage as reported by NHANES I subjects was 3.7. In 1973 U.S. alcoholic beverage sales totaled 2.6

gallons per capita (of those individual aged 15 and over). A weekly consumption level of 3.7 drinks is roughly equal to 200 drinks per year or 100 ounces of ethanol, which is less than one gallon. This may indicate that NHANES I subjects significantly under reported alcohol intake. A polychotomous variable may provide estimation of the effects of alcoholic beverage consumption on mortality that are less subject to the problems of under reporting, especially if the under reporting was done by the moderate and heavy drinking classes. It should be noted that the NHANES I interview asked individuals about consumption of their favorite drink. This could be responsible for the difference in average drinking habits of NHANES I participants and per capita sales figures. For example, it is possible that a NHANES I subject considers an expensive brandy to be their favorite beverage even though they indulge in this product infrequently and report this low consumption level, while every day they consume a six pack of beer which is not reported because it is not their favorite beverage.

Estimation of equation (9) requires a procedure which allows for the estimation of a polychotomous dependant variable. Since the dependant variable measuring alcohol consumption levels can take one of three values 0,1,2 for the categories abstainer, moderate, and heavy consumption respectively, then a procedure that will allow for the prediction that a subject falls into one of these categories

is required. Maddala (1983) shows the probability of the trichotomous variable C taking on any value i (i=0,1,2) to be equal to:

$$Pi = 1/(1+\exp(-b - XiB)) \qquad (10)$$

In this equation X represents a vector of exogenous variables, B represents parameters, and b is an intercept term. To obtain estimates of the probabilities the following logit function is fit:

$$\ln (Pi/(1-Pi)) = b + B_1 Pa + B_2 Pc + B_3 X + V \qquad (11)$$

In the above equation i can take on the value 0,1,or 2 where 0 indicates the subject is an abstainer, 1 indicates the subject is a moderate drinker and 2 indicates the subject is a heavy drinker.

The above equation, once estimated using a maximum-likelihood estimation procedure (Gauss-Newton), yields predicted values of the probability of each subject being a moderate and heavy drinker. These predicted drinking values are then used along with the predicted values of lifetime cigarette smoking in the mortality production function. This takes the form:

$$M = c_0 + c_1 C + c\ Pmod + c_2 Phev + B\ Z + W \qquad (12)$$

M is defined as mortality outcome and can take on the following values as determined by ICD72 codes. If the subject died from ICD72 code category 360, acute myocardial infarction or category 390, old myocardial infarction and other forms of chronic ischemic heart disease M = 1; if the subject died due to causes classified as ICD72 code category 440, intracerebral and other intracranial hemorrhage, or category 450, cerebral thrombosis and unspecified occlusion of cerebral arteries, or category 460, cerebral embolism, or category 470, other and late effects of cerebrovascular disease, M = 2; if the subject died from any other cause, M = 3; and if the subject survived M = 4. C, Pmod and Phev are predicted values obtained in the first stage for number of cigarettes smoked and moderate and heavy alcohol consumption variables. Z is a vector of exogenous values including age as measured in NHANES I, systolic and diastolic blood pressure readings from NHANES I, family income and a measure of exercise and physical activity are also all obtained from NHANES I.

Estimation of (12) was carried out through a maximum-likelihood procedure that took account of polychotomous outcomes that M takes on. The method of estimation is similar to that method which was used to estimate (11).

CHAPTER V

Table one lists and defines the variables used. Pmod, Phev and Yhat are variables obtained from the first stage, which are used to estimate mortality outcomes in the second stage.

Table two lists the causes of death as defined by the ICD72 causes. This table shows roughly thirty five percent of the white male deaths and approximately twenty seven percent of the white female deaths to be attributed to what this study classifies as death due to coronary heart disease (ICD72 codes 360 and 390). Stroke deaths, according to this study (ICD72 codes 440,450,460 and 470) accounted for approximately 7.4 percent of white male deaths and roughly eleven percent of white female deaths. Cancers were responsible for around twenty two and twenty six percent of white male and white female deaths respectively.

It is interesting to note that deaths due to chronic liver disorders and cirrhosis (ICD72 code 620) were responsible for approximately one percent of both white male and female deaths. Cirrhosis and liver disorders have been strongly linked to alcohol consumption. Any report showing a potential beneficial effect of alcohol consumption on health should consider the negative health effects of alcohol consumption on liver disease. This data reveals that relatively few deaths were due to cirrhosis compared to

deaths due to coronary heart disease (one percent versus thirty five percent for white males, one percent versus twenty seven percent for white females).

Table 1

Variable Names, Abbreviations, and Definitions

Age (AGEH1)	Age in years at time of NHANES I interview
Diastolic Blood Pressure (DSTH1)	Continuous blood pressure reading from NHANES I physical exam; sitting position (mmhg)
Divorce (DIVOR)	Percentage of 25-34 year old females who are divorced; by state
Education (EDUC)	Education attainment; from NHANES I
Heavy Drinker Dummy (HEAVYH1)	1=drinkers in percentiles 90 and above. See MODH1 definition
Income (INC)	Family income from NHANES
Life Smoking (LIFESMOK)	Number of cigarettes smoked in life divided by 365; from NHEFS
Liquor Dummy (LIQ)	Dummy variable indicating weekly alcohol consumption divided by weight (0=abstainer, 1=moderate, 2=heavy)
Moderate Drinker Dummy (MODH1)	From combined male and female distribution of weekly number of drinks of favorite beverage divided by body weight; 1=drinkers from 26th to 89th percentiles
PBLEND	Price of a fifth of Seagram's Seven Crown Blended Whiskey
PCALLBEV	State specific per capita consumption of ethanol; from U.S. Alcohol Epidemiologic Data

Table 1 (continued)

PCATH	Probability of being Catholic by state
PCSALES	State's per capita tax paid on cigarette sales; on fiscal year basis
PHEV	Predicted probability of being a heavy drinker; from first stage estimations
Physical Activity Dummy	1 if very active, 0 if not; (PHYSACT) from NHANES
PMOD	Predicted probability of being a moderate drinker; from first stage estimations
PMORM	Probability of being a Mormon; by state
PRICE	State average retail cigarette price per pack, inclusive of state and the average of local excise taxes
PSBAP	Probability of being a Southern Baptist; by state
SURVIVE	Indicates subject: survived=0, died from heart disease=1, died from stroke=2, died from other causes=3
Systolic Blood Pressure (SYSH1)	Continuous blood pressure reading from NHANES I physical exam; sitting position (mmhg)
YHAT	Predicted lifetime number of cigarettes smoked; from first stage estimations

