Do the wealthy have a health advantage? An investigation of wealth as a measure of socioeconomic status

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Abstract

Anjum Hajat: Do the wealthy have a health advantage? An investigation of wealth as a measure of socioeconomic status
(Under the direction of Jay S. Kaufman)

Background: In the health literature, wealth is often overlooked as a measure of socioeconomic status.

Objectives: We explored the association between wealth and various health outcomes, namely: hypertension, obesity, smoking, self-reported general health status (GHS) and mortality.

Methods: We used data from the Panel Survey of Income Dynamics (PSID), a longitudinal study of a nationally representative US population. PSID data from 1984 to 2005 were used to evaluate the association between wealth and mortality and GHS and data from 1999 to 2005 to explore the relationship between wealth and hypertension, smoking and obesity. Inverse probability weights were employed to handle time-varying confounding and to estimate both relative and absolute measures of effect. Wealth was defined as inflation adjusted net worth and specified as a 6 category variable: a category for those with less than or equal to 0 wealth and 5 quintiles of positive wealth.

Results: In the fully adjusted model, the risk of becoming obese was inversely related to wealth; there was a 40% to 89% higher risk of becoming obese and 11 to 25 excess cases of obesity (per 1000 persons) among the less wealthy groups relative to the wealthiest quintile. Smoking initiation had a similar but more moderate effect, while hypertension incidence had a weak association with wealth, showing fewer excess cases (between 4 and 9) among the

less wealthy groups. There was a 17% to 54% higher risk of falling into poor health and 6 to 22 excess cases of poor health (per 1000 persons) among the 4 less wealthy groups relative to the wealthiest quintile. The overall wealth-mortality association revealed between a 25% and 83% increased risk and between 2 and 5 excess cases of death (per 1000) among the less wealthy compared to the wealthiest.

Conclusion: There is a strong inverse association between wealth and incidence of obesity, poor health status and mortality, a moderate inverse association between wealth and smoking initiation and a weak inverse association between wealth and hypertension incidence. Wealth is a useful measure of SES and should be considered by future health researchers.

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List of Abbreviations

SES: socioeconomic status

PSID: Panel Survey on Income Dynamics

BMI: body mass index

GHS: general health status

CVD: cardiovascular disease

NDI: National Death Index

DAG: directed acyclic graph

MSM: marginal structural model

IPW: inverse probability weight

CI: confidence interval

IDA: individual development account

Chapter 1

Introduction to wealth

The connection between health and socioeconomic status (SES) is well established. From as early as the 17th century scientists have observed that the poor are more susceptible to death and disease than the rich. In modern epidemiologic practice, many health researchers give little thought to which measure of SES would be best for their population or study question. SES is usually measured with income, education or occupation; while the use of wealth as a measure of SES is relatively rare. There are, in fact, several reasons why wealth may be a better measure of SES and should be considered as a measure of SES for future health studies. This dissertation investigates the association between wealth and various health outcomes.

Definitions & theories

Wealth is defined as a household's stock or storehouse of resources (Keister 2000). Income, on the other hand, is defined as the flow of resources into the household and consumption as the flow of resources out of the household. Savings is the difference between income and consumption and is transformed into wealth (through the purchase of cars, houses, stocks and bonds or the reduction of debt). Thus by definition income and wealth are related but separate constructs.

There are 3 major theories about household accumulation of wealth. The first, known as the consumption model, posits that the purpose of wealth is for future consumption (1).

This model, developed by economists, has dominated the literature on wealth and to a large extent has shaped the definition and measurement of wealth (1). The consumption model indicates several different roles that wealth can plan. First, wealth can function as a store of value, which allows households to smooth consumption over time. In periods where income is high, families can save thus accumulating wealth and during times when income is insufficient families can use wealth to sustain consumption. In addition, wealth is a form of insurance, also known as "precautionary savings", which may be needed for emergencies or unexpected costs. And lastly, wealth can generate income through capital. Using wealth to purchase machines or skills will eventually increase income and generate future wealth.

A second theory related to wealth is social stratification. Social stratification theorists believe that assets are used mainly in perpetuating social and economic inequalities from generation to generation. Inheritance and bequests play a major role in maintaining social class, thus are critical to this perspective (2). Lastly, the assets-for-development perspective maintains that assets can be used for capacity building, thus they are a tool for socioeconomic development (1).

Several individual and household level factors impact wealth accumulation as well. The household's position in the life course dictates the purpose of their savings. For example, older individuals may be saving for retirement, while young families may be saving for their children's college education. In addition, social class dictates a household's perspective on savings. Comparing the wealthy to the middle-class and poor, several differences relating to wealth accumulation become immediately apparent. First, the middle-class have a different motivation for saving money compared to the wealthy. They save primarily for retirement, emergencies and large consumer purchases, while the wealthy save

primarily for future investment. Unfortunately, the poor make little progress in saving any money at all. These savings motivations help the wealthy continue to accumulate wealth while doing little for the middle-class and much less for the poor.

Advantages of wealth

There are several advantages to using wealth as a measure of SES. First, since it is less subject to fluctuations over one's lifetime, wealth is often a more stable measure of SES than income. The stability of wealth comes from the fact that it is often inherited over the generations thus reflecting a historical accumulation of assets. However, it should be kept in mind that at certain times of the life course wealth is meant to be drawn upon or depleted (during sickness, emergencies or retirement). During these times, wealth is serving one of its chief purposes to act as a store of resources thus ensuring minimal change in consumption during difficult times.

In addition, wealth may be a better measure of social hierarchy compared to other SES measures (3). Wealth buys economic security, political power, social prestige and educational or occupational opportunities in a way that income alone may not allow (4, 5). That is, wealth encompasses economic circumstances as well as prestige and status. In Thomas Shaprio's book The Hidden Cost of Being African American he posits that wealth is used by many white families to secure their social position in society (2). For example, assistance with down-payments on homes, college tuition and college savings funds for future generations can help struggling middle-class families maintain the position their parents held. Social stratification researchers also pay special attention to home-ownership as an important component of a household's assets. Neighborhood residence affords families access to high quality schools and efficient social services, thus furthering the use of assets to

maintain social position (1). If Shapiro's theory is true, the use of wealth as a measure of SES may provide a more nuanced measure compared to others.

Finally, wealth may be a better measure of one's economic situation at various points in the life course. For example, during times of unemployment or illness when income is lost, wealth may help maintain living standards. During these times, measures of employment status or income may misrepresent an individual's economic situation. Some studies have shown that SES measures are not interchangeable; instead they may work through different mechanisms at different times in the life course (6, 7). For example, wealth may be an especially good SES measure for older populations, when income is limited or absent (5, 8). In fact, several studies have shown a strong association between wealth and health in the elderly population (9-11). Thus some have posited that wealth works independently of income to impact health (8).

Issues in wealth measurement

Wealth is not often used in health studies because it is difficult to measure effectively. A thorough assessment of wealth would require asking several sensitive questions about the value of personal property, debt and financial instruments. Since wealth data are self-reported and difficult to validate, the possibility of poor measurement exists. Interestingly, the underestimation of assets by the wealthiest Americans has limited our ability to understand the true disparity in wealth that exists in the US (12). In the health literature, measures of wealth are not consistent and are sometimes simplistic; thus making comparisons across studies difficult and masking potential associations between wealth and health outcomes (8).

For example, several health studies use questions on home ownership (yes/no) as a proxy for wealth. Other studies rely on a single question to measure wealth, asking respondents to estimate their total assets minus any liabilities. Research has shown that using fewer items results in significant underestimation of wealth (8). Instead, using multiple more detailed questions about specific assets is likely to produce the most accurate picture of wealth. Clearly, many health studies do not have the luxury of asking many detailed questions on wealth, a variable most likely used as an SES adjustment. Fortunately, work is underway to develop a set of questions that can be used to ascertain wealth specifically for health studies (13).

Even in studies that measure wealth well, there is a lack of consistency in the definition of wealth. One common point of debate is whether to include or exclude the value of one's home. This reflects the distinction between net financial assets and net worth. Assets refer to the immediate liquid resources available in times of need, thus this measure excludes home equity. Net worth, on the other hand, refers to resources available to the next generation and for future consumption, thus including home equity (2). The appropriate measure depends on the study question at hand. In addition, some researchers include future pension and social security assets in the calculation of family wealth, while others prefer to count this as income only when it is actually received (14). More broadly, the issue of future pension income changes a household's expectations about retirement. If families can count on a consistent pension or social security income they will not need to draw on wealth stores as much as families who do not have such a luxury. This difference in pension incomes between households implies the need to somehow account for this household characteristic.

One component of wealth that remains unmeasured is education. Although it does not take the same form as the other forms of wealth, education is pivotal in generating income. The contribution of education to younger individuals is likely greater than its contribution to older individuals, thus the young tend to have their wealth systematically underestimated. Controlling for age and education in all models dealing with wealth is thus essential.

An additional wealth measure, asset poverty, deserves a brief mention. Researchers and policy advocates generally focus on income poverty as a measure of deprivation, with much less attention paid to the concept of asset poverty. Asset poverty can be defined in many ways. One common approach is to assess whether or not families have sufficient funds to maintain their households for up to 3 months. The choice of 3 months reflects the average length of unemployment, when individuals are between jobs (1). Asset poverty tends to be more persistent than income poverty, making it more difficult to move out of asset poverty. Asset poverty could be a useful addition to health studies interested in better understanding the effects of financial deprivation.

There are several large longitudinal studies that collect high quality wealth data. Besides the Panel Survey on Income Dynamics (PSID), the Health and Retirement Survey (HRS) and the Survey of Income and Program Participation (SIPP) are other examples of longitudinal efforts which collect good wealth data. (The study known as Asset and Health Dynamics of the Oldest Old (AHEAD) merged with HRS in 1998.) The gold standard for wealth data comes from the Survey of Consumer Finances (SCF), which is a cross-sectional survey undertaken every 3 years. The SCF oversamples wealthy households in order to provide a better estimate of household wealth in the US. Given the skewed nature of wealth,

an assessment of the wealthiest Americans is essential to understanding the true distribution of wealth (5). The SCF also asks many more detailed questions than other surveys resulting in more accurate wealth measurement. The PSID, on the other hand, oversamples poor families; thus clearly underestimating the true distribution of wealth in the US.

Wealth inequality

Wealth inequalities in the US are considerable. Historical data show the existence of inequalities in household wealth dating back to the 1920s, where the top 1% of wealth holders owned on average 30% of the total wealth (15). Wealth inequality began increasing after 1979 and continued to increase through the 1980's. In the 1990's inequality levels remained fairly consistent. Recent estimates still indicate tremendous inequality. A 2004 estimate of the Gini coefficient for wealth was 0.81; the corresponding value for income was 0.54 (16). The Gini coefficient falls between 0 (wealth is evenly distributed across the population) and 1 (a single individual holds all the wealth). Given this interpretation, it is clear that wealth inequality in the US is far more extreme than income. Put in different terms, in 2004 the top 1% of wealth owners owned about 33% of the total wealth, while the top 10% owned about 70% of total wealth (16), a more skewed distribution than found in other developed nations (15).

Looking at the portfolios of different social classes also sheds light on the nature of wealth inequality. Most middle-class wealth is held in homes (about 60%) and in cash accounts (10%) compared to the wealthy that hold only a small amount of their assets in their homes (6%) but more in stocks (30%) and business assets. Among the middle-class and poor there is a much larger burden of consumer debt compared to the wealthy. Consumer debt does not allow for much savings and simply prolongs the process of consumption (5).

Wealth inequalities also fall strongly along racial lines. According to the Census Bureau in the year 2000 non-Hispanic whites had a median net worth of $67,000 compared to $6,166 for non-Hispanic blacks and $6,766 for Hispanics (17). Whites have more assets in all major subcategories (home equity, financial assets and real assets), but the differences is largest in financial assets and smallest in home equity (18). Since financial assets are more liquid than the others, white families have more resources in times of emergency. It should be noted that disparities exist within race groups as well, but the between race wealth differences are even larger (18). In addition, there is little room for wealth mobility in America. Most families are not able to accumulate enough wealth during their lifetimes to show any appreciable gains in wealth. Thus the advantages the wealthy have, primarily from inheritance, are perpetuated because wealth is difficult to accumulate (5).

Chapter 2

Critical review of the literature

WE will briefly review the existing literature for each of the health outcomes studied in this dissertation. Tables 1 and 2 provide a list of studies using longitudinal data to explore the relationship between wealth and smoking, obesity, hypertension, general health status or mortality. Most past studies used cross sectional data to explore the relationship between wealth and CVD risk factors (7, 16, 19-28) and the relationship between wealth and general health status (7, 11, 16, 29-32). It should be noted that several other health outcomes, not discussed here, have also been studied in relation to wealth. Researchers often use functional status as a health outcome, given the effectiveness of wealth as an SES measure for elderly populations. Other health outcomes such as depression and several healthcare utilization measures (e.g. physician visits and hospital stays) have also been found in the literature (8).

Smoking

The literature on smoking and wealth consisted mostly of cross-sectional studies of European or Australian populations (22, 26, 28) with 1 study of a US population (16). All cross sectional studies consistently found that the least wealthy had a higher prevalence of smoking (16, 22, 26, 28). One longitudinal study was interested in the economic effects of smoking (wealth was the outcome and smoking the main exposure); Zagorsky found that heavy smokers had 200% and light smokers had 50% less wealth compared to non-smokers

after adjusting for other demographic and SES factors (33). To my knowledge there have been no longitudinal studies where smoking was the health outcome and wealth the exposure.

Obesity

Few studies have looked at the relationship between obesity and wealth. All the studies described below use self-reported height and weight to calculate BMI. One cross-sectional study of a US population found a negative association between wealth and BMI, only among women (20). Another US study found no association between wealth and BMI, however wealth was crudely measured (24). A cross-sectional study of a British population found a negative association with wealth, again only among women (7), while a recent German study compared those with excess debt to those without debt and found those in debt to have higher odds of obesity (23).