Table 2
Causes of Death from NHEFS, White Males and White Females

White Males			White Females		
ICD72	Freq	Percent	ICD72	Freq	Percent
40	1	0.11	40	1	0.17
90	4	0.43	90	4	0.66
120	1	0.11	140	4	0.66
140	2	0.21	160	2	0.33
160	6	0.64	170	44	7.28
170	54	5.78	180	17	2.81
180	71	7.59	190	34	5.63
200	19	2.03	200	15	2.48
210	18	1.93	210	8	1.32
220	20	2.14	220	19	3.15
230	10	1.07	230	7	1.16
240	11	1.18	240	10	1.66
250	3	0.32	250	3	0.50
260	10	1.07	260	21	3.48
280	1	0.11	280	1	0.17
320	2	0.21	320	4	0.66
330	12	1.28	330	7	1.16
360	229	24.49	340	1	0.17
370	5	0.53	360	102	16.89
390	103	11.02	370	2	0.33
400	5	0.53	390	62	10.26
410	56	5.99	400	3	0.50
420	3	0.32	410	42	6.95
440	6	0.64	420	3	0.50
450	16	1.71	440	12	1.99
460	1	0.11	450	9	1.49
470	46	4.92	460	1	0.17
480	7	0.75	470	46	7.62
490	13	1.39	480	7	1.16
520	29	3.10	490	9	1.49
550	1	0.11	520	7	1.16
560	17	1.82	530	1	0.17
570	1	0.11	550	1	0.17
580	35	3.74	560	5	0.83
610	2	0.21	580	4	0.66
620	10	1.07	590	4	0.66
630	3	0.32	610	2	0.33
670	6	0.64	620	5	0.83
680	2	0.21	630	1	0.17
730	1	0.11	660	3	0.50
770	13	1.39	670	7	1.16
780	40	4.28	680	1	0.17
800	8	0.86	730	2	0.33
810	15	1.60	770	6	0.99
820	11	1.18	780	30	4.97

Table 2 (continued)

White Males			White Females		
ICD72	Freq	Percent	ICD72	Freq	Percent
830	6	0.64	800	12	1.99
			810	3	0.50
			820	9	1.49
			830	1	0.17

Table three contains the ICD72 code classifications. Table four lists the summary statistics for the variables used in this work. The 1982 and 1972 values refer to the two different time frame specifications used to estimate the demand for cigarette smoking.

Table five shows OLS first stage results for demand for the input cigarette smoking for white males. Panel A refers to 1982 values of the independent variables. Panel B refers to 1972 independent variables' values. Two specifications were estimated, one included all variables, the other omits unemployment and state specific per capita sales of cigarette and alcohol.

With the omission of these variables, cigarette prices are significant in the 1972 specification. In the 1982 specifications, however, neither the sign is expected nor is it significant. Income has the anticipated sign but is not significant. Education is highly significant and indicates that one more year of schooling is associated with a decrease in smoking of roughly seventeen cigarettes per year in both specifications. The religion variables are harder to decipher, but the probability of being a Catholic is statistically significant and positively associated with smoking. The cross price variable (Pblend) is positive, which conflicts with the assumption that cigarette smoking and alcohol consumption are compliments, but only in one specification is this result significant. Divorce rates have

Table 3
NCHS 9-TH REVISION 72 CAUSES OF DEATH

Code	Definition
160	Malignant neoplasms of lip, oral cavity, and pharynx (140-149)
170	Malignant neoplasms of digestive organs and peritoneum (150-159)
180	Malignant neoplasms of respiratory and intrathoracic organs (160-165)
190	Malignant neoplasm of breast (174-175)
200	Malignant neoplasms of genital organs (179-187)
210	Malignant neoplasms of urinary organs (188-189)
220	Malignant neoplasms of all other and unspecified sites (170-173,190-199)
230	Leukemia (204-208)
240	Other malignant neoplasms of lymphatic and hematopoietic tissues (200-203)
250	Benign neoplasms, carcinoma in situ, and neoplasms of uncertain behavior and of unspecified nature (210-239)
260	Diabetes mellitus (250)
270	Nutritional deficiencies (260-269)
280	Anemias (280-285)
290	Meningitis (320-322)
320	Rheumatic fever and rheumatic heart disease (390-398)
330	Hypertensive heart disease (402)
340	Hypertensive heart and renal disease (404)
360	Acute myocardial infarction (410)
370	Other acute and subacute forms of ischemic heart disease (411)

Table 3 (continued)

Code	Definition
380	Angina pectoris (413)
390	Old myocardial infarction and other forms of chronic ischemic heart disease (412,414)
400	Other diseases of endocardium (424)
410	All other forms of heart disease (415-423,425-429)
420	Hypertension with or without renal disease (401,403)
440	Intracerebral and other intracranial hemorrhage (431-432)
450	Cerebral thrombosis and unspecified occlusion of cerebral arteries (434.0,434.9)
460	Cerebral embolism (434.1)
470	All other and late effects of cerebrovascular diseases (430,433,435-438)
480	Atherosclerosis (440)
490	Other diseases of arteries, arterioles, and capillaries (441-448)
500	Acute bronchitis and bronchiolitis (466)
520	Pneumonia (480-486)
530	Influenza (487)
550	Bronchitis, chronic and unspecified (490-491)
560	Emphysema (492)
570	Asthma (493)
580	Other chronic obstructive pulmonary diseases and allied conditions (494-496)
590	Ulcer of stomach and duodenum (531-533)
600	Appendicitis (540-543)

Table 3 (continued)

Code	Definition
610	Hernia of abdominal cavity and intestinal obstruction without mention of hernia (550-553,560)
620	Chronic liver disease and cirrhosis (571)
630	Cholelithiasis and other disorders of gallbladder (574-575)
650	Acute glomerulonephritis and nephrotic syndrome (580-581)
660	Chronic glomerulonephritis, nephritis and nephropathy, not specified as acute or chronic, and renal sclerosis, unspecified (582-583,587)
670	Renal failure, disorders resulting from impaired renal function, and small kidney of unknown cause (584-586,588-589)
680	Infections of kidney (590)
690	Hyperplasia of prostate (600)
710	Pregnancy with abortive outcome (630-638)
720	Other complications of pregnancy, childbirth, and the puerperium (640-676)
730	Congenital anomalies (740-759)
750	Birth trauma, intrauterine hypoxia, birth asphyxia, and respiratory distress syndrome (767-769)
760	Other conditions originating in the perinatal period (760-766,770-779)
770	Symptoms, signs, and ill-defined conditions (780-799)
780	All other diseases (Residual)
800	Motor vehicle accidents
810	All other accidents and adverse effects
820	Suicide

Table 3 (continued)

Code	Definition
830	Homicide and legal intervention (E960-E978)
840	All other external causes (E980-E999)

Table 4

Summary Statistics
White Males

Variables	Mean	Stnd.Dev.
AGEH1	50.56	15.13
DSTH1	84.69	11.74
EDUC	11.15	3.61
HEAVYH1	0.102	0.30
INC	11420.0	7266.0
LIFESMOK	584.2	665.3
LIQ	0.902	0.54
MODH1	0.69	0.45
PCATH	0.22	0.17
PHEV	0.97	0.54
PHYSACT	0.42	0.49
PMOD	0.78	0.20
PMORM	0.006	0.013
PSBAP	0.067	0.12
SURVIVE	3.68	0.77
SYSH1	134.29	20.29
	1982 Values	
DIVOR	10.22	1.76
PBLEND	6.96	0.50
PCALLBEV	1.75	0.33
PCSALES	127.46	11.83
PRICE	74.02	4.76
YHAT	595.88	169.13
UNEMP	10.10	2.29
	1972 Values	
DIVOR	5.35	1.27
PBLEND	5.26	0.49
PCALLBEV	1.86	0.35
PCSALES	121.03	16.29
PRICE	42.72	4.15
YHAT	594.28	169.27
UNEMP	5.07	1.37

Table 4 (continued)
White Females

Variables	Mean	Stnd.Dev.
AGEH1	46.42	15.04
DSTH1	80.59	12.06
EDUC	11.45	2.93
HEAVYH1	0.03	0.17
INC	11053.0	7192.0
LIFESMOK	214.97	378.59
LIQ	0.72	0.51
MODH1	0.66	0.47
PCATH	0.22	0.17
PHEV	0.03	0.02
PHYSACT	0.42	0.49
PMOD	0.69	0.20
PMORM	0.006	0.013
PSBAP	0.06	0.12
SURVIVE	3.88	0.47
SYSH1	134.29	20.29
	1982 Values	
DIVOR	10.20	1.73
PBLEND	6.96	0.50
PCALLBEV	1.73	0.32
PCSALES	127.97	12.40
PRICE	73.81	4.91
YHAT	211.64	37.25
UNEMP	10.17	22.90
	1972 Values	
DIVOR	5.33	1.26
PBLEND	5.25	0.49
PCALLBEV	1.86	0.34
PCSALES	121.61	17.47
PRICE	42.48	4.21
YHAT	211.52	37.10
UNEMP	5.08	1.38

Table 5
White Males (1982 Values)

Variables	Paremeter (T stat)	Paremeter (T stat.)
PRICE	-5.181 (-1.642)	-1.927 (-0.521)
INC	0.002 (1.213)	0.002 (1.231)
EDUC	-16.873 (-4.17)	-18.11 (-4.623)
PCATH	217.640 (2.469)	163.01 (1.848)
PMORM	-139.5? (-0.14)	425.22 (-0.41)
PSBAP	141.24 (1.18)	96.49 (0.78)
PBLEND	59.40 (2.09)	44.43 (1.54)
DIVOR	18.34 (2.17)	20.40 (2.38)
AGEH1	8.92 (10.13)	8.88 (10.54)
UNEMP	. .	-9.22 (-1.58)
PCSALES	. .	2.29 (1.68)
PCALLBEV	. .	-33.00 (-0.85)
R-SQUARE ADJ. R-SQUARE	0.064 0.061	0.065 0.061

Table five (continued)
White Males (1972 Values)

Variables	Paremeter (T stat.)	Paremeter (T stat.)
PRICE	0.085 (0.03)	1.50 (0.38)
INC	0.002 (1.22)	0.02 (1.21)
EDUC	-18.21 (-4.67)	-18.16 (-4.64)
PCATH	141.62 (1.71)	145.15 (1.65)
PMORM	301.60 (0.30)	384.40 (0.38)
PSBAP	216.55 (2.02)	207.23 (1.75)
PBLEND	-24.73 (-0.97)	-22.47 (-0.82)
DIVOR	18.80 (1.80)	19.28 (1.61)
AGEH1	8.85 (10.51)	8.85 (10.50)
UNEMP	. .	2.34 (0.22)
PCSALES	. .	0.53 (0.55)
PCALLBEV	. .	-19.16 (-0.36)
R-SQUARE	0.063	0.063
ADJ. R SQUARE	0.060	0.060

the anticipated sign and are significant at least at the ten percent level in all specifications. Age has a positive and significant effect on cigarette smoking. Unemployment does not have the expected sign and is not significant.

Table six shows the white female results that table five reports for white males. Income, education, divorce, unemployment and per capita sales of cigarettes have the expected signs and are significant. Own price effects, however, have unexpected signs even though in both specifications they are significant. This indicates possible problems with multicollinearity or model specification. The r-square and adjusted r-square for the female version of the model of the demand for cigarettes indicates a very poor fit.

Table seven reports polychotomous logit estimates for the demand for alcohol consumption equation. Panel A represents two specifications for white males and Panel B shows results for white females.

In Panel A, the results indicate that income and education have positive signs and in at least one specification are significant. This positive association of income and education with moderate and heavy alcohol consumption is expected. Sentiment variables show expected and significant results for the probability of being Catholic and the probability of being Southern Baptist; the former being positively associated with moderate and heavy

Table 6
White Females (1982 Values)

Variables	Parameter (T stat.)	Parameter (T stat.)
PRICE	4.82 (3.15)	7.29 (3.92)
INC	0.0017 (1.96)	0.0020 (2.39)
EDUC	-5.05 (-2.23)	-6.40 (-3.01)
PCATH	124.49 (3.01)	84.95 (2.12)
PMORM	-230.22 (-0.48)	-343.56 (-0.71)
PSBAP	130.93 (2.28)	146.74 (2.65)
PBLEND	-12.13 (-0.86)	-16.55 (-1.22)
DIVOR	10.30 (2.52)	13.96 (2.43)
AGEH1	-0.36 (-0.87)	-0.34 (-0.89)
UNEMP	. .	8.43 (1.87)
PCSALES	. .	1.16 (2.81)
PCALLBEV	. .	13.10 (0.52)
R-SQUARE	0.009	0.013
ADJ. R-SQUARE	0.007	0.010

Table 6 (continued)
White Females (1972 Values)