Two studies by Zagorsky conducted longitudinal analyses using the National Longitudinal Survey of Youth. These studies specified wealth as the main outcome and BMI as the exposure and found a strong association: increasing BMI resulted in decreasing wealth both overall and among black and white women (34, 35). The covariate-adjusted results indicated that for every 1 point increase in BMI, net worth fell on average $1000 (34). The largest decline in wealth was seen among white women where $1800 was lost with every 1 point increase in BMI, followed by black women, where the decline in wealth was close to $600 (35). To my knowledge no longitudinal studies have looked at the effects of wealth (as the exposure) on obesity (the outcome).

Hypertension

Several studies have assessed the association between hypertension and wealth; with the exception of 1 (the study by Rooks and colleagues) all have used self-reported

hypertension data. Several cross-sectional studies found strong inverse associations between wealth and hypertension (7, 19, 21, 27), while another found no association between wealth and hypertension; however wealth was measured crudely (25). Similarly, a longitudinal analysis of an elderly population in the US showed no causal link between low wealth and increased incidence of hypertension (36). Adams and colleagues used the Granger test of causality to make this claim, a common method in econometrics used to understand if one time series predicts another. The authors did not, however, attempt to uncover the true causal structure of the relationship between wealth and hypertension (36).

Table 1. Longitudinal studies of the health-wealth relationship for CVD risk factors

Author (year)	Database	Wealth measure	Health measure	Results
Zagorsky (2004)	NLSY, 1984 to 1998	10 asset categories minus debt, continuous net worth variable excluding top 1% of wealth holders and log transformation of net worth	Smoking (heavy, light and non-smoker)	Heavy smokers and light smokers had less wealth compared to non-smokers
Zagorsky (2005)	NLSY, 1985 to 2000	10 asset categories minus debt, continuous net worth variable excluding top 1% of wealth holders and log	Obesity (self-reported height and weight)	Increase in BMI results in a large decrease in wealth for white women, a small decrease for black

		transformation of net worth		women and white men and no association for black men
Zagorsky (2004)	NLSY, 1985 to 2000	10 asset categories minus debt, continuous net worth variable excluding top 1% of wealth holders	Obesity (self-reported height and weight)	Increase in BMI by 1 point results in decrease in wealth of $900 per year
Adams (2003)	AHEAD, 1993 – 1998	11 asset categories minus debt, continuous variable transformed	Hypertension (self-reported)	No association between wealth and hypertension

NLSY: National Longitudinal Study of Youth

AHEAD: Asset and Health Dynamics of the Oldest Old

General Health Status

Most cross-sectional studies that have assessed the association between wealth and general health status have found a strong inverse association; that is higher wealth was associated with better health status (7, 11, 16, 29-32, 37). Several longitudinal studies from the economics literature found some evidence that poor health causes low wealth; testing the mechanism in the opposite direction that epidemiologists do (36, 38, 39). The study by Adams et al found limited evidence of the health to wealth association among an elderly US population. Their results indicated that poor or fair health status among married men actually resulted in *increased* savings (36). Hurd and Kapteyn found associations in both directions. Those in poor health had 16% less wealth relative to those in excellent health, while those in

fair health had 7% less wealth. Assessing the relationship in the other direction, they found that the wealthy had a higher probability of remaining healthy over time (38). Smith looked at the onset of mild and severe health problems and concluded that wealth declines by $3600 with the onset of a mild health problem and by almost $17,000 with the onset of a severe one (39).

Other research has concluded that the direction of the relationship is from wealth to health (38, 40, 41). Shuey and Willson used PSID data from 1984 to 2001 and excluded all those that were in poor health at baseline. They found strong evidence that greater wealth was associated with better health (41). Rodriguez and colleagues found a wealth-health association only among white men and women, wealthy white women have 19% higher odds of being in good health and wealthy white men 36% higher odds (40). A study by Meer et al, also using PSID data, employed an instrumental variable approach (inheritance is the instrument) and found that there is no short term effect of wealth on perceived health status. This study did, however, find an effect of very small magnitude when using traditional statistical approaches (42).

Mortality

Several US studies that used a reliable measure of wealth found a positive association between wealth and mortality (10, 43-47). There are, however, 2 US studies that did not find an association with mortality (36, 48). The study by Feinglass et al concluded the lack of association between wealth and mortality after 10 years of follow-up was a result of controlling for baseline health status, an important mediator in the wealth-mortality association (48). In addition, Adams and colleagues tested specifically for the "absence of causal links from wealth to mortality" and found evidence to support the claim. In addition,

they concluded that death of a spouse had a negative effect on the *subsequent* wealth of the surviving family members (36). All the mortality and wealth studies published thus far, use wealth data from a single time point and follow participants for several years to ascertain mortality. The longest follow-up period was 17 years, both from the studies using data from the NLSMM (10, 43) and the shortest was Attanasio's study which had a 2.5 year follow-up period (47).

Table 2. Longitudinal studies of the health-wealth relationship for general health status and mortality

Adams (2003)	AHEAD, 1992 – 1998	11 asset categories minus debt, continuous variable transformed	GHS	Poor health causes low wealth only among married men
Hurd (2002)	HRS, 1994 – 2000	8 asset categories minus debt	GHS	Finds evidence in both directions: change in health status causes change in wealth AND change in wealth causes change in health status.
Meer (2003)	PSID, 1984 to 1999	9 asset categories minus debt	GHS	Wealth effects health but the magnitude is small. Using inheritance as an instrument results in no association between wealth and health.

				Thus they conclude there is no short-term causal effect of wealth on health.
Smith (1999)	HRS, 1992 - 1996 AHEAD, 1993 - 1996	See Adams and Hurd above	GHS	New episodes of poor health lead to lower wealth accumulation.
Rodriguez (1999)	NSFH, 1987 – 1992	Assets and debts, log transformed	GHS	Assets were associated with good health among white men and women, but not among black men or women.
Shuey (2008)	PSID, 1984 – 2001	See Meer above. Log transformed and adjusted for inflation and household size	GHS	Greater wealth associated with better health.
Mare (1990)	NLSMM, 1966	10 asset categories minus debt, wealth specified as quartiles	Mortality	Less wealthy have higher mortality
Menchik (1993)	NLSMM, 1966 17 years of	10 asset categories minus debt	Mortality	Less wealthy have higher mortality

	follow-up			
Duncan (2002)	PSID, 1984 10 years of follow-up	See Meer above	Mortality	Less wealthy have higher mortality
Bond Huie (2003)	HRS, 1992 6 years of follow-up	See Hurd above, log transformed	Mortality	Less wealthy have higher mortality
Cunningham (2005)	HCSUS, 1996 4 years of follow-up	1 question minus assets, specified as 5 categories	Mortality	Low wealth categories have higher risk of mortality, HR not significant for higher wealth categories
Attanasio (1995)	SIPP, 1984 – 1987. 2.5 yr follow up	8 asset categories minus debt	Mortality	Less wealthy have higher mortality
Feinglass (2007)	HRS, 1992 10 years of follow-up	See Hurd above, specified as 3 category variable ($< 20^{th}$ percentile, $20 – 60^{th}$ percentile and $> 60^{th}$ percentile)	Mortality	After adjusting for baseline health status, no association between wealth and mortality
Adams	AHEAD,	See Adams above	Mortality	Found an absence of a causal

(2003)	1992 6 years of follow-up			link from wealth to mortality

AHEAD: Asset and Health Dynamics among the Oldest Old

GHS: self-reported general health status

HRS: Health and Retirement Survey

PSID: Panel Survey on Income Dynamics

NSFH: National Survey of Families and Households

NLSMM: National Longitudinal Survey of Older Men

HCSUS: HIV costs and service utilization study

SIPP: Survey of Income and Program Participation

Chapter 3

Specific Aims

The overall goal of this research is to better understand the magnitude and direction of the relationship between wealth and several health outcomes on both the absolute and relative scales using longitudinal data.

Aim 1: Investigate the association between wealth and 3 cardiovascular disease risk factors: smoking, hypertension and obesity on the absolute and relative scales in the time period 1999 - 2005.

Rationale: One of the leading hypotheses for why the poor have worse health outcomes compared to the wealthy relates to the presence of chronic stress in the lives of the poor. Cardiovascular disease risk factors were chosen as health outcomes because chronic stress has been hypothesized to result in more smoking, obesity and hypertension. Thus the association between wealth and CVD risk factors has implications for the pathways by which low SES causes ill health. Furthermore, very little work has explored the relationship between wealth and these health behaviors.

Aim 2: Investigate the overall, gender and race specific association between wealth and all-cause mortality and self-reported general health status on the absolute and relative scales in the time period 1984 - 2005.

Rationale: Many studies have used all-cause mortality and self-reported GHS as health outcomes when investigating the wealth-health relationship. Using these commonly

used outcomes will allow this research to be directly comparable to past research. In addition, the long follow-up time makes these outcomes of particular interest. The known race and gender differentials in mortality require that these outcomes be looked at within strata of these demographic variables.

Chapter 4

Methods

Population

Data for this study came from the Panel Survey on Income Dynamics (PSID), a rich longitudinal study with up to 38 years of follow-up on a nationally representative sample of US households. PSID began in 1968 and continues today. It is intended to be representative of the non-institutionalized, civilian US population. In 1968, PSID oversampled low-income African-American families from urban areas in the North and rural areas in the South. A second sample, also collected in 1968, selected participants from the 48 contiguous states with an equal probability of selection. All participants selected in 1968 were between the ages of 25 and 74. For this study both samples were used (referred to as the PSID core).

Over time, PSID has been forced to make changes to its original sample in an attempt to address the large and growing immigrant population. A large number of Latino families (n=2,000) were followed from 1990 to 1995. This sample was discontinued because it did not account for America's growing Asian population and because of funding constraints. In 1997 a small sample of immigrant families (n=511) joined the study and have been followed ever since.

As members of the original PSID families have children who in turn form their own family units, the new families are also followed. PSID collects data on each member of the family unit, defined as all those living in the same household not including temporary roommates or visitors, however, more detailed information is collected on the head of

household (usually male), followed by his "wife" (either by marriage or cohabitation). Since health data is only collected for the head and "wife", they are of primary interest in this study. As of 2005 there were more than 8,000 families enrolled in PSID, resulting in more than 15,000 individuals interviewed. Today, most PSID families descend from the original sample and are either non-Hispanic black or non-Hispanic white.

Data collection was done annually from 1968 to 1997, but since 1997, PSID has moved to a biennial data collection. Currently all surveys are done via computer-assisted telephone interviews and lasts about 75 minutes. Originally designed as a way to study the dynamics of income and poverty, PSID's core content focuses on family economics and demographics. Income data are a central part of the PSID's core content and are collected at every wave for each person in the family that is older than 16 and earning some income.

Variables

Wealth: Main exposure

Data collection on family wealth began as a supplement to the core and was funded by National Institute for Aging. Currently wealth data are asked in every survey year. Wealth data were collected in the 1984, 1989 and 1994 (as supplements) and 1999 – 2005 waves of the survey. When measuring family wealth PSID takes into account several sources: net value of the respondent's main home, other real estate, vehicles, any farms or businesses, stocks, IRAs and other financial instruments, cash accounts such as money market funds and certificates of deposit and other assets including value of estates, life insurance policies, pensions and inheritance. Any outstanding mortgage principal and other debts are subtracted from these assets. Table 3 lists the questions from the wealth module in PSID. Future social security income and pension income are not included as potential

sources of wealth because they are not a reliable liquid asset and are only available to individuals older than 65 years old. Instead social security and pension incomes are incorporated into family income for those receiving either one.

Table 3. Survey questions used to measure wealth in PSID, 1984 - 2005

1. The next questions I will be asking are designed to give estimates of wealth of families in the United States and how this has changed in the last five years. In these questions when I refer to family, I mean your family living there with you. Do you (or your family living here) have any **real estate other than your main home**, such as a second home, land, rental real estate, or money owed to you on a land contract?*
1a. If you sold all that and paid off any debts on it, how much would you realize on it?
2. What about the value of what you (or anyone in your family living there) own **on wheels**? Including personal vehicles you may have already told me about and any cars, trucks, a motor home, a trailer, or a boat--what are they worth all together, minus anything you still owe on them?**
3. Do you (or anyone in your family living there) own part or all of a **farm or business**?
3a. If you sold all that and paid off any debts on it, how much would you realize on it?
4. Do you (or anyone in your family living there) have any **shares of stock in publicly held corporations, mutual funds, or investment trusts**, not including stocks in employer-based pensions or IRA's?
4a. If you sold all that and paid off anything you owed on it, how much would you have?

5. Do you (or your family living there) have any **money in private annuities or Individual Retirement Accounts** (IRAs)?
5a. How much would they be worth?
6. Do you (or anyone in your family living there) have any **money in checking or savings accounts, money market funds, certificates of deposit, government savings bonds, or Treasury bills**, NOT including assets held in employer based pensions or IRA's?
6a. If you added up all such accounts (for all of your family living there), about how much would they amount to right now?
7. Do you (or anyone in your family living there) have any **other savings or assets, such as bond funds, cash value in a life insurance policy, a valuable collection for investment purposes, or rights in a trust or estate** that you haven't already told us about?
7a. If you sold that and paid off any debts on it, how much would you have?
8. Aside from the debts that we have already talked about, like any mortgage on your main home or vehicle loans – do you (or anyone in your family living there) currently have any **other debts such as credit card charges, student loans, medical or legal bills, or loans from relatives**?
8a. If you added up all of these debts (for all of your family living there), about how much would they amount to right now?

*Questions about the value of main home and money owed on it are derived from a series of questions in the housing section (Section A: Housing in 2005 survey).

**Questions about car loans are derived from a series of questions in the transportation section (Section F: Housework, childcare, food, vehicles and expenses in 2005 survey).