Variables	Parameter (T stat.)	Parameter (T stat.)
PRICE	4.89 (3.45)	7.52 (4.36)
INC	.002 (2.55)	.002 (2.42)
EDUC	-6.69 (-3.15)	-6.35 (-2.99)
PCATH	108.42 (2.86)	96.20 (2.54)
PMORM	267.69 (0.57)	251.31 (0.53)
PSBAP	106.80 (2.14)	129.62 (2.55)
PBLEND	-21.54 (-1.77)	-15.39 (-1.25)
DIVOR	19.74 (3.95)	14.37 (2.78)
AGEH1	-0.37 (-0.97)	-0.34 (-0.87)
UNEMP	. .	7.83 (1.84)
PCSALES	. .	1.35 (3.51)
R-SQUARE	0.010	0.012
ADJ. R-SQUARE	0.008	0.010

Table 7 - Panel A
First Stage Alcohol Model - White Males

Variable	Parameter (Chi-square)	Parameter (Chi-square)
INTERCEPT 1	-0.098	0.753
	(0.03)	(0.67)
INTERCEPT 2	-3.978	-3.144
	(46.50)	(11.55)
PRICE	0.041	0.020
	(18.49)	(2.72)
INC	0.000018	0.000018
	(10.23)	(9.73)
EDUC	0.0474	0.0443
	(14.45)	(12.56)
PCATH	1.545	1.267
	(32.78)	(19.56)
PMORM	3.785	3.414
	(1.39)	(1.06)
PSBAP	-1.939	-1.52
	(34.63)	(17.54)
PBLEND	-0.208	-0.186
	(6.56)	(4.64)
DIVOR	0.1513	0.1058
	(19.94)	(7.54)
AGEH1	-0.0177	-0.017
	(41.81)	(42.87)
UNEMP	.	-0.083
	.	(6.45)
PCALLBEV	.	0.6839
	.	(16.35)
PCSALES	.	-0.0045
	.	(2.59)
R [1]	0.255	0.292

[1] The R statistic is similar to the multiple correlation coefficient in the normal setting, after correction is made to penalize for the number of parameters estimated.

Table 7 - Panel B
First Stage Alcohol Model - White Females

Variables	Parameter (Chi-square)	Parameter (Chi-square)
INTERCEPT 1	-1.796	-1.796
	(12.39)	(4.84)
INTERCEPT 2	-6.778	-6.805
	(166.04)	(67.88)
PRICE	0.014	0.003
	(3.00)	(0.11)
INC	0.00004	0.00004
	(68.56)	(66.19)
EDUC	0.1009	0.0950
	(63.21)	(55.29)
PCATH	2.695	2.28
	(131.24)	(85.08)
PMORM	3.996	4.411
	(1.81)	(2.09)
PSBAP	-2.637	-2.06
	(80.14)	(40.85)
PBLEND	0.0393	0.116
	(0.29)	(2.28)
DIVOR	0.1690	0.102
	(30.64)	(8.94)
AGEH1	-0.022	-0.023
	(95.26)	(99.47)
UNEMP	.	-0.1249
	.	(8.45)
PCALLBEV	.	0.934
	.	(38.43)
PCSALES	.	-0.0017
	.	(0.49)
R	0.376	0.383

alcohol consumption and the latter being negatively associated with the drinking categories. The probability of being Mormon has an unexpected sign and is not significant, possibly due to the very low probability of being a mormon found in all test sites. The own price effect (PBLEND) was found to be negative and statistically significant. Divorce rates were found to have the expected positive sign and be statistically significant. Unemployment rates and per capita alcoholic beverage consumption have expected positive signs although unemployment rates were not significant.

In Panel B, results for females follow those found for men except for own price effect which has a positive sign, although it is not significant.

From these first stage equations, the variables Yhat, Pmod, and Phev are obtained. These variables are then used in the second stage to model mortality outcomes. Tables eight A,B,C, and D show the mortality production function estimates for white males; tables nine A,B,C, and D present the equivalent results for white females. The A and B tables show the results using the inputs that were predicted in the first stage (Yhat, Pmod, and Phev). Tables labeled A use as a right hand side variable diastolic blood pressure readings (DSTH1), the B tables use systolic blood pressure. This was done to account for the collinearity between diastolic and systolic measurements. If a subject has high diastolic blood pressure it is likely that this individual also has high

systolic readings. It is worth considering the impact that individuals who control high blood pressure with medication have on mortality outcomes. Individuals taking blood pressure medication have a medical condition that almost certainly impacts mortality outcomes, but the medication may mask the blood pressure readings, thereby reducing the estimated effect of blood pressure on mortality. To account for this possibility, individuals who indicated during the NHANES I interview that they were taking blood pressure medication were eliminated from the sample.

Tables C and D show mortality outcomes using alcohol consumption variables (Modh1 and Heavyh1) from NHANES I and lifetime smoking variables (Lifesmok) from NHEFS.

In tables eight and nine both the "log odds ratio" and their calculated marginal effects are reported. These marginal effects are interpreted as the effect on a mortality outcome of a one unit increment in the right hand side variable relative to the omitted outcome. That is, in table seven A, an additional year of age increases the probability of death due to coronary heart disease, stroke and all other causes by .002, .0018 and .0058 relative to the probability of staying alive through the roughly ten year interval between NHANES I and NHEFS. Therefore, the probability of dying from any cause for an additional year of age is the sum of the probabilities from each cause (.002 + .0018 + .0058). The probability of dying from any cause

for two additional years of life would simply be two times the sum of one additional year of age.

Reported in tables eight and nine A and B are the estimates obtained using the predicted values from the more inclusive specifications of the first stage equations (B).

The results reported in table eight A show that age, income, diastolic blood pressure, physical activity, and predicted lifetime smoking have anticipated signs, although diastolic blood pressure and income are not significant. Predicted moderate drinking, although significant, does not have a negative impact on mortality due to coronary heart disease while predicted heavy drinking does seem to be associated with lower mortality due to coronary heart disease. This finding is not expected since most previous studies identify moderate drinking and not heavy consumption to be associated with lower mortality.

For mortality due to stroke, predicted probability of moderate drinking has a negative impact, and predicted heavy drinking has a positive impact, though neither is significant.

For mortality due to all other causes the findings are unanticipated and not significant.

Table eight B shows the mortality outcome results replacing diastolic blood pressure measurements with systolic blood pressure readings. This replacement does not appreciably change alcohol consumption's impact on mortality

outcomes.

Table 8 - A
Multinomial Logit Mortality Model
Using Predicted Inputs
For White Males

Number of Cases For Each Outcome;

1 = Coronary Heart Disease Deaths 179
2 = Stroke Deaths 36
3 = Other Cause Deaths 335
4 = Survive 2627

OUTCOME	VARIABLE	PARAMETER	T STAT.	MARGINAL
1	CONSTANT	-8.695	-7.84	-0.433
1	AGEH1	0.430 D-1	3.06	0.214 D-2
1	DSTH1	0.601 D-2	0.91	0.299 D-3
1	YHAT	0.376 D-2	3.21	0.187 D-3
1	PMOD	2.533	1.90	0.12
1	PHE	-12.888	-2.33	-0.642
1	PHYSACT	-0.513	-2.97	-0.256 D-1
1	INC	-0.151 D-4	-1.00	-0.756 D-6
2	CONSTANT	-15.737	-5.59	-0.169
2	AGEH1	0.165	4.34	0.178 D-2
2	DSTH1	0.333 D-1	2.61	0.359 D-3
2	YHAT	0.984 D-4	0.03	0.106 D-5
2	PMOD	-2.23	-0.84	-0.241 D-1
2	PHEV	11.77	0.92	0.127
2	PHYSACT	-0.390	-1.03	-0.420 D-2
2	INC	-0.740 D-4	-1.83	-0.798 D-6
3	CONSTANT	-6.515	-7.55	-0.541
3	AGEH1	0.703 D-1	6.36	0.584 D-2
3	DSTH1	-0.685	-1.31	-0.569 D-3
3	YHAT	0.166 D-2	1.78	0.138 D-3
3	PMOD	1.118	1.11	0.928 D-1
3	PHEV	-4.893	-1.25	-0.406
3	PHYSACT	-0.520	-3.89	-0.432 D-1
3	INC	-0.307 D-4	-2.57	-0.255 D-5

Chi-squared = 716.64

Table 8 - B
Multinomial Logit Mortality Model
Using Predicted Inputs
For White Males

Number of Cases For Each Outcome:

1 = Coronary Heart Disease Deaths 179
2 = Stroke Deaths 36
3 = Other Cause Deaths 335
4 = Survive 2627

OUTCOME	VARIABLE	PARAMETER	T STAT.	MARGINAL
1	CONSTANT	-8.928	-8.66	-0.444
1	AGEH1	0.398 D-1	2.83	0.198 D-2
1	SYSH1	0.727 D-2	1.95	0.362 D-3
1	YHAT	0.364 D-2	3.11	0.181 D-3
1	PMOD	2.576	1.93	0.128
1	PHEV	-13.267	-2.40	-0.661
1	PHYSACT	-0.523	-3.03	-0.260 D-1
1	INC	-0.145 D-4	-0.96	-0.723 D-6
2	CONSTANT	-15.578	-5.78	-0.166
2	AGEH1	0.147	3.84	0.157 D-2
2	SYSH1	0.270 D-1	4.04	0.289 D-3
2	YHAT	-0.195 D-3	-0.79	-0.208 D-5
2	PMOD	-1.712	-0.64	-0.182 D-1
2	PHEV	8.308	0.65	0.887 D-1
2	PHYSACT	-0.472	-1.24	-0.504 D-2
2	INC	-0.686 D-4	-1.69	-0.734 D-6
3	CONSTANT	-7.366	-9.14	-0.613
3	AGEH1	0.707 D-1	6.40	0.589 D-2
3	SYSH1	0.267 D-2	0.90	0.223 D-3
3	YHAT	0.152 D-2	1.73	0.126 D-3
3	PMOD	1.068	1.06	0.890 D-1
3	PHEV	-4.726	-1.21	-0.393
3	PHYSACT	-0.519	-3.89	-0.432 D-1
3	INC	-0.313 D-4	-2.62	-0.260 D-5

Chi-squared = 719

Table 8 - C
Multinomial Logit Mortality Model
Using NHANES I and NHEFS Inputs
For White Males

Number of Cases For Each Outcome;

1 = Coronary Heart Disease Deaths	179
2 = Stroke Deaths	36
3 = Other Cause Deaths	335
4 = Survive	2627

OUTCOME	VARIABLE	PARAMETER	T STAT.	MARGINAL
1	CONSTANT	-8.859	-9.19	-0.437
1	AGEH1	0.101	11.79	0.502 D-2
1	DSTH1	0.647 D-2	1.01	0.319 D-3
1	LIFESMOK	0.485 D-3	0.18	0.139 D-5
1	MODH1	-0.325	-1.86	-0.160 D-1
1	HEAVYH1	0.250	0.91	0.123 D-1
1	PHYSACT	-0.389	-2.25	-0.192 D-1
1	INC	-0.964 D-5	-0.73	-0.476 D-6
2	CONSTANT	-16.542	-6.16	-0.163
2	AGEH1	0.177	5.78	0.175 D-2
2	DSTH1	0.301 D-1	2.37	0.297 D-3
2	LIFESMOK	0.369 D-3	0.96	0.223 D-5
2	MODH1	-0.161	-0.43	-0.160 D-2
2	HEAVYH1	-0.397	-0.50	-0.393 D-2
2	PHYSACT	-0.916	-2.11	-0.906 D-2
2	INC	-0.484 D-4	-1.40	-0.479 D-6
3	CONSTANT	-6.989	-9.25	-0.547
3	AGEH1	0.103	15.51	0.809
3	DSTH1	-0.341 D-2	-0.65	-0.267 D-3
3	LIFESMOK	0.289 D-3	1.89	0.111 D-4
3	MODH1	-0.343	-2.45	-0.269 D-1
3	HEAVYH1	0.291	1.35	0.228 D-1
3	PHYSACT	-0.465	-3.39	-0.364 D-1
3	INC	-0.261 D-4	-2.41	-0.204 D-5

Chi-squared = 730.26

Table 8 - D
Multinomial Logit Mortality Model
Using NHANES I and NHEFS Inputs
For White Males

Number of Cases For Each Outcome;

1 = Coronary Heart Disease Deaths	179	
2 = Stroke Deaths	36	
3 = Other Cause Deaths	335	
4 = Survive	2627	