Total family wealth is then calculated by PSID in 2 ways. First, net financial assets add all the sources of wealth excluding home equity and second, net worth adds all the sources of wealth including home equity. Net worth was used in all final models. Given the importance of home values to the wealth portfolio of many middle-class American families, using net assets would have underestimated wealth for many PSID families. The average annual consumer price index for urban households was used to adjust family wealth for inflation to 2000 dollars. The final wealth variable was specified as a 6 category variable, where all families with less than or equal to 0 wealth were put in 1 category and the remaining 5 categories were quintiles of positive wealth. A categorical wealth specification was decided upon in order to avoid the problems associated with the skewed nature of wealth. Some researchers have undertaken log transformations of wealth; however, this still results in a skewed distribution. To avoid violating the assumption of linearity the 6 category wealth variable was specified as 5 indictor terms in the final models, where the highest wealth quintile was the referent group.

As wealth was the main exposure of interest in this dissertation, we tried several specifications of the wealth variable. One specification of wealth tried early on was a categorical variable that used deciles of positive wealth and 2 additional categories, 1 for those with negative wealth and the other for those with 0 wealth. Descriptive analysis with this 12 category wealth variable quickly revealed that the number of individuals in each category was relatively small resulting in large standard errors. In addition, we also tried a 7 category wealth variable. This variable is similar to the one used in the final models except,

those with 0 wealth were separated from those with negative wealth. Additional assessment of this 7 category variable revealed little difference in point estimates between these 2 low wealth groups and large confidence intervals around the point estimates, thus deeming the separation of the less than or equal to 0 wealth category unnecessary. In addition, concerns about violation of the positivity assumption (having no respondents with a particular covariate pattern) further drove the decision to use the 6 category wealth variable.

Health outcomes

Data collection on the health of PSID participants began in 1984 with a short module assessing general health status, activity limitation, hospitalization and health insurance. Similar questions were asked in 1985 and then again in 1987 - 1997. Substantial changes to the health module occurred in 1999, with the inclusion of questions regarding physician diagnosed conditions such as stroke, heart attack, cancer, asthma and hypertension, as well as questions about risk factors such as exercise, smoking, alcohol and self-reported height and weight. A summary of the health outcomes used in this study are presented in Table 4.

Smoking data were obtained from 1999 – 2005 of the PSID. The questions used to ascertain smoking have not changed over the years and were used to categorize respondents as current versus non-smokers. Respondents were first asked "Do you smoke cigarettes?" Those who responded "yes" were classified as current smokers. Those who responded "no" were then asked "Did you ever smoke cigarettes?" Those who responded "no" to the follow-up question were considered never smokers, while those who respond "yes" were considered former smokers. These 2 groups were collapsed to form the non-smokers group.

Body Mass Index (BMI) is a commonly used metric to assess obesity or overweight in a population. Using an individual's height and weight, BMI was calculated by dividing

weight in pounds by height in inches squared and multiplying by a conversion factor of 703. BMI values that are less than 24.9 were considered normal weight; 25 to 29.9 was considered overweight and 30 and above was considered obese (49). Self-reported height and weight were obtained from PSID in 1999, 2001, 2003 and 2005. The questions are: "About how much do you weight?" and "How tall are you?" Respondents were asked to respond in pounds and inches, or as a second choice in kilograms and centimeters. For analysis, we were interested in the incidence of obesity, therefore normal and overweight individuals were grouped together.

Hypertension was ascertained from the following question: "Has a doctor ever told you that you have or had high blood pressure or hypertension". This question was asked in the 1999, 2001, 2003 and 2005 waves of the survey. Those with controlled hypertension should be captured by this question, as it asks about past diagnosis of hypertension. Anyone responding "yes" to the question was considered hypertensive, and those who respond "no" were not.

Self-rated general health status (GHS) is the respondent's assessment of his/her current health status. General health status was captured with the following question: "Would you say your health in general is excellent, very good, good, fair, or poor?" The wording of this question has not changed since it was first asked in 1984. GHS is available for 7 waves of the study 1984, 1989, 1994, 1999, 2001, 2003 and 2005. Responses were dichotomized by grouping all respondents who indicated excellent, very good or good versus those who responded fair or poor.

Mortality data was available through special contract with PSID because of its sensitive nature. The mortality file was linked to the core PSID dataset by matching

respondents on their unique identification number. Death was recorded as a possible reason for non-response. The discovery of a participant's death occurred during the following year's interview with surviving family members or mail returned from the post office. After a death was discovered, PSID used the National Death Index (NDI) to confirm the death. The death file included several variables on which the quality of the match with the NDI can be assessed: date of birth and death and place of both birth and death. Social security numbers were not collected by PSID. Additional discussion of the quality of the mortality data in PSID can be found in the discussion section of Chapter 6.

Table 4. Outcome variables from PSID, 1984 - 2005

Name	Definition	Source	Form
Mortality	Respondent's with a date of death were classified as dead; those without a date of death were considered alive	PSID mortality file from 1968 - 2005	0 = alive 1 = dead
General Health Status	Excellent, very good or good indicated good health; fair or poor indicated poor health status	PSID core file 1984 - 2005	0 = good 1 = poor
Smoking	Those who currently smoke cigarettes were considered smokers; those who used to smoke or who never smoked were non-smokers	PSID core file 1999 -2005	0 = never or former smokers 1 = current smokers
Obesity	$\leq$ 29.9 was overweight or normal weight $\geq$ 30 was obese	PSID core file 1999 -2005	0 = normal weight or overweight 1 = obese
Hypertension	Respondents answering "No" to question on hypertension were not hypertensive, those	PSID core file 1999-2005	0 = not hypertensive 1 = hypertensive

	answering “Yes” were hypertensive		

Covariates

The following confounders were included in this study: age, race, sex, education, region of residence, general health status, marital status, health insurance status and family income. Table 5 lists the baseline and time-varying covariates which were used in conjunction with each outcome variable. A more detailed description of how each covariate was specified can be found in Chapter 5 and 6 below.

Table 5. Baseline and time-varying covariates for each health outcome, PSID 1984 - 2005

	Smoking	Obesity	Hypertension	General health status	Mortality
PSID waves	1999, 2001, 2003, 2005	1999, 2001, 2003, 2005	1999, 2001, 2003, 2005	1984, 1989, 1994, 1999, 2001, 2003, 2005	1984, 1989, 1994, 1999, 2001, 2003, 2005
Covariates measured at baseline	Age, race, sex, region, education, general health status, years	Age, race, sex, region, education, general health status, years	Age, race, sex, region, education, general health status, years	Age, race, sex, region, education, years participated	Age, race, sex, region, education

	participated in study	participated in study	participated in study	in study	
Time-varying covariates*	Income, marital status, health insurance status**	Income, marital status, health insurance status	Income, marital status, health insurance status	Income, marital status	Income, marital status, general health status

*Baseline status of all time varying variables were also included in all models.

**Health insurance status was not collected in 1984, 1989 or 1994.

Region of residence was not available in the 1994 PSID, therefore, 1993 values were used in there stead. In addition, several covariates had modest missing data (generally around 2%). Data from the previous year was used to impute any missing data values for age, education, marital status, region, general health status and race. For example, among those entering the study in 1984, 132 individuals were missing education data. If those with missing data had participated in the 1983 survey, data from 1983 were used to impute these missing education values. This reduced the amount of missing education data in 1984 by 18%.

As per the suggestion of a former committee member, religion was also explored as a potential covariate for the wealth health relationship. Some sociological work has documented different wealth patterns among the various religious faiths (50). Ultimately, we did not include religion as a covariate because the questions about religion in PSID did not

allow for the creation of meaningful religious groups. Furthermore, the religion questions changed substantially from year to year of the survey.

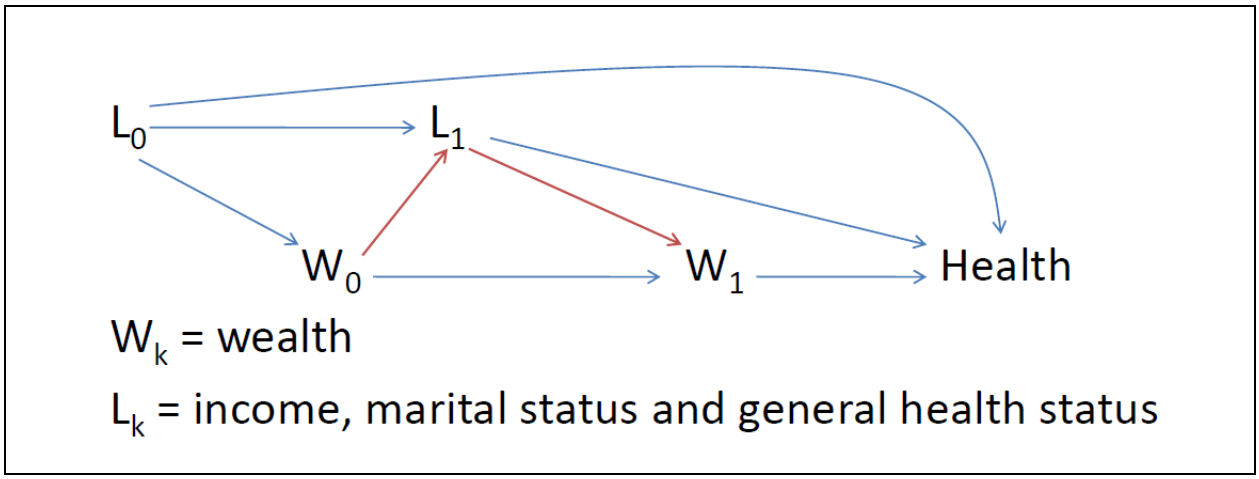
Person time was also included as a covariate in each model, except for mortality. Person time was calculated in years from baseline to the end of follow-up. Some assumptions were made in order to calculate person time for those who were lost to follow-up. Each individual was given 1 person year for each year they participated in the study between 1984 and 1996. For example, if someone entered the study in 1984 and their last interview was in 1990, they were assigned 7 person years. The move to biennial data collection changed the way person time was calculated. Individuals who participated for consecutive years were assigned 2 person years, but those that dropped out between 1997 and 2005, were assigned 1 person year. This was done because the exact date of drop out was unknown. For example, if someone entered the study in 1992 and their last interview was 2001, this individual was assigned 11 person years. Since we know this individual did not participate in 2003, we can assume they were lost to follow up sometime after the 2001 interview but before the 2003 interview. The additional year was assigned to account for the uncertainty of drop out time.

Statistical methods

In epidemiology and related fields, marginal structural models (MSM) are increasingly used to handle time-varying confounding. A variable is a time-varying confounder if it can be influenced by previous exposure and can subsequently influence future values of the exposure (51). Figure 1 below depicts a simplified directed acyclic graph (DAG) of time-varying variables, where the vector of time varying confounders is represented by L_k and the main exposure wealth is represented by W_k. The red line from W_0

to L_1 represents the influence of wealth at time 0 on the time-varying confounders and the second red line from L_1 to W_1 represents the subsequent influence of the time-varying confounders at time 1. Traditional methods may produce biased estimates in the face of time-varying confounding (51, 52), therefore, the use of MSM is recommended.

Figure 1. Directed acyclic graph depicting time-varying confounding



The use of inverse probability weights (IPW) is central to MSM. Weighting creates a pseudopopulation where an individual can be represented as a fraction of oneself or more than once. In this pseudopopulation confounding is controlled by eliminating the association between the confounders and the exposure (51). The IPW can be informally described as the probability that an individual received their observed exposure history. Calculating IPW is the first step in using MSM. In this study IPW were calculated separately for each outcome because exclusion criteria resulted in different analytic populations.

IPW are made up of 2 components. The equation below shows the treatment weight, $sw_i(t)$, which was modeled using an ordinal logistic regression where the outcome is the 6 category wealth variable, A. As indicated above, the highly skewed nature of wealth does not allow for the use of a continuous specification in the treatment weight model. Some

MSM experts believe that the specification of exposure in the treatment weight model can be finer than the specification of exposure in the final model, but not vice versa. This may better control for time-varying confounding (personal communication with Stephen Cole). Thus our use of the 6 category wealth variable in both the weighting and final models was necessary given the highly skewed nature of wealth would not allow for a continuous specification in the weighting model when wealth is considered the outcome.

The participant's covariate history is designated by $\bar{A}$. The numerator of the treatment weight model includes all baseline covariates, V. For the CVD risk factor model this includes baseline values for age, race, sex, education, region, income, marital status, general health status and health insurance status. The denominator includes all baseline covariates, time-varying covariates (income, marital status, general health status and insurance) as well as significant interaction terms of baseline covariates, L(t). Although the inclusion of the baseline covariates in the numerator and the denominator may seem redundant, it helps to reduce the variability of the weight avoiding a situation where a few observations contribute excessively to the weighted analysis. In turn a less variable weight produces a final parameter estimate that has a smaller variance and is approximately normally distributed (51). The treatment weight is defined as the conditional probability that the subject received his/her own observed level of wealth at each time point.

$$sw_i(t) = \prod_{k=0}^{t} \frac{pr\ (A(k) = a_i(k) | \bar{A}(k-1) = \bar{a}_i(k-1), V = v_i)}{(A(k) = a_i(k) | \bar{A}(k-1) = \bar{a}_i(k-1), \bar{L}(k) = \bar{l}_i(k-1))}$$

The second component of the IPW, the censoring weight, $sw_i^{\dagger}$, was modeled with logistic regression. The outcome is defined as whether or not an individual was censored or

loss to follow up, C, where individuals who are lost to follow up are coded as 1 and 0 otherwise. The participant's censoring history is designated by $\bar{C}$. Individuals are censored at the last time point for which they have data. The censoring weight is defined as the conditional probability that the subject remains uncensored up to time t and is presented in the equation below.

$$sw_i^{\dagger} = \prod_{k=0}^{t} \frac{pr\ (C(k) = 0 | \bar{C}(k-1) = 0, \bar{A}(k-1) = \bar{a}_i(k-1), V = v_i)}{pr\ (C(k) = 0 | \bar{C}(k-1) = 0, \bar{A}(k-1) = \bar{a}_i(k-1), \bar{L}(k-1) = \bar{l}_i(k-1)}$$

The censoring and treatment weights were then multiplied to produce the final stabilized weight. The stabilized weight thus incorporates all measured time-varying confounding. The final binomial MSM is as follows, where V is the vector of baseline covariates:

$$\ln(Pr(Y|a)) = \beta_0 + \beta_1(a) + \beta_2 V$$

Notice the absence of time-varying covariates in this model. The incorporation of the IPW makes the addition of time-varying confounders unnecessary. The baseline covariates are in both then numerator and denominator of both weighting models effectively cancelling each other out, therefore, adding baseline covariates to the final model is necessary. The parameter β_1 is the causal risk ratio of wealth on smoking, obesity or hypertension, for example.