OUTCOME	VARIABLE	PARAMETER		T STAT.	MARGINAL	
1	CONSTANT	-9.038		-10.11	-0.446	
1	AGEH1	0.970	D-1	11.05	0.479	D-2
1	SYSH1	0.805	D-2	2.26	0.397	D-3
1	LIFESMOK	0.102	D-3	0.33	0.139	D-5
1	MODH1	-0.321		-1.84	-0.158	D-1
1	HEAVYH1	0.217		0.78	0.107	D-1
1	PHYSACT	-0.395		-2.28	-0.195	D-1
1	INC	-0.950	D-5	-0.71	-0.469	D-6
2	CONSTANT	-16.388		-6.29	-0.161	
2	AGEH1	0.161		5.15	0.158	D-2
2	SYSH1	0.255	D-1	3.77	0.251	D-3
2	LIFESMOK	0.353	D-3	1.00	0.235	D-5
2	MODH1	-0.125		-0.33	-0.123	D-2
2	HEAVYH1	-0.481		-0.60	-0.473	D-2
2	PHYSACT	-0.948		-2.16	-0.932	D-2
2	INC	-0.500	D-4	-1.43	-0.492	D-6
3	CONSTANT	-7.589		-10.77	-0.595	
3	AGEH1	0.101		14.84	0.792	D-2
3	SYSH1	0.452	D-1	1.55	0.354	D-3
3	LIFESMOK	0.295	D-3	1.93	0.114	D-4
3	MODH1	-0.353		-2.53	-0.277	D-1
3	HEAVYH1	0.244		1.13	0.192	D-1
3	PHYSACT	-0.468		-3.41	-0.367	D-1
3	INC	-0.262	D-4	-2.43	-0.205	D-5

Chi-squared = 718.12

Table 9 - A
Multinomial Logit Mortality Model
Using Predicted Inputs
For White Females

Number of Cases For Each Outcome;

1 = Coronary Heart Disease Deaths	72
2 = Stroke Deaths	33
3 = Other Cause Deaths	214
4 = Survive	4078

OUTCOME	VARIABLE	PARAMETER		T STAT.	MARGINAL	
1	CONSTANT	-12.089		-8.68	-0.184	
1	AGEH1	0.122		7.15	0.186	D-2
1	DSTH1	0.114	D-1	2.51	0.169	D-3
1	YHAT	0.634	D-2	1.90	0.967	D-4
1	PMOD	0.104		0.10	0.159	D-2
1	PHEV	-36.818		-1.45	-0.561	
1	PHYSACT	-0.420		-1.88	-0.826	D-2
1	INC	0.267	D-4	1.11	0.407	D-6
2	CONSTANT	-11.423		-5.12	-0.824	D-1
2	AGEH1	0.130		4.60	0.941	D-3
2	DSTH1	0.163	D-1	2.63	0.121	D-3
2	YHAT	-0.118	D-2	-0.24	-0.855	D-5
2	PMOD	-0.701		-0.53	-0.505	D-2
2	PHEV	16.735		0.58	0.121	
2	PHYSACT	-0.643		-1.47	-0.464	D-2
2	INC	-0.833	D-4	-1.64	-0.601	D-6
3	CONSTANT	-8.332		-12.05	-0.359	
3	AGEH1	0.872	D-1	11.75	0.376	D-2
3	DSTH1	0.179	D-2	0.60	-0.790	D-4
3	YHAT	0.238	D-2	1.15	0.103	D-3
3	PMOD	0.759		1.38	0.327	D-1
3	PHEV	-2.390		-0.31	-0.103	
3	PHYSACT	-0.303		-1.91	-0.130	D-1
3	INC	-0.159	D-4	-1.09	-0.687	D-6

Chi-squared = 540.74

Table 9 - B
Multinomial Logit Mortality Model
Using Predicted Inputs
For White Females

Number of Cases For Each Outcome:

1 = Coronary Heart Disease Deaths	72
2 = Stroke Deaths	33
3 = Other Cause Deaths	214
4 = Survive	4078

OUTCOME	VARIABLE	PARAMETER	T STAT.	MARGINAL
1	CONSTANT	-13.516	-9.08	-0.205
1	AGEH1	0.115	6.61	0.175 D-2
1	SYSH1	0.126 D-1	2.69	0.191 D-3
1	YHAT	0.663 D-2	1.99	0.100 D-3
1	PMOD	0.145	0.13	0.221 D-2
1	PHEV	-37.005	-1.45	-0.561
1	PHYSACT	-0.537	-1.86	-0.816 D-2
1	INC	0.272 D-4	1.13	0.413 D-6
2	CONSTANT	-13.235	-5.57	-0.953 D-1
2	AGEH1	0.124	4.33	0.900 D-3
2	SYSH1	0.151 D-1	2.32	0.109 D-3
2	YHAT	-0.837 D-3	-0.17	-0.603 D-5
2	PMOD	-0.668	-0.50	-0.481 D-2
2	PHEV	16.982	0.58	0.122
2	PHYSACT	-0.646	-1.40	-0.465 D-2
2	INC	-0.840 D-4	-1.60	-0.605 D-6
3	CONSTANT	-8.349	-11.00	-0.360
3	AGEH1	0.871 D-1	11.29	0.376 D-2
3	SYSH1	0.160 D-3	0.05	0.691 D-5
3	YHAT	0.238 D-2	1.14	0.103 D-3
3	PMOD	0.758	1.38	0.327 D-1
3	PHEV	-2.398	-0.31	-0.103
3	PHYSACT	-0.302	-1.90	-0.130 D-1
3	INC	-0.158 D-4	-1.09	-0.684 D-6

Chi-squared = 546.88

Table 9 - C
Multinomial Logit Mortality Model
Using NHANES I and NHEFS Inputs
For White Females

Number of Cases For Each Outcome;

1 = Coronary Heart Disease Deaths	72
2 = Stroke Deaths	33
3 = Other Cause Deaths	214
4 = Survive	4078

OUTCOME	VARIABLE	PARAMETER	T STAT.	MARGINAL
1	CONSTANT	-13.093	-11.25	-0.201
1	AGEH1	0.101	8.94	0.201 D-2
1	DSTH1	-0.152 D-1	-1.39	-0.207 D-3
1	LIFESMOK	0.585 D-4	0.68	0.793 D-6
1	MODH1	-0.139	-0.60	-0.207 D-2
1	HEAVYH1	-0.316	-0.41	-0.470 D-2
1	PHYSACT	-0.325	-2.25	-0.162 D-1
1	INC	-0.846 D-4	-1.88	-0.416 D-6
2	CONSTANT	-13.040	-8.17	-0.134
2	AGEH1	0.132	6.44	0.137 D-2
2	DSTH1	0.165	2.47	0.120 D-3
2	LIFESMOK	0.348 D-3	0.65	0.220 D-5
2	MODH1	-0.490	-1.44	-0.336 D-2
2	HEAVYH1	-0.478	-0.41	-0.337 D-2
2	PHYSACT	-0.883	-2.11	-0.846 D-2
2	INC	-0.736 D-4	-1.58	-0.589 D-6
3	CONSTANT	-8.027	-19.82	-0.337
3	AGEH1	0.915 D-1	15.08	0.385 D-2
3	DSTH1	0.236 D-2	0.37	0.842 D-4
3	LIFESMOK	0.289 D-3	1.59	0.111 D-4
3	MODH1	-0.189	-1.27	-0.796 D-2
3	HEAVYH1	-0.218 D-2	-0.48	-0.918 D-4
3	PHYSACT	-0.365	-1.98	-0.134 D-1
3	INC	-0.186 D-4	-0.97	-0.684 D-6

Chi-squared = 541.04

Table 9 - D
Multinomial Logit Mortality Model
Using NHANES I and NHEFS Inputs
For White Females

Number of Cases For Each Outcome;

1 = Coronary Heart Disease Deaths	72
2 = Stroke Deaths	33
3 = Other Cause Deaths	214
4 = Survive	4078

OUTCOME	VARIABLE	PARAMETER	T STAT.	MARGINAL
1	CONSTANT	-13.971	-11.69	-0.207
1	AGEH1	0.163	19048	0.243 D-2
1	SYSH1	0.100 D-1	1.51	0.135 D-3
1	LIFESMOK	0.112 D-3	0.87	0.134 D-5
1	MODH1	-0.121	-0.52	-0.179 D-2
1	HEAVYH1	-0.336	-0.41	0.500 D-2
1	PHYSACT	-0.492	-1.79	-0.812 D-2
1	INC	0.199 D-4	-1.01	-0.425 D-6
2	CONSTANT	-12.617	-8.57	-0.865 D-1
2	AGEH1	0.131	6.08	0.901 D-3
2	SYSH1	0.169 D-1	1.55	0.108 D-3
2	LIFESMOK	0.369 D-3	1.98	0.234 D-5
2	MODH1	-0.433	-1.20	-0.297 D-2
2	HEAVYH1	-0.445	-0.40	-0.305 D-2
2	PHYSACT	-0.595	-1.50	-0.462 D-2
2	INC	-0.790 D-4	-1.31	-0.605 D-6
3	CONSTANT	-8.024	-19.67	-0.337
3	AGEH1	0.918 D-1	14.93	0.386 D-2
3	SYSH1	0.558 D-2	1.31	0.215 D-3
3	LIFESMOK	0.295 D-3	1.52	0.114 D-4
3	MODH1	-0.161	-1.21	-0.804 D-2
3	HEAVYH1	0.331	0.85	0.139 D-3
3	PHYSACT	-0.299	-1.68	-0.121 D-1
3	INC	-0.149 D-4	-1.02	-0.683 D-6

Chi-squared = 538.42

Predicted cigarette smoking's impact on mortality due to coronary heart disease has the expected sign and is significant, which is not the case when the smoking variable from NHEFS is used (table eight C and D). Predicted smoking does not have a significant effect on stroke deaths but is significant at the ten percent level on deaths due to all other causes. The NHEFS smoking variable is significant and has the anticipated sign in the deaths due to other causes outcome; this parameter is not significant for stroke deaths.

The results for white females reported in tables nine A,B,C, and D generally follow the results for white males. Age, diastolic and systolic blood pressure, predicted smoking and physical activity all impact mortality due to coronary heart disease in the same way as they do with white males. Income for white females however, is positively associated with mortality due to coronary heart disease. This is not the case for white males. Income is not significant for either sex. Predicted drinking variables also have the unexpected signs and are not significant.

For stroke mortality outcomes, white females show a non significant effect from predicted smoking. This lack of significance also applies to predicted alcohol consumption's effect on stroke deaths and deaths due to other causes. Predicted smoking is not significant on mortality due to other causes for white females, although it has the

anticipated sign. The impact of this variable on white males is positive and significant at the ten percent level for mortality due to other causes. Physical activity is negatively related to all mortality outcomes and is significant for all but stroke outcomes for white females, which is the same result obtained for white males. Systolic and diastolic blood pressure readings have positive and significant impacts on mortality due to coronary heart disease and mortality due to stroke for white females. White male results agree with this, but diastolic blood pressure is not significant on the outcome of mortality due to coronary heart disease. Neither male nor female diastolic or systolic blood pressure was found to be significant for outcomes due to other causes.

Comparing the male and female models using the predicted values of alcohol consumption and cigarette smoking (tables eight and nine, A and B) with the models which use these values obtained from NHANES I and NHEFS (tables eight and nine, C and D) yields interesting conclusions. Most variables tend to agree. The drinking variables however, do not seem to correspond. In the models for males, using the inputs obtained from the first stage, moderate consumption of alcohol is positively related to mortality due to coronary heart disease, and heavy drinking is negatively related to this mortality outcome; both results are significant. The models using the values

obtained from the NHANES I and the follow-up show results that are opposite of the results from the model using the predicted inputs. However, the heavy drinking parameter from the model using the data is not significant.

For stroke deaths, estimates using either source (first stage or NHEFS) are not significant. Deaths due to other causes, using the predicted values, yields results that are not significant. The female mortality outcome models yield results that are not significant for the drinking variables.

Predicted cigarette smoking provides more conclusive results than the cigarette smoking variables obtained from NHEFS. For the outcome of mortality due to coronary heart disease, predicted cigarette smoking variables have the anticipated sign and are significant for both white males and white females. The cigarette smoking variables obtained directly from NHEFS yield insignificant results. Cigarette smoking's impact on stroke deaths is insignificant regardless of whether the cigarette smoking variable comes from the first stage or NHEFS. For the outcome of death due to other causes for white males, the predicted smoking variable yields estimates that are significant at the ten percent level and have the expected sign. The smoking variable obtained from NHEFS yields results that are not significant. For females, predicted cigarette smoking has the expected impact on death due to coronary heart disease and is significant; it is not significant on stroke deaths

or mortality due to other causes. The smoking variable obtained from NHEFS does not yield significant results for any of the mortality outcomes.

SELECTED BIBLIOGRAPHY

1. Becker, G.S. "A Theory of the Allocation of Time." Economic Journal, 75, No. 299 (September 1965), p. 493-517

2. Becker, G.S. "Don't Raise the Drinking Age, Raise Taxes." Business Week, November 25, 1985, p. 76

3. Bureau of Labor Statistics, U.S. Department of Labor. Geographical Profile of Employment and Unemployment. Washington, D.C.: U.S. Government Printing Office, various years.

4. Coate, D. and Grossman, M. "Effects of Alcoholic Beverage Prices and Legal Drinking Ages on Youth Alcohol Use." Journal of Law and Economics, 31, p. 145 - 170, 1988.