Further discussion of the creation of IPW is warranted. IPW are predicted probabilities which are calculated from the data. Data must be formatted so that there is 1 observation per time point per person. Given this data structure, a different weight is calculated for each time point the individual participates in the study (the weight is time-

varying). Baseline and time-varying variables are assigned to each observation in the dataset.

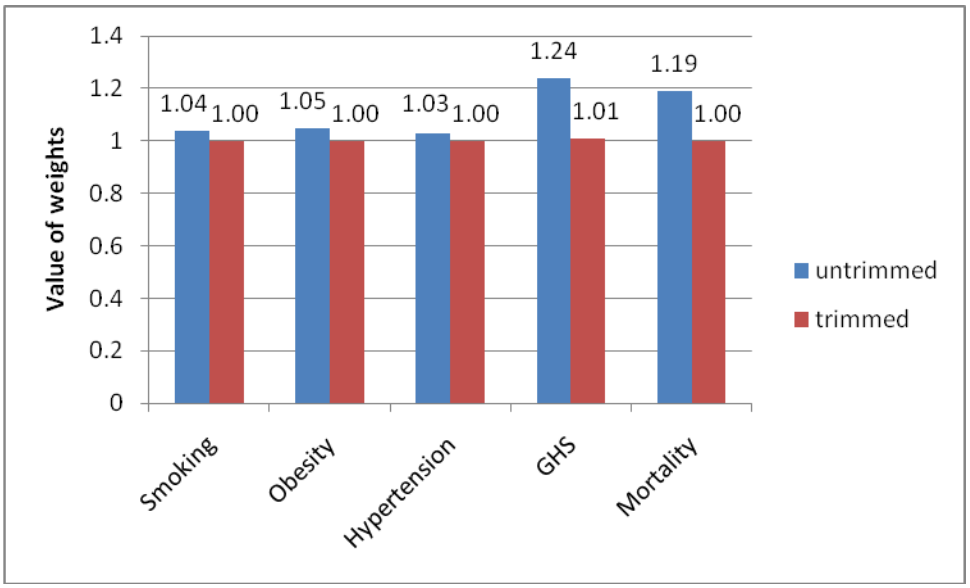
As stated above, the treatment weights were calculated from ordinal logistic regression, where wealth is the outcome. Given that wealth is a 6 category ordinal variable, the model calculates the predicted probabilities for each level of wealth for each participant at each time point. The predicted probability which corresponded with each participant's observed wealth value for each time point was then calculated and extracted into a separate dataset.

The goal of the weighting models is to produce the best predicted probability possible. In order to achieve this goal variables should be specified flexibly and all significant predictors should be included. Therefore, several interaction terms were added to the denominator of each weighting model. Interactions between baseline variables had to be at the 0.35 significance level to be included in the denominator of the treatment and censoring weight. Hierarchically specified models are not a requirement for prediction models, thus if one level of an interaction term was significant but another was not, only the significant term was retained. The censoring weight models are similar to the treatment weight models; this is done for ease of fitting the models, but is not a requirement. Predicted probabilities calculated from logistic regression are the censoring weights. The predicted probability from the numerator models is then divided by predicted probability from the denominator models to produce the final IPW.

At each time point, IPW should have a mean of 1 and a small range (53). In our study the initial weights had a mean that was slightly greater than 1. For example for the obesity model the overall mean of the IPW was 1.10, the smallest weight was 4.09×10^{-7} and

the largest was 164. Very large and very small IPW are generally a result of odd covariate patterns. For example, if respondents had extreme fluctuations of income over the study period, this could result in excessively large or small weights. In order to reduce the range of the weight, which ultimately reduces the variability of the final estimates, weights can be truncated (53). In this study 2 percent of the largest and smallest weights were truncated to obtain better behaved IPW. For obesity the weight corresponding with the 2nd percentile becomes the minimum, 0.31, and the weight corresponding to the 98th percentile becomes the maximum, 2.56. The distribution of the trimmed and untrimmed weights is given in Figure 2.

Figure 2. Mean value of trimmed and untrimmed weights for each outcome



Several weighting models were fit to determine which produced the best weights. We started with the simplest weighting model, which includes age and income specified as continuous variables, all other covariates as indicator terms and no interaction terms, and moved to the most complex model which includes age and income specified as squared, cubed and spline terms and the inclusion of all significant interaction terms. In general the

more complex weighting models produced the best weights (mean close to 1 and narrow range).

One final detail involving the intercept of the weighting model should be discussed. The person-time specific intercept for all weighting models was estimated using a smoothing technique. This is recommended to reduce the variability of the final weight (52). A SAS macro, rcspline, was used to fit natural cubic splines (54). For the CVD risk factor models, we used 3 knots placed at the 5^{th}, 15^{th} and 50^{th} percentile of time. Usually more knots are placed at the tails of the distribution, however in this study of CVD risk factors the time distribution was skewed making multiple knots at the high end of the distribution redundant. For the GHS analysis 5 knots were used; placed at the 5^{th}, 27.5^{th}, 50^{th}, 72.5^{th} and 95^{th} percentile.

Assumptions

Several assumptions must be tested and met in order to ensure the integrity of the model. MSM are sensitivity to the assumption of positivity, which states there must be both exposed and unexposed individuals at every level of the confounders (53). That is if it is impossible to be exposed at 1 or more levels of the confounders than positivity is violated. In this study, it is feasible for some individuals to have low or no wealth regardless of the level of the confounders. Although some may find it unlikely that an individual with high income would have low wealth, this situation may indeed be fairly common among persons with substantial debt. In practical terms, the positivity assumption may be violated because of so-called random zeros. Continuous variables are especially sensitive to random zeros since it may be difficult to find both an exposed and unexposed person at every level of the continuous variable. For categorical variables, however, collapsing similar categories may

help alleviate problems with random zeros. We looked for the presence of random zeros among the categorical confounders and collapsed several categories as a result.

For continuous and ordinal variables we also checked the linearity assumption. Since the weighting models are prediction models, violation of linearity is less important for these first stage models. However, in the final models, linearity must be met. Thus we assessed the linearity of baseline age and income in relation to each of the 5 health outcomes. If non-linear patterns were detected, the variables were specified as indicator variables to avoid the violation of linearity. As noted above, the skewed nature of wealth required that it be specified as 5 indicator terms in order to avoid the violation of linearity.

Clustering

Since data for both heads of household and their partners was available from PSID, there was clustering by household. Furthermore, given the longitudinal nature of the analysis data were formatted vertically (there was 1 observation per time point per person), resulting in additional clustering by individual. Using the highest level of clustering (the household) produced results that control for both household and individual clustering (55, 56). Therefore, a family id variable was used to indicate the unit of clustering (with an independent correlation structure) thus obtaining robust standard errors. As a check, we ran several models where individual level clustering was specified. The standard errors for these models were very similar but, as expected, smaller than those from models where clustering at the household level was specified. Table 6 provides a comparison of the standard errors from these 2 models for each category of wealth for 2 randomly selected outcomes.

Table 6. Standard errors for models with clustering at the family level versus the individual level

		≤ 0 wealth	Quintile 1	Quintile 2	Quintile 3	Quintile 4
Obesity	Family level clustering	0.118	0.121	0.113	0.106	0.102
	Individual level clustering	0.115	0.117	0.110	0.103	0.097
GHS	Family level clustering	0.101	0.093	0.093	0.085	0.081
	Individual level clustering	0.095	0.088	0.87	0.081	0.076

Risk difference models

Recently researchers have proposed methods for obtaining absolute measures of effect (i.e. risk difference) for MSM (57, 58). We attempted 3 methods for calculated risk differences before a satisfactory risk difference model was found. First, we attempted a method proposed by Westrich and colleagues (58). This method calculated survival probabilities for each level of exposure by summing the inverse probability weighted number of cases and dividing by the total number of individuals in the pseudopopulation (i.e. the sum of the weights). This quantity was then subtracted from 1 to obtain the survival probability. The risks calculated from these survival probabilities effectively controlled for time-varying

confounding. The problem with this method, however, is that it does not control for baseline covariates. In our study, controlling for baseline covariates such as age, race and sex are essential to obtaining an unbiased estimate. Therefore, after attempting various fixes to this method, we concluded it could not be used.

Second, we tried running linear risk models. These were general linear models which specified the identity link and the binomial distribution. Although these models allow for the control of both baseline and time-varying confounders, several of these models did not converge. Even after attempting to use starting value from the normal distribution, non-convergence problems persisted. Therefore, these models were also abandoned.

Lastly, we used a method common to economists, marginal effects. Predicted probabilities from logistic regression were used to calculate risk differences. In Stata, the post estimation command mfx calculated confidence intervals for these risk differences using the delta method (59). Estimates were standardized to the covariate distribution of the total study population. A full description of this method can be found in chapter 21 of the Econometrics textbook by William Greene (60).

Chapter 5

Cardiovascular disease risk factors and wealth

Abstract

Background: The use of wealth as a measure of socioeconomic status (SES) remains uncommon in epidemiological studies. When used, wealth is often measured crudely and at a single point in time.

Objective: Our study explores the relationship between wealth and 3 cardiovascular disease (CVD) risk factors (smoking, obesity and hypertension) in a US population.

Methods: We improve upon the existing literature by using a detailed and validated measure of wealth in a longitudinal setting. We used 4 waves of data from the Panel Survey on Income Dynamics (PSID) collected between 1999 and 2005. Inverse probability weights were employed to control for time-varying confounding and to estimate both relative (risk ratio) and absolute (risk difference) measures of effect. Wealth was defined as inflation-adjusted net worth and specified as a 6 category variable: 1 category for those with less than or equal to 0 wealth and quintiles of positive wealth.

Results: In the fully adjusted model, the risk of becoming obese was inversely related to wealth. There was a 32% to 77% higher risk of becoming obese among the less wealthy relative to the wealthiest quintile and 11 to 27 excess cases (per 1000 persons) among the less wealthy groups over 6 years of follow up. Smoking initiation had similar but more moderate effects; risk ratios and differences both revealed a smaller magnitude of effect compared to obesity. Of the 3 CVD risk factors examined here, hypertension incidence had the weakest

association with wealth, showing a smaller increased risk and fewer excess cases among the less wealthy groups.

Conclusion: This study found a strong inverse association between wealth and obesity incidence, a moderate inverse association between wealth and smoking initiation and a weak inverse association between wealth and hypertension incidence.

Introduction

The goal of this study was to explore the relationship between wealth and three cardiovascular disease (CVD) risk factors, smoking, obesity and hypertension; specifically, do less wealthy individuals have a higher risk of becoming obese, smokers or hypertensive. In epidemiological studies the use of wealth as a measure of socioeconomic status (SES) is uncommon. Wealth is defined as the stockpile of financial resources amassed over the lifetime, while income is the flow of resources into the household at any given point in time (4, 5).

There are several advantages to using wealth as a measure of SES. First, since it is less subject to fluctuations over one's lifetime than income, wealth is often a more stable measure of SES. Since wealth is often inherited over the generations it reflects a historical accumulation of assets. Second, wealth may be a better measure of social hierarchy compared to other SES measures (3). In addition to being an economic indicator, wealth buys political power, social prestige, and educational and occupational opportunities that income alone may not allow (4, 5). That is, wealth encompasses economic circumstances as well as prestige and status.

Finally, wealth may be a better measure of one's economic situation at various points in the life course. For example, during times of unemployment or illness when income is lost, wealth may help maintain living standards. During these times, measures of employment status or income may misrepresent an individual's economic situation. Some studies have shown that SES measures are not interchangeable; instead they may work through different mechanisms at different times in the life course (6, 7). For example, wealth may be an especially good SES measure for older populations, when income is limited or

absent (5, 8). In fact, several studies have shown a strong association between wealth and health in the elderly population (9-11). Given these examples, it is easy to understand how wealth can work independently of income to impact health (8).

Wealth is not often used in health studies because it is difficult to measure effectively. A thorough assessment of wealth would require asking several sensitive questions about the value of personal property, debt and financial instruments. Since wealth data are self-reported and difficult to validate, the possibility of poor measurement exists. Interestingly, the underestimation of assets by the wealthiest Americans has limited our ability to understand the true disparity in wealth that exists in the US (12). In the health literature, measures of wealth are not consistent and are sometimes simplistic; thus making comparisons across studies difficult and masking potential associations between wealth and health outcomes (8).

Most past studies used cross sectional data to explore the relationship between wealth and CVD risk factors (7, 16, 19-28). Our study of wealth and CVD risk factors improves upon the existing literature by using a thorough and reliable measure of wealth (61) from a longitudinal study, the Panel Survey on Income Dynamics (PSID). Our choice of PSID was motivated not only by its detailed wealth measurement, but also by a desire to evaluate the wealth-health relationship in a non-elderly adult US population. Much work has looked at wealth and CVD risk factors in the elderly (7, 19-21, 25, 27, 36), but fewer studies have examined the effect of wealth on health in a population of established adults.

Methods

Data

PSID is a longitudinal study of the US population which began in 1968 and continues today. Currently data are collected biennially. Although PSID was designed to be representative of the non-institutionalized, civilian US population, survey weights were not used in this study. The use of survey weights would have complicated the analytic method we used which also relies on analytic weights. Thus results from this study are not generalizable to the broader US population. Much has been written about the design and content of the PSID elsewhere (62). Data for this study came from the 1999, 2001, 2003 and 2005 waves of the PSID. Regular data collection of the health module began in 1999 and has continued in each wave since. Health questions were asked only of the head of household and his/her partner.

Measures

Health outcomes: The three health outcomes examined in this study, obesity, smoking and hypertension, were self-reported. Obesity was derived from self-reported height and weight and was classified using a body mass index of 30 or higher. Smoking status was derived from a series of questions on smoking behavior allowing participants to be classified as current, former or never smokers. Lastly, hypertension was ascertained from a single question, "Has a doctor ever told you that you have or had high blood pressure or hypertension".

Wealth: Family wealth was defined as total net worth, which includes the value of one's primary home, farm or business assets, checking or savings accounts, vehicles, second homes, stocks and bonds. Debt was subtracted from these assets. All wealth data were adjusted for inflation using the 2001 consumer price index. Wealth was specified as a six

category variable, where category one included all those that have negative or zero wealth and categories two through six were quintiles of positive wealth. In the final models, wealth was specified as five indicator terms (i.e. dummy variables) to avoid violating the linearity assumption.

Confounders: Other covariates included in this study were income, marital status, self-reported general health status, region of residence, age, education, race, sex and health insurance status. Income was specified as a continuous poverty-to-income ratio using the annual poverty thresholds from the Census Bureau and accounting for number of people in the household. In the final model, income was linearly related to obesity and smoking but required indicator terms to avoid violating the linearity assumption for hypertension. Marital status was categorized as married (the referent group), never married or divorced, separated or widowed, and specified as indicator terms. Self-reported general health status was dichotomized as excellent, very good and good versus fair and poor. The three indicator variables for region classified state of residence as northeast, midwest or south; west was the referent group. Age was linearly associated with hypertension, but due to convergence problems was specified as six indicator terms (30-34, 35-39, 40-44, 45-54, 55-64 and $\leq$ 65 years old, referent was $\leq$ 29 years old). The final model for obesity used two indicator terms representing less than or equal to 44 years old and 45-64 years old and the final model for smoking used less than or equal to 39 years old and 40-64 years old (referent group was $\leq$ 65 years old for both models). These categories were based on analysis that explored the shape of the relationship (e.g. linear, u-shaped etc) between age and obesity and age and smoking. Education was specified as two indicator variables, (< high school and high school degree) and greater than high school was the referent group. An indicator variable for race included

all non-white participants, which in PSID consists mostly of African-Americans and the referent group was non-Hispanic white. Lastly health insurance status was ascertained as a dichotomous variable indicating whether or not someone in the family had insurance.

Statistical analysis

Both absolute and relative measures of effect were used to estimate the adjusted associations between wealth and the three health outcomes. Binomial marginal structural models (MSM) yielded adjusted risk ratios directly from exponentiated regression coefficients. Risk differences were calculated by taking the differences between predicted probabilities estimated from logistic regression models. Variances for the differences were estimated by the delta method (63) via the marginal effects post-estimation procedures available in Stata version 10 (59). Risk differences were taken holding all covariates at their mean values, which corresponds to standardization of the effect estimates to the covariate distribution in the total study population. The final binomial MSM is shown below:

$$\ln(Pr(Y|a)) = \beta_0 + \beta_1(a) + \beta_2 V$$

MSM are an effective tool for analyzing data in the face of time-varying confounding. The major time-varying confounders of interest in this study were income, marital status and insurance status. The main exposure, wealth, was also time-varying. Sex, race, age, education, self-reported general health status and region were ascertained at baseline and treated as time-invariant. Baseline values for all time-varying confounders were also included in the final MSM. In the event that the binomial model did not converge, we used starting values from Poisson regression (64).

Since data for both heads of household and their partners was available from PSID, there was clustering by household. Furthermore, given the longitudinal nature of the analysis

data were formatted vertically (there was one observation per time point per person), resulting in additional clustering by individual. Using the highest level of clustering (the household) produces results that control for both household and individual clustering (55, 56). Therefore, a family id variable was used to indicate the unit of clustering and to obtain robust standard errors.

In creating the analytic dataset for this study, individuals who were obese, smokers or hypertensive at baseline were excluded, allowing for the analysis of incident cases. In the smoking analysis, new smokers are either those who resumed smoking after quitting or those who initiated for the first time. Given the age of this population, most new smokers resumed smoking rather than initiated. Participants were allowed to enter the study at any point in time (between 1999 and 2003) as long as they participated for more than one year of data collection (i.e. each individual had to have at least two waves of data).

Calculating inverse probability weights

Inverse probability weights (IPW) are a key feature of MSM. Time-varying confounding is controlled through the use of these weights (51, 52). IPW were estimated from predicted probabilities obtained from logistic and multinomial models. Logistic models were used to obtain censoring weights, where the outcome of interest was whether or not the individual was lost to follow-up at that time point (those who were lost to follow up were coded as one and zero otherwise). The formula for the censoring weight is shown below:

$$sw_i^{\dagger} = \prod_{k=0}^{t} \frac{pr\ (C(k) = 0 | \bar{C}(k-1) = 0, \bar{A}(k-1) = \bar{a}_i(k-1), V = v_i)}{pr\ (C(k) = 0 | \bar{C}(k-1) = 0, \bar{A}(k-1) = \bar{a}_i(k-1), \bar{L}(k-1) = \bar{l}_i(k-1)}$$

Multinomial (i.e. proportional odds) models were used to obtain treatment weights, where the six category wealth variable was the outcome. The formula for the treatment weight is shown below:

$$sw_i(t) = \prod_{k=0}^{t} \frac{pr\,(A(k) = a_i(k) | \bar{A}(k-1) = \bar{a}_i(k-1), V = v_i)}{(A(k) = a_i(k) | \bar{A}(k-1) = \bar{a}_i(k-1), \bar{L}(k) = \bar{l}_i(k-1))}$$

Multiplying the treatment and censoring weights resulted in the final IPW. Continuous variables, income and age (centered on the mean), were entered into the weighting model flexibly: as linear terms, squared terms and quadratic splines. The numerator of the weight contains all covariates measured at baseline (age, race, sex, education, region, general health status, marital status, income and health insurance status) as well as baseline wealth while the denominator contains baseline and time-varying confounders (income, marital and health insurance status). By including baseline covariates in the numerator of the weight, the IPW were stabilized thus producing smaller variances for the final estimates (52).

We tested several weighting models. Continuous variables were first tried as linear terms and then as higher order and spline terms. Significant interaction terms were also included in weighting models. Modeling continuous variables more flexibly (as higher order terms and splines) and excluding interaction terms proved to be the best weighting model. This decision was based on the distribution of the IPW, where the mean at each time point is close to one and the range is small. (51, 53). In addition, time-varying covariates were entered into the weighting model as lagged covariates (at time t – 1), this made little difference to the final parameter estimates, but negatively impacted the sample size.

Very large values of the IPW or means far from one indicated a possible violation of the positivity assumption or a misspecified weighting model. One strategy to deal with

extreme weights was to truncate or trim the weights (53). In our study, two percent of the IPW were trimmed at each end. This resulted in weights with a mean close to one and a narrower range (see Figure 2).

Results

The distribution of wealth was highly skewed in our study (as it is in the US). This was indicated by a large difference between the median and the mean. After adjustment for inflation, median wealth at baseline was approximately $35,200 but mean wealth was close to $185,000. At the 25th percentile, wealth was about $3700 and at the 75th percentile it was $138,700. At baseline, 11.5% of persons had negative wealth and 5.3% reported zero wealth. After excluding baseline cases we were left with a sample size of 10475 in the obesity analysis, 10110 in the smoking analysis and 10744 in the hypertension analysis.

Table 7 provides demographic characteristics for the study population at baseline prior to excluding incident cases (n=13031). Recall that these data were not weighted by the PSID survey weights. Wealth is measured at the household level, thus is the same for each member of the household. The description of wealth by various individual level demographic characteristics may be driven by the marital status. In an attempt to better understand how wealth is distributed by individual demographic characteristics, an alternative to Table 7 is presented in Table 8. The total number of unique households in the 1999-2005 PSID sample were 10,042.

The percentage of women in the poorest wealth category was larger compared to men (59.2% vs 40.8%). Among the wealthiest quintile, however, the percentage of males and females were similar (48.5% vs. 51.5%). As for race, in the wealthiest quintile there were eight times as many white respondents compared to non-whites; while in the poorest wealth

category there were slightly more non-whites (55.5%) than whites (44.5%). Education, age and income all showed the expected pattern. Older, more educated and higher income individuals all had much more wealth than their younger, less educated and lower income counterparts. For these three characteristics there was a steady and significant trend. In terms of marital status, in the highest quintile of wealth there were about 21 times more married persons compared to the never married and 8.5 times more married compared to widowed, divorced or separated persons. As far as insurance coverage reflects employment, another SES marker, the data revealed far fewer uninsured individuals among the wealthy; the percent uninsured in the least wealthy group is nine times the percent in the wealthiest group. All bivariate associations presented in Table 7 had $p < 0.0001$.

As for the incidence of CVD risk factors, obesity and smoking declined as wealth increased, the incidence of obesity was almost 2.5 times higher and the incidence of smoking 5 times higher for those in the poorest wealth category compared to those in the wealthiest. Hypertension generally increased with wealth. The increase in the incidence of hypertension reflects the increase in hypertension that occurs with age.

Figure 3 shows risk differences and 95% CI for the effect of wealth on obesity, smoking and hypertension. Risk difference results are expressed as the number of excess cases of the outcome attributable to low wealth relative to wealth quintile five from 1999 to 2005 per 1000 persons. Figure 3 is presented in tabular form in Table 10. The model for obesity produced estimates with the highest magnitude compared to the other two outcomes. After adjusting for baseline and time-varying confounders, the number of excess cases of obesity were 22 (CI: 13, 31) for the less than or equal to zero wealth group, 25 (CI: 15, 35)

for wealth quintile one, 14 (CI: 6, 21) for quintile two, 14 (CI: 7, 21) for quintile three and 11(CI: 4, 17) for quintile four.

The estimates from the smoking model had the best precision compared to estimates from the other two models. The number of excess smokers was 10 (CI: 5, 16) for those in the less than zero wealth group, six (CI: 1,11) for the first quintile, five (CI: 0.2, 9) for the second quintile, four (CI: -0.5, 8) for the third quintile and three (CI: -1, 7) for the highest wealth group, the fourth quintile. Compared to the other models the hypertension model revealed a similar (and smaller) number of excess cases regardless of wealth quintile. The number of excess hypertensives were four (CI: -4, 12) for the least wealthy group, six for the first and second wealth quintiles (CI: -2, 14 and -1, 14 respectively), four for the third and fourth quintile (CI: -2, 11 and -2, 10 respectively)

Risk ratios and 95% confidence intervals (CI) for the effect of wealth on the three CVD risk factors are shown in Table 9. In the fully adjusted model, the risk of becoming obese was inversely related to wealth; as wealth increased the risk of obesity declined. Those with less than or equal to zero wealth and those with little wealth (quintile one) had a similar and significantly higher risk of obesity relative to the wealthiest group (quintile five), 1.80 (CI: 1.43, 2.26) and 1.89 (CI: 1.49, 2.40) respectively. Those in quintiles two and three had about a 51% (CI: 1.21, 1.88 and 1.23, 1.86 respectively) increased risk and those in quintile four a 40% (CI: 1.14, 1.71) increased risk of becoming obese compared to the wealthiest group. The confidence limit ratios suggested these estimates were relatively precise compared to estimates from other models.

The smoking model suggested a stronger magnitude of effect for the poorest wealth group. The least wealthy had the highest risk of becoming smokers (2.10, CI: 1.41, 3.12).

Those in low wealth quintiles one and two had an increased risk of becoming smokers, with RR of 1.61 (CI: 1.07, 2.44) and 1.50 (CI: 1.01, 2.21) respectively. The risk ratio for those in quintiles three was 1.39 (CI: 0.95, 2.04) and the lowest risk was among quintile four (1.30 CI: 0.91, 1.87).

The effect estimates for hypertension were more precise but of a smaller magnitude than the estimates from the other two outcomes. Among the least wealthy the model suggested that the risk of hypertension was 10% (CI: 0.90, 1.35) higher compared to the wealthiest quintile. Those in quintile one and two had about a 16% (CI for quintile one: 0.95, 1.42 and CI for quintile two: 0.97, 1.41) increased risk of developing hypertension relative to the wealthiest group, while those in quintiles three and four had a 11% increased risk of hypertension (CI: 0.93, 1.33 and 0.95, 1.29 respectively).

In addition, models were stratified by race and sex. Results are not shown here, but are available from the authors. For obesity, estimates for whites and women were of the largest magnitude while those for men were moderate and blacks showed small effects. In the smoking models, women showed the strongest effects, followed by whites, males and finally blacks. And for hypertension, females and blacks had moderate sized effects while men and whites had many estimates close to null. We also attempted to stratify by age, but we did not have sufficient power to use a meaningful cutoff, specifically looking at the retired versus the working age population.

In order to assess the impact of time-varying confounding we compared results from the MSM to traditional models controlling for time-varying confounders. Traditional binomial models were similar in magnitude and direction to binomial MSM (see

supplemental table 1). For each estimate the confidence intervals for the traditional estimate overlapped with that of its MSM counterpart.

Discussion

This study found a strong association between wealth and obesity incidence, a moderate association between wealth and smoking initiation and a weak association between wealth and hypertension incidence. This was true on both the absolute and relative scales, where both the risk ratios and risk differences revealed similar overall patterns. Given the importance of cardiovascular disease to the US population, a closer examination of these CVD risk factors was warranted.