5. Cook, P.J., and Tauchen, G. "The Effect of Liquor Taxes on Heavy Drinking." Bell Journal of Economics, 13, No. 2 (Autumn 1982), p. 379-390

6. Cook, P.J. "The Effect of Liquor Taxes on Drinking, Cirrhosis, and Auto Fatalities." Alcohol and Public Policy: Beyond the Shadow of Prohibition, edited by M.H. Moore and D.R. Gerstein. Washington, D.C.: National Academy Press, 1981.

7. Distilled Spirits Council of the United States. Annual Statistical Review. Washington, D.C.: Distilled Spirits Council of the United States, various years.

8. Distilled Spirits Council of the United States. "Price per Fifth for Selected Types and Brands of Liquor in Control and License States." Various years.

9. Edwards, L.N., and Grossman, M. "The Relationship between Children's Health and Intellectual Development." Health: What is it Worth, edited by S.J. Mushkin and D.W. Dunlop. Elmsford, New York: Pergamon Press, 1979.

10. Farrell, P. and Fuchs, V.R. "Schooling and Health: The Cigarette Connection." Journal of Health Economics, 1, No. 3 (December 1982), p. 205-230

11. Fuchs, V.R. "Time Preference and Health: An Exploratory Study." Economic Aspects of Health, edited by V.R. Fuchs. Chicago: University of Chicago Press for the National Bureau of Economic Research, 1982.

12. Gordon, T. and Kannel, W. "Drinking and Its Relation to Smoking, BP, Blood Lipids, and Uric Acids: The Framingham Study." Archives of Internal Medicine, 143, p. 1366 - 1373, 1983.

13. Gordon, T. and Kannel, W. "Drinking Habits and Cardiovascular Disease: The Framingham Study." American Heart Journal, p. 667 - 673, 1983.

14. Grossman, M. and Benham, L. "Health, Hours, and Wages." The Economics of Health and Medical Care, edited by M. Perlman. Proceedings of a conference sponsored by the International Economics Association. London: Macmillan, 1974.

15. Grossman, M. "The Correlation between Health and Schooling." Household Production and Consumption, edited by N.E. Terleckyj. Studies in Income and Wealth, Volume 40, by the Conference on Research in Income and Wealth. New York: Columbia University Press for the National Bureau of Economic Research, 1975.

16. Grossman, M. The Demand for Health: A Theoretical and Empirical Investigation. New York: Columbia University Press for the National Bureau of Economic Research, 1972.

17. Jacobson, M. and Albion, M. "Raising Alcohol Taxes is the Way to Cut Drinking and the Debt." Washington Post, August 11, 1985.

18. Johnson, D.W.; Picard, P.R.; and Quinn, B. Churches and Church Membership in the United States, 1971. Washington, D.C.: Glenmary Research Center, 1974.

19. Kennedy, P. A Guide to Econometrics. Cambridge: The MIT Press, 1979.

20. Lancaster, K.J. "A New Approach to Consumer Theory." Journal of Political Economy, 75, No.2 (April 1966), p. 132-157.

21. Maddala, G.S. Limited Dependent and Qualitative Variables in Econometrics. Econometric Society Monographs No. 3. New York: Cambridge University Press, Inc., 1983.

22. McFadden, D. "Conditional Logit Analysis of Qualitative Choice Behavior." In Frontiers of Econometrics, edited by P. Zarembka. New York: Academic Press, 1973.

23. Midanik, L. "The Validity of Self-Reported Alcohol Consumption and Alcohol Problems: A Literature Review." British Journal of Addiction, 77, 1982, p. 123-142.

24. Mincer, J. Schooling, Experience, and Earnings. New York: Columbia University Press for the National Bureau of Economic Research, 1974.

25. Moore, M.H., and Gerstein, D.R., editors. Alcohol and Public Policy: Beyond the Shadow of Prohibition. Washington, D.C.: National Academy Press, 1981.

26. Moore, R.D., and Pearson, T.A. "Moderate Alcohol Consumption and Coronary Artery Disease." Medicine, 65, p. 242 - 267, 1986.

27. National Center for Health Statistics. Plan and Operation of the Second Health and Nutrition Examination Survey, 1976-1980. U.S. Department of Health and Human Services, Vital and Health Statistics, DHHS Publication No. (PHS) 81-1317, Series 1, No. 15, 1981.

28. Ornstein, S.I. "Control of Alcohol Consumption Through Price Increases." Journal of Studies on Alcohol, 41, No. 9, 1980, p. 807-881.

29. Polich, J.M. "The Validity of Self-Reports in Alcoholism Research." Addictive Behaviors, 7, 1982, p. 809-822.

30. Quinn, B., et al. Churches and Church Membership in the United States, 1980. Atlanta, Georgia: Glenmary Research Center, 1982.

31. Rosen, S. and Taubman, P. "Some Socioeconomic Determinants of Mortality." Economics of Health Care, edited by J. van der Gaag, W.B. Neenan, and T. Tsukahara, Jr. New York: Praeger Publishers, 1982.

32. Rosenzweig, M.R., and Schultz, T.P. "Consumer Demand and Household Production: The Relationship between Fertility and Child Mortality." American Economic Review, 73, p. 38 - 42 (May 1983), p. 38-42.

33. Rosenzweig, M.R., and Schultz, T.P. "Estimating a Household Production Function: Heterogeneity, the Demand for Health Inputs, and their Effects on Birth Weight." Journal of Political Economy, 91, No. 5 (October 1983), p. 723-746.

34. Safer, H. and Grossman, M. "Drinking Age Laws and Highway Mortality Rates: Cause and Effect." Economic Inquiry, 25, No. 3, p. 403 - 418 (July 1987).

35. Shakotko, R.A.; Edwards, L.N.; and Grossman, M. "An Exploration of the Dynamic Relationship between Health and Cognitive Development in Adolescence." Contributions to Economic Analysis: Health, Economics, and Health Economics, edited by J. van der Gaag and M. Perlman. Amsterdam: North-Holland Publishing Company, 1981.

36. Taubman, P. and Rosen, S. "Health, Education, and Marital Status." Economic Aspects of Health, edited by V.R. Fuchs. Chicago: University of Chicago Press for the National Bureau of Economic Research, 1982.

37. Tobacco Tax Council. Municipal Tax Surveys. Richmond, Virginia: Tobacco Tax Council, various years.

38. Tobacco Tax Council. The Tax Burden on Tobacco: Historical Compilation, Volume 20. Richmond, Virginia: Tobacco Tax Council, 1986.

39. Wagstaff, A. "The Demand for Health: Some New Empirical Evidence." Journal of Health Economics, 5, No.3 (September 1986), p. 223-240.