Other studies that examined CVD risk factors and wealth reported similar findings. Several studies on obesity found a negative association with wealth, specifically among women and whites (7, 20, 34, 35). A German study which compared those with excess debt to those without debt found those in debt to have higher odds of obesity (23). A cross-sectional study found no association between obesity and wealth; however wealth was crudely measured (24). It should be noted that several of these studies were interested in the equally important question of whether poor health results in a reduction of wealth, by using wealth as the outcome and BMI as the main exposure of interest (20, 34, 35). The studies by Zagorsky conducted longitudinal analyses thus implying that obesity causes a reduction in wealth (34, 35).

The literature on smoking and wealth consisted mostly of cross-sectional studies of European or Australian populations (22, 26, 28) with one study of a US population (16). All the cross sectional studies consistently found that the least wealthy had a higher prevalence

of smoking (16, 22, 26, 28). One longitudinal study interested in reverse causality found that smokers had several thousand dollars less wealth compared to non-smokers (33).

Several cross-sectional studies that looked at hypertension and wealth found stronger more significant inverse associations compared to our study (7, 19, 21, 27), while another found very similar results to ours (25). Similarly, a longitudinal analysis of an elderly population in the US showed no causal link between low wealth and increased incidence of hypertension (36). It should also be noted that studies using other measures of SES such as income and education have also found a weak but positive association with hypertension (65, 66).

Our study had limitations. First, the use of self-reported data for some of the outcomes was not ideal. Specifically self-reported height and weight is known to underestimate the true prevalence of obesity, especially among overweight women (67). Concerns of misclassification bias arise when classification of the outcome depends on the exposure. The less wealthy were more likely to be overweight than the wealthy, and thus were more likely to misreport their weight leading to the potential for differential misclassification. Because the less wealthy were more likely to be misclassified in their obesity status, our data showed fewer obese cases among the poor. Thus the expected direction of the misclassification for the obesity-wealth relationship in our study was towards the null.

Hypertension was most likely underreported among younger respondents who have yet to be diagnosed with hypertension, but accurately reported among older individuals who were involved in the ongoing management of the condition (68). Several studies have found that women, older and more educated individuals have less misclassification for self-reported

hypertension than other groups (68-71). Given the relationship between wealth, age, education and health insurance status (the least wealthy tend to be younger, less educated and uninsured) we may have differential misclassification of hypertension. In light of this misclassification bias, our data would reveal fewer hypertensives among the less wealthy, thus potentially explaining the attenuated results we see for this outcome.

Misclassification of smoking status is less of a problem than for obesity or hypertension. In general researchers believe that self-reports are good indicators of actual smoking status (72-74) because they allow us to understand the duration and severity of smoking, which biological markers (such as cotinine) do not.

In order to reduce measurement error around wealth, we also attempted additional sensitivity analysis adjusting for household size; there was little difference in the final parameter estimates comparing models that did and did not adjust for this covariate.

The exclusion of baseline cases from the analysis raises questions about selection bias, another potential source of bias in our study. Those with preexisting hypertension are more likely to be older and of lower SES. By excluding them, the effect estimate will likely be biased downward. Similarly for obesity, the exclusion of baseline cases excludes more low SES individuals which may result in underestimates of the effect. That is, a sample of healthier, wealthier individuals will create an underestimate of the effect estimate for obesity and hypertension (75).

With smoking however, the picture was more complex because smoking status was a composite of a few different outcomes, each with the probability of being affected by wealth differentially. We conducted a sensitivity analysis for smoking where three separate outcomes were created for three separate subpopulations (see Table 11). Among the non-

smokers, we looked at the probability of smoking initiation. Among the current smokers we looked at the probability of quitting and among the former smokers, we looked at the probability of resuming smoking. Each of these outcomes was fit using a logistic MSM (because outcomes were rare). Results from these models all went in the expected direction. Although the number of persons who initiated smoking was small, there was an elevated odds of smoking initiation among the least wealthy individuals, which declined as wealth increased. The smoking cessation model showed that the least wealthy were less likely to quit smoking, while the wealthier quintiles were more likely to do so. Lastly, in the smoking resumption model, the least wealthy were more likely to resume smoking while the wealthier quintiles were more likely to remain non-smokers. The results of these models provided some assurance that selection bias was not severely affecting the estimates in the overall smoking model. It should be noted that in addition to modeling incidence, we also modeled prevalence for each of the outcomes. Results were similar, however, incidence models produces estimates of larger magnitude.

There were several strengths to our study. First, by using data from the PSID, we were assured that wealth was measured in a rigorous and comprehensive fashion (61). As discussed earlier many epidemiologic studies measured wealth poorly, thus the estimates based on these studies were fraught with measurement error. In addition, the longitudinal nature of the PSID allowed us to explore the question of wealth and health over a 6 year period. Most studies of wealth and health to date have used cross sectional data. A related advantage was our use of marginal structural models as the analytic technique to answer the study question. After satisfying several important assumptions, such as the absence of unmeasured confounding, MSM can have a causal interpretation even in the presence of

time-varying confounding. Although some of the assumptions of MSM are difficult to meet, we believe this study produced improved estimates relative to past studies (76). Ideally a randomized controlled trial which randomly assigns wealth to each individual at baseline and waits for the development of one of the CVD risk factors would yield an estimate with a causal interpretation. Since wealth is not easily randomizable, the consistency assumption is suspect; therefore, the true causal estimates of the relationship between wealth and health may not equal the estimates presented in our study, regardless of our improved methodology.

As noted earlier, the results we obtained from MSM were similar to those from traditional models (see Table 17 in the appendix). This indicates that there was not a substantial amount of time-varying confounding in our study. We hypothesized that income would be the strongest time-varying confounder. However, given our study's short time interval, it is likely that income trajectories remained relatively stable during this period; thus it is understandable that income was not a strong time-varying confounder in these data.

There are many potential mechanisms through which low wealth results in poor health outcomes. Poor physical and social environments can encourage health damaging exposures (77). For example, the lack of economic resources associated with having little wealth may limit an individual's access to health care, quality housing and nutritious foods among other things. A lack of economic and social resources can also result in insufficient investment in "human, physical, social and health infrastructure" which may be detrimental to the health of populations (78). Assuming that less wealthy individuals live in less wealthy communities, insufficient infrastructure may take the form of fewer or less convenient public transportation options, a lack of parks and unsafe streets, and more liquor and convenient stores. These deficiencies can then lead to increased isolation, a more sedentary lifestyle and

poor diets, which directly result in higher rates of smoking and obesity among less wealthy individuals (79, 80). Other research has found that low SES individuals may have less social support, higher job strain and less job control. All these factors have been associated with higher rates of smoking and obesity (81).

Chronic stress may underlie many of these health damaging exposures (77). Since wealth is a stockpile of financial resources a lack of wealth (which translates into the absence of a safety net) is ostensibly a cause for long-term financial stress. It has been hypothesized that chronic stress and other psychosocial factors trigger a series of biological events, through central nervous system activation of autonomic, neuroendocrine and immune responses (82). These biological pathways may be especially germane to hypertension and other cardiovascular functions (83). The choice of CVD risk factors as outcomes for this study was further underscored in light of the potential mechanisms discussed above.

Although wealth may be more difficult to measure than other SES variables, both its empirical performance and its theoretical relevance make it an important factor that should be considered by more health researchers. In addition, asset building programs focusing on the poor and middle class have shown modest success in helping families build wealth (84). Thus, not only is wealth a useful empirical and theoretical construct, it is also amenable to policy interventions that could have long term benefits for improving the health of the poor.

Table 7. Unweighted demographic characteristics of study population at baseline, PSID 1999- 2005

	<= zero wealth	Quintile 1	Quintile2	Quintile3	Quintile 4	Quintile 5
Sex						
Male	40.8	44.2	46.7	47.3	47.1	48.5
Female	59.2	55.9	53.4	52.8	52.9	51.5
Race						
White	44.5	44	54.7	60	73.3	89.0
Non-white	55.5	56	45.3	40	26.7	11.0
Education						
Less than high school	22.7	25.3	16.5	15.0	10.2	5.7
High school graduate	37.9	44.2	42.0	41.1	39.8	29.9
Greater than high school	39.4	30.6	41.5	43.9	50.0	64.3
Marital status						
Never married	42.1	32.5	20.3	11.1	7.3	3.7
Married	37.5	44.5	62.7	72.5	76.6	85.9

Widowed, divorced or separated	20.4	23.1	17.1	16.5	16.1	10.4
No health insurance	27.1	26.5	17.1	11.0	5.8	3.0
Mean age in years (sd)	33.7 (12.6)	34.6 (13.4)	36.6 (12.4)	41.3 (13.7)	46.8 (14.7)	52.0 (14.0)
Mean income in dollars (sd)	29,961 (24,609)	30,428 (26,542)	43,860 (24,488)	52,798 (31,547)	67,362 (52,717)	108,832 (120,000)
Median wealth in dollars (25%, 75%)	-3039 (-12,000, 0)	3189 (1488, 5528)	18,288 (12,756, 24,981)	50,494 (40,448, 62,718)	120,866 (95,916, 153,076)	396,508 (270,008, 730,298)
Incidence of health outcomes (per 100 person years)						
Obesity	9.1	9.4	7.9	7.6	5.9	3.8
Smoking	5.7	4.2	3.4	2.6	2.0	1.1
Hypertension	6.5	6.8	7.0	7.3	7.9	7.4

Results are expressed in percentages unless otherwise indicated.

Sample size for Table 1 is 13,031.

All bivariate analysis had $p < 0.0001$. P-values are from chi-squared test for sex, race, education, marital status, health insurance and from ANOVA for age, income and wealth.

Table 8. Descriptive characteristics at baseline by household size, PSID 1999-2005

	≤ 0 wealth	Quintile 1	Quintile 2	Quintile 3	Quintile 4	Quintile 5
Sex						
1 person household						
1 Male	26.6%	25.0%	18.6%	12.1%	10.2%	7.6%
1 Female	29.7%	24.0%	16.4%	11.7%	10.8%	7.3%
2 person household						
1M, 1F	11.4%	13.1%	16.3%	16.8%	19.3%	23.1%
2M or 2F	42.9%	28.6%	14.3%	7.1%	7.1%	0.0%
Race						
1 person household						
1 White	20.4%	20.6%	18.0%	13.7%	14.6%	12.7%
1 Non-white	36.7%	28.2%	16.3%	10.1%	6.6%	2.1%
2 person household						
2 Whites	9.0%	8.7%	13.5%	15.4%	21.6%	31.9%
2 Non-whites	15.3%	21.6%	21.8%	20.3%	14.4%	6.5%
2, mixed	14.1%	14.1%	17.9%	14.5%	20.7%	18.6%
Education						
1 person household						
1 adult, less than high school	40.0%	30.4%	11.3%	9.2%	6.0%	3.0%
1 adult, high school	27.5%	28.1%	17.0%	11.6%	10.0%	5.8%

degree						
1 adult, greater than high	25.3%	19.3%	19.2%	12.8%	13.0%	10.4%
2 person household						
2 adults, less than high school	13.9%	27.8%	22.5%	16.8%	12.7%	6.3%
2 adults, high school degree	11.7%	14.5%	18.7%	18.6%	20.0%	16.5%
2 adults, greater than high school	10.3%	5.9%	13.0%	14.6%	19.6%	36.5%
2 adults, mixed	10.7%	14.7%	16.1%	17.6%	19.9%	21.0%
Marital status						
1 person household						
1 adult married, but spouse is absent	20.2%	17.5%	21.0%	15.9%	14.0%	11.5%
1 adult, widowed/divorced/sep	20.7%	21.4%	17.0%	15.2%	15.2%	10.6%
1 adult, never married	37.4%	29.1%	16.3%	7.9%	5.7%	3.6%
2 person household						
2 adults, married	9.4%	10.9%	16.1%	17.5%	20.5%	25.6%
2 adults, not married	23.2%	25.5%	17.5%	12.9%	12.1%	8.8%
Wealth (median $)						
1 person	-1,500	2,888	18,339	52,620	123,275	381,626

	2 people	-15,847	3,842	19,348	52,181	126,701	818,168
Income (mean $)							
	1 person	23,022	24,733	39,219	44,573	53,550	58,000
	2 people	38,819	37,003	46,633	56,203	71,421	113,046
Age (mean)							
	1 person	33	34	37	43	49	52
	2 people*	35	35	38	42	48	54

*age of oldest person

Figure 3. Cardiovascular disease risk factors by wealth quintile, risk differences and 95% confidence intervals, PSID 1999-2005

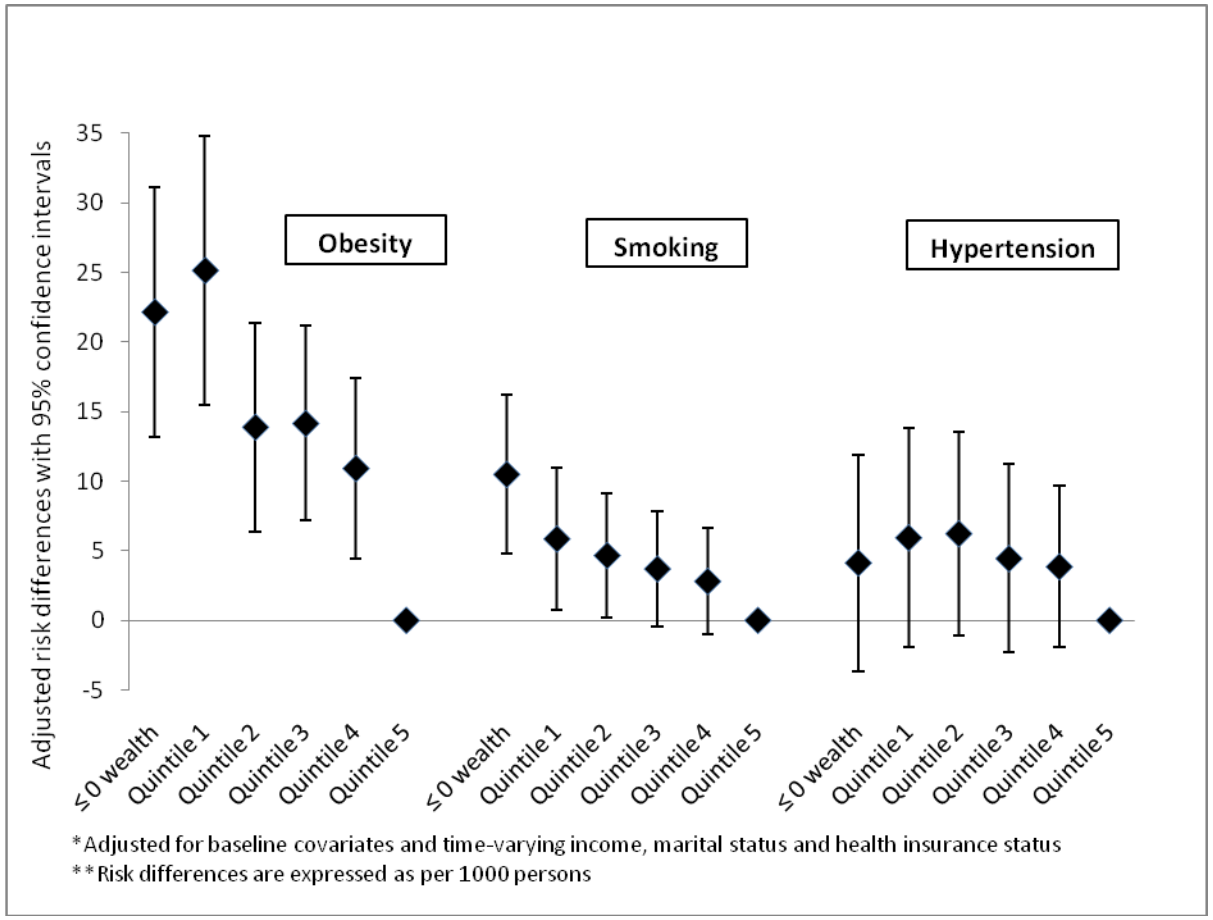


Table 9. Risk ratios and 95% confidence intervals of the association between wealth and obesity incidence, smoking initiation and hypertension incidence, PSID 1999 - 2005

	Obesity			**Smoking**			**Hypertension**		
	risk ratio	**Lower 95% CI**	**Upper 95% CI**	**risk ratio**	**Lower 95% CI**	**Upper 95% CI**	**risk ratio**	**Lower 95% CI**	**Upper 95% CI**
≤ zero wealth	1.80	1.43	2.26	2.10	1.41	3.12	1.10	0.90	1.35
Quintile 1	1.89	1.49	2.40	1.61	1.07	2.44	1.16	0.95	1.42
Quintile 2	1.51	1.21	1.88	1.50	1.01	2.21	1.17	0.97	1.41
Quintile 3	1.52	1.23	1.86	1.39	0.95	2.04	1.11	0.93	1.33
Quintile 4	1.40	1.14	1.71	1.30	0.91	1.87	1.11	0.95	1.29
Quintile 5	Referent			Referent			Referent		

*All models adjusted for baseline covariates (sex, age, race, education, region, general health status, marital status, income and insurance status) and time-varying covariates (income, marital status and insurance status) through the IPW.

Table 10. Risk differences and 95% confidence intervals of the association between wealth and obesity incidence, smoking initiation and hypertension incidence expressed as cases per 1000 persons over 6 year follow up, PSID 1999 - 2005

	Obesity			**Smoking**			**Hypertension**		
	risk difference	**Lower 95% CI**	**Upper 95% CI**	**risk difference**	**Lower 95% CI**	**Upper 95% CI**	**risk difference**	**Lower 95% CI**	**Upper 95% CI**
≤ zero wealth	22	13	31	10	5	16	4	-4	12
Quintile 1	25	15	35	6	1	11	6	-2	14
Quintile 2	14	6	21	5	0.2	9	6	-1	14
Quintile 3	14	7	21	4	-0.5	8	4	-2	11
Quintile 4	11	4	17	3	-1	7	4	-2	10
Quintile 5	Referent			Referent			Referent		

*All models adjusted for baseline covariates (sex, age, race, education, region, general health status, marital status, income and insurance status) and time-varying covariates (income, marital status and insurance status) through the IPW.

Table 11. Risk ratios and 95% confidence intervals of the association between wealth and the initiation, initial cessation and resumption of smoking, PSID 1999 – 2005

	Initiation		**Cessation**		**Resumption**	
	risk ratio	**95% Confidence Interval**	**risk ratio**	**95% Confidence Interval**	**risk ratio**	**95% Confidence Interval**
≤ zero wealth	2.17	0.84, 5.60	0.78	0.57, 1.06	1.88	1.17, 3.02
Quintile 1	1.79	0.67, 4.82	0.82	0.60, 1.11	1.39	0.84, 2.30
Quintile 2	1.46	0.58, 3.69	0.80	0.60, 1.08	1.41	0.89, 2.25
Quintile 3	1.31	0.52, 3.28	0.91	0.69, 1.20	1.41	0.89, 2.22
Quintile 4	1.22	0.51, 2.89	1.08	0.82, 1.41	1.16	0.75, 1.77
Quintile 5	Referent		Referent		Referent	

*All models adjusted for baseline covariates (sex, age, race, education, region, general health status, marital status, income and insurance status) and time-varying covariates (income, marital status and insurance status) through the IPW.

Chapter 6

Long-term effects of wealth on mortality and health status

Abstract

Background: The use of wealth as a measure of socioeconomic status remains uncommon in epidemiological studies. When used, wealth is often measured crudely and at a single point in time.

Objective: This study explores the relationship between wealth and 2 health outcomes, mortality and self-reported general health status (GHS) in a US population.

Methods: To improve upon the existing literature, this study uses a detailed and validated measure of wealth in a longitudinal setting. Data for this study are from 7 waves of the Panel Survey on Income Dynamics collected between 1984 and 2005. Inverse probability weights were employed to control for time-varying confounding and to estimate both relative and absolute measures of effect. Binomial marginal structural models (MSM) were used to analyze general health status and logistic MSMs were used to investigate the wealth-mortality association. Wealth was defined as inflation-adjusted net worth and specified as a 6 category variable: 1 category for those with less than or equal to 0 wealth and quintiles of positive wealth.

Results: In the fully adjusted model, the incidence of poor health status was inversely related to wealth. There was a 17% to 54% higher risk of falling into poor health and 6 to 22 excess cases of poor health (per 1000 persons) among the less wealthy relative to the wealthiest quintile. Stratified models revealed that less wealthy men, women and whites also had

higher risk of poor health status relative to their wealthy counterparts. The overall wealth-mortality association revealed an 83% increased risk and 5 excess cases of death (per 1000) among the least wealthy compared to the wealthiest. Less wealthy women had the largest relative mortality risks (risk ratios between 2.29 and 1.37) and men with less than or equal to 0 wealth had the largest absolute mortality risk (7 excess cases) compared to the wealthiest quintile.

Conclusion: There is a strong inverse association between wealth and incidence of poor GHS and between wealth and mortality.

Introduction

The goal of this study was to explore the relationship between wealth, a measure of socioeconomic status (SES) not commonly used in the health literature, and 2 health outcomes: (1) self-reported general health status (GHS) and (2) mortality. Wealth is defined as the stockpile of resources amassed over the lifetime (4, 5). Income is defined as the flow of resources into the household and consumption as the flow of resources out of the household. Savings is the difference between income and consumption and is transformed into wealth (through the purchase of cars, houses or stocks and bonds or the reduction of debt). Thus, by definition, income and wealth are related but separate constructs.

Wealth is a more stable measure of SES (it is less subject to fluctuation than income) and it may better represent the true social hierarchy as it encompasses both economic circumstances and prestige or status (3). In addition, wealth may better measure SES during certain times in the life course (when income is lost due to unemployment or illness) and for certain populations (the elderly) (8). However, given the many components of wealth it is more difficult to measure than other measures of SES, thus it is used less often in health literature. Using data from the Panel Survey on Income Dynamics (PSID), a study with detailed wealth measurement and extensive follow-up, we evaluated the wealth-health relationship in a non-elderly adult US population.

Methods

PSID, a longitudinal study of the US population, began in 1968 and continues today. The use of survey weights when analyzing PSID data produces nationally representative estimates. However, survey weights were not used in the current analysis. Because we are interested in an etiologic hypothesis rather than population representative prevalence

estimates, we treat the PSID population as a cohort rather than a sample. Causal parameters estimated in our models therefore have in-sample interpretations and are not applicable to the broader US population. Much has been written about the design and content of the PSID elsewhere (62). Data for this study came from 7 waves of the PSID: 1984, 1989, 1994, 1999, 2001, 2003 and 2005. Health questions were asked only of the head of household and his/her partner.

Measures

The health outcomes examined in this study were GHS and mortality. The GHS variable was derived from the standard question "Would you say your health in general is excellent, very good, good, fair or poor?" In keeping with several other papers on wealth and health, it was dichotomized as excellent, very good and good versus fair and poor. At the onset of the study PSID relied on non-response, returned postal materials and information from surviving family members to identify deaths. PSID subsequently verified most deaths using the National Death Index. The mortality status of respondents who were lost to follow-up, however, remained unknown (85). The maximum follow-up time for mortality was 21 years (from 1984 to 2005).

Family wealth was defined as total net worth, which includes the value of one's primary home, farm or business assets, checking or savings accounts, vehicles, second homes, stocks and bonds. Debt was subtracted from these assets. This approach to measuring wealth included all major components and resulted in estimates that were consistent with other studies (61). Wealth was adjusted for inflation using the 2001 consumer price index and specified as a 6 category variable, where category 1 included all those that had negative or 0 wealth and categories 2 through 6 were quintiles of positive

wealth. In the final models, wealth was specified as 5 indicator terms (i.e. dummy variables) to avoid making any assumptions about linearity. The referent group was the highest wealth quintile. For stratified models, wealth categories were redefined for only the population of interest (e.g. the model for women defined wealth categories only among women).

Other covariates included in models for both outcomes were income, marital status, region of residence, education, age, race and sex. Income was specified as a continuous poverty-to-income ratio using the annual poverty thresholds from the Census Bureau and accounting for number of people in the household. Marital status was categorized as married (the referent group), never married or divorced, separated or widowed, and specified as indicator terms. The 3 indicator variables for region classified state of residence as northeast, midwest or south; west was the referent group. Education was specified as 2 indicator variables, (< high school and high school degree) and greater than high school was the referent group. Age was specified as a continuous centered variable for both outcomes. An indicator variable for race included all non-white participants, which in PSID consists mostly of African-Americans and the referent group was non-Hispanic white. The mortality model also included dichotomized GHS as an additional covariate.

Statistical analysis

Adjusted associations were estimated using both absolute and relative measures of effect for the total population and for men, women, whites and non-whites separately. For GHS, a binomial marginal structural model (MSM) yielded adjusted risk ratios directly. For mortality, a cumulative incidence model, fitted with a logistic MSM, was used to ascertain risk of death. Risk differences were calculated by taking the differences between predicted probabilities estimated from logistic regression models. The delta method was used to

estimate the variances for risk differences (63) via the marginal effects post-estimation procedures available in Stata version 10 (59). Risk differences were calculated holding all covariates at their mean values, which corresponds to standardization of the effect estimates to the covariate distribution in the total study population (86).

The major time-varying confounders of mortality were income, marital status and GHS; for GHS they were income and marital status. The main exposure, wealth, was also time-varying. Wealth categories were based on the distribution of wealth for each specific model; for example in the model for men, wealth quintiles reflected the distribution of wealth only among men. Sex, race, age, education and region were ascertained at baseline and treated as time-invariant. Baseline values for all time-varying confounders were also included in the final MSM. In GHS models, starting values from Poisson regression were used if the binomial model did not converge (64).

In households where both the head of household and partner were present, there was clustering by household. Furthermore, there was 1 observation per time point per person, resulting in additional clustering by individual. We adjusted the variances for clustering at the highest level of aggregation, which produced valid estimates by accounting for both sources of dependence (55, 56). Thus a family id variable was used to obtain robust standard errors.

For the GHS analysis, individuals who reported poor GHS at baseline were excluded. Participants were allowed to enter the study at any point in time (between 1984 and 2003) as long as they participated for more than 1 year of data collection.

Calculating inverse probability weights

Inverse probability weights (IPW) are a key feature of MSM. Through the use of these weights time-varying confounding is controlled (51, 52). IPW were estimated from predicted probabilities obtained from logistic and multinomial models. Logistic models were used to obtain censoring weights, where the outcome of interest was whether the individual was lost to follow-up at that time point. Multinomial (i.e. proportional odds) models were used to obtain treatment weights, where the 6 category wealth variable was the outcome. Multiplying the treatment and censoring weights resulted in the final IPW. In the weighting model, continuous variables, income and age (centered on the mean), were specified flexibly; age as a linear term, squared term and 2 quadratic splines and income as linear, squared and cubic terms. The numerator of the weight contains all baseline covariates while the denominator contains baseline and time-varying confounders. Several weighting models were tested and the best one was chosen depending on the distribution of the IPW. Desirable properties for the IPW are a mean close to 1 and a small range (51, 53).

Very large values of the IPW or means far from 1 indicated a possible violation of the positivity assumption or a misspecified weighting model. One strategy used to deal with extreme weights is to truncate or trim the weights (53). In our study, 2 percent of the IPW were trimmed at each end. This resulted in weights with a mean close to 1 and a much narrower range of values (see Figure 2).

Results

For the mortality analysis the sample size was 26,614. After excluding 2,419 participants with fair or poor health status at baseline, the sample size for the GHS analysis was 15,745. Overall there were 2,162 deaths (8.12%) and the prevalence of poor or fair

health status was 13.4% at baseline. Table 12. Unweighted demographic characteristics of the study population at baseline, PSID 1984 – 2005Table 12 provides demographic characteristics by quintiles of wealth for the study population at baseline prior to excluding incident GHS cases (n=18,164). All members of the household share the same level of wealth, but some demographic characteristics reflect individual level attributes.

After adjustment for inflation, median wealth at baseline was approximately $20,500 but mean wealth was close to $109,100; indicating the highly skewed nature of household wealth. The percentage in the poorest wealth category was higher for women compared to men (about 60% vs 40%). As wealth increased, however, the male/female gap declined and was nearly eliminated at the highest quintile of wealth. In the wealthiest quintile there were almost 7 times as many white respondents compared to non-whites; while in the poorest wealth category the gap was reduced to 1.5 times as many non-white respondents. Education, age and income all showed the expected pattern. Older, more educated and higher income individuals all had much more wealth than their younger, less educated and lower income counterparts. For these 3 characteristics there was a steady trend (p for trend: <0.0001). Marital status is a known predictor of wealth; in the highest quintile of wealth there were about 19 times more married persons compared to the never married and 10 times more married compared to widowed, divorced or separated persons. The percent of persons reporting poor GHS declined as wealth increased, but the percentage of deaths increased with greater wealth. In this crude analysis, the increasing deaths were likely a result of increasing age. All bivariate associations presented in Table 12 were significant at the 0.0001 level.

There was a strong association between GHS and wealth on both the relative and absolute scales. In the fully adjusted models, the risk of developing poor GHS was inversely

related to wealth; as wealth increased the risk of poor GHS declined. In the risk ratio model (depicted in Figure 4), those with less than or equal to 0 wealth had a significantly higher risk of poor health relative to the wealthiest group (quintile 5), 1.54 (95% confidence interval (CI): 1.27, 1.88) and those in quintile 1 had a 41% (CI: 1.18, 1.69) increased risk of poor health. Those in quintiles 2 through 4 also had a significantly elevated risk of poor health, but of a lower magnitude than the lower wealth groups (1.21, 1.25 and 1.17 respectively).

Figure 5 shows risk differences expressed as the number of excess cases of poor perceived health attributable to having low wealth (≤ 0 wealth) instead of high wealth (quintile 5) from 1984 to 2005 per 1,000 persons. After adjusting for covariates, among those with less than or equal to 0 wealth, there were 22 (CI: 13, 32) excess cases between 1984 and 2005. In quintile 1, there were 16 (CI: 8, 24) and in quintile 2 there were 8 (CI: 1, 16) excess cases. Quintiles 3 and 4 had 9 and 6 excess cases of poor health, respectively. Tabular data for Figure 4 and Figure 5 is presented in Table 15. Stratified models for GHS revealed a similar pattern (Table 13 and Table 14). Among men and whites the least wealthy had the highest risk of poor health on the risk ratio and risk difference scales. Among women those with less than or equal to 0 wealth had a similarly elevated risk of poor health as those in quintile 1 (1.56 and 1.62 respectively). In general, for men, women and whites the risk of poor health declined as wealth increased. The binomial model for non-whites, however, showed a near null association for the poorest wealth quintiles and a protective effect for the wealthier quintiles. The results for non-whites on the absolute scale revealed highly imprecise estimates compared to the other stratified models.

In the mortality analysis, both the absolute and relative models revealed an increased risk of death among less wealthy groups (Figure 6, Figure 7 and Table 16). In the total

population, those with less than or equal to 0 wealth had an 83% higher risk of death compared to the wealthiest group (CI: 1.44, 2.31). Low wealth quintiles 1 and 2 had a similarly elevated risk of death (40% and 41% respectively), while the risk of death fell for those in quintiles 3 and 4 (risk ratio 1.35 and 1.25 respectively). On the absolute scale, this represents an excess of 5 deaths among the least wealthy, an excess of 2 deaths among quintiles 1, 3 and 4 and an excess of 3 deaths among those in quintile 2.

In the stratified mortality analysis, the relative measures for women revealed the strongest magnitude compared to the other subgroups (Table 13). Women in the least wealthy group had an increased risk of 129% relative to the wealthiest women, while women in quintile 4 still had a 37% increased risk of death. The models for men, whites and blacks revealed an increased risk of death among the lowest wealth quintile (1.61, 1.87 and 1.69 respectively), with a declining risk among higher wealth quintiles. There was an excess of 7 deaths among men, 6 among blacks, 5 among women and 4 among whites in the least wealthy group (CI: 2,11; 2,10; 2,7 and 1,7 respectively). Several of the higher wealth quintiles in the models for men and blacks contained the null value (Table 14).

Discussion

Our study found a strong inverse association between wealth and GHS and wealth and mortality. Stratified GHS models revealed a similarly strong association for men, women and whites, while stratified mortality models suggested strong relative effects among women and whites.

Several previous studies on wealth and mortality have found similar results. Several US studies that used a well-measured wealth variable found a positive association between wealth and mortality (10, 43-47). There are, however, 2 US studies that did not find an

association between wealth and mortality. The study by Feinglass et al concluded the lack of association between wealth and mortality after 10 years of follow-up was a result of controlling for baseline health status, an important mediator in the wealth-mortality association (48). In addition, an econometric study tested specifically for the "absence of causal links from wealth to mortality" and found evidence to support the claim (36).

A potential source of bias in our mortality analysis was the relatively high level of attrition in PSID. Respondents who were lost to follow up were included in the analysis and censored at their last time point; however, their mortality status remained unknown. If there was differential mortality between the censored and the uncensored respondents, (censored individuals had higher mortality rates and were less wealthy) we would observe bias towards the null. That is our results may be underestimating the real mortality risk. PSID conducted an internal examination of the impact of attrition on mortality and found that the mortality status of 11% of a sample of persons lost to follow-up could not be determined and about 3% were actually deceased (85).

Initial mortality models included total time of study participation as an additional covariate. We found that this time variable was negatively associated with death; that is the longer one was in the study the less likely s/he was to die. This selection bias towards healthy individuals was sufficiently strong as to skew the results in the opposite direction, thus we excluded it from future analysis.

Our findings for GHS were also similar to that of several other studies (7, 16, 29-32, 38-41). Most studies to date, however, used cross-sectional data (7, 11, 16, 29-32). Two longitudinal studies found results similar to ours; low wealth was associated with poor health status (40, 41). Several econometric analyses of the wealth – health question, however,

found little support for the conclusion that low levels of wealth cause poor health (36, 38, 39, 42). In fact their results suggested the opposite, poor health causes declines in wealth (36, 38, 39). These studies, however, were interested in the short term effects of wealth on perceived health status (36, 38, 39, 42). A serious change in health status is likely to have an immediate effect on a household's financial well being. This is evidenced by the well known statistic that in 2007 over 60% of personal bankruptcies were caused by illness of a family member (87). Thus in the short term whether poor health causes reductions in family wealth is a critical question, however, this short term approach could explain the null results of the wealth to health hypothesis found in these studies (36, 42, 88).

By using longitudinal data with an extensive follow up (about 21 years) and excluding baseline cases of poor health status, the estimates presented in our study provide firm evidence for the hypothesis that low levels of wealth cause poor perceived health status in the long term. In light of the cumulative disadvantage hypothesis (which posits that inequalities in SES worsen over the life-course) understanding the long term effects of SES on health is important (89, 90). Other studies with long follow up times further corroborated our results (41).

Limitations of our study included the potential for selection bias caused by the exclusion of baseline cases in the GHS analysis. Those in poor health were more likely to be older and less wealthy, causing the effect estimates to be biased downwards. Thus, our results may have underestimated the true effect (75). As previously discussed, another limitation to our study was the misclassification of mortality for those lost to follow up. With any longitudinal study of considerable follow-up, the introduction of bias is unavoidable in the face of attrition.

We believed that adjusting for income was necessary in order to understand the true effect of wealth on these health outcomes. However, the close conceptual relationship between income and wealth may cause some concern about this decision. Empirically, there is a low correlation between income and wealth in the PSID, which has been noted in other studies as well (5). GHS models that did not include either baseline or time varying income revealed risk ratios of a substantially larger magnitude. As expected, income is attenuating the effect of wealth on GHS, thus at the very least our results provide conservative estimates of the wealth-health relationship.

There were several strengths to our study. First, the wealth data in PSID were measured in a rigorous and comprehensive fashion (61). Many epidemiologic studies measured wealth crudely, resulting in invalid estimates. Other researchers have been working on devising a novel set of questions to measure wealth effectively yet more succinctly specifically for health research looking at US populations (13). In addition, the longitudinal nature of the data (21 years of follow up) allowed us to explore the question of wealth and health from a life course perspective. Many studies of wealth and health to date have used cross sectional data and several that approach the question longitudinally have relatively short follow up times. A related advantage was our use of MSM as the analytic technique to answer the study question. After satisfying several important assumptions, MSM can have a causal interpretation even in the presence of time-varying confounding. The use of this analytic technique to obtain a "causal" estimate presumes a manipulable exposure and a realistic intervention regime (91). Since wealth distributions are partly determined by government policies such as tax laws, this assumption of manipulability is not wholly unreasonable.

Several mechanisms have been proposed for how low SES impacts health outcomes. The less wealthy may be more subject to poor physical and social environments which can encourage health damaging exposures (77). In addition, the lack of a safety net associated with having little or no wealth can cause chronic stress among the poor. It has been hypothesized that chronic stress and other psychosocial factors trigger a series of biological events, through central nervous system activation of autonomic, neuroendocrine and immune responses resulting in poor health (82).

Wealth has been underused in health research. In US populations, it has several advantages compared to other more commonly used measures of SES. Eliminating health disparities in SES will require a more complete understanding of the economic and social resources available to poor families; one that can hopefully be gained through the increase use of wealth in health research.

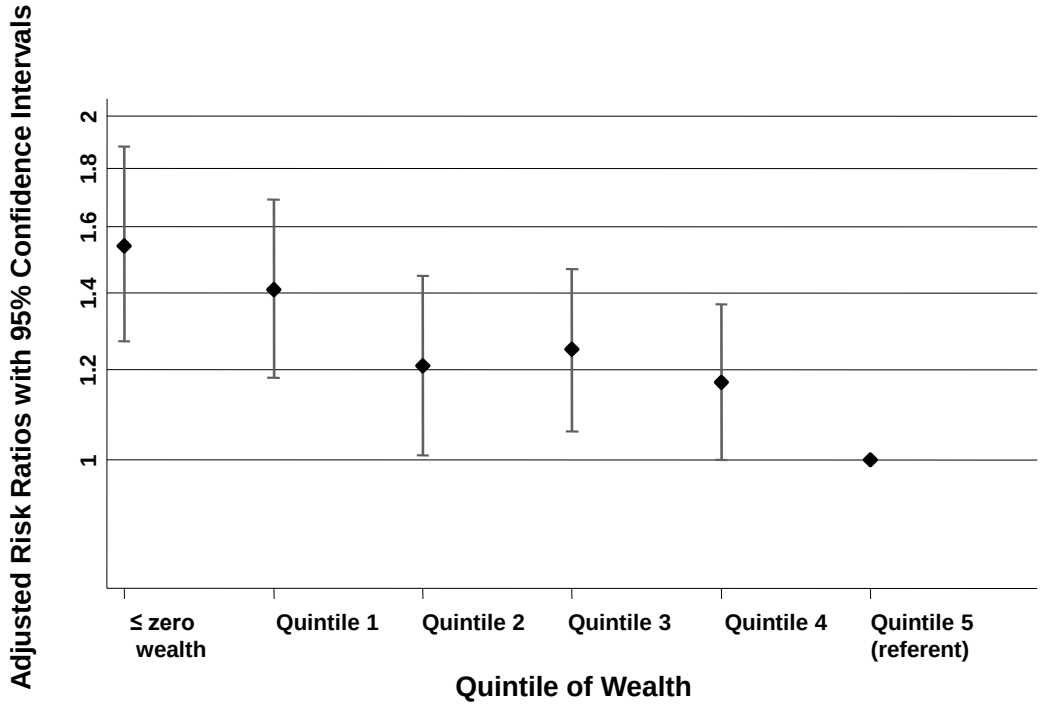
Table 12. Unweighted demographic characteristics of the study population at baseline, PSID 1984 – 2005

	<= 0 wealth	Quintile 1	Quintile2	Quintile3	Quintile 4	Quintile 5
Sex						
Male	40.1	45.9	47.7	47.4	47.8	48.3
Female	59.9	54.1	52.3	52.6	52.2	51.8
Race						
White	40.0	48.2	59.2	65.1	75.9	87.3
Non-white	60.0	51.8	40.8	34.9	24.1	12.7
Education						
Less than high school	30.3	27.9	19.2	21.9	16.2	12.4
High school graduate	36. 5	44.6	40.9	40.0	39.6	37.2
Greater than high school	33.3	27.5	40.0	38.1	44.3	50.4
Marital status						
Never married	43.3	31.5	20.6	11.7	7.6	4.6
Married	39.2	51.8	67.6	76.5	80.3	86.7
Widowed, divorced or	17.5	16.7	11.8	11.8	10.2	8.8

separated						
Mean age in years (sd)	30.7 (11.8)	31.0 (12.1)	32.6 (12.0)	36.5 (12.9)	41.3 (13.8)	47.7 (14.4)
Mean income in dollars (sd)	20,291 (20,421)	22,298 (19,216)	31,856 (20,271)	36,916 (24,439)	44,000 (31,584)	60,874 (66,912)
Median wealth in dollars (25%, 75%)	-7,517 (-5,700, 0)	3,640 (1,364, 5,634)	19,203 (12,787,25,095)	51,909 (40,909, 61,363)	116,306 (92,264, 136,207)	608,342 (218,179, 507,778)
Prevalence of health outcomes						
% poor health	18.9	14.1	11.3	12.1	11. 6	11.1
% of deaths	5.7	5.4	6.2	7.9	10.6	17.7

Results are expressed in percentages unless otherwise indicated.

Figure 4. Risk ratios and 95% confidence intervals for incidence of poor health status by wealth quintile, PSID 1984-2005



*Adjusted for baseline covariates and time-varying income and marital status

Figure 5. Risk differences and 95% confidence intervals for incidence of poor health status by wealth quintile, PSID 1984-2005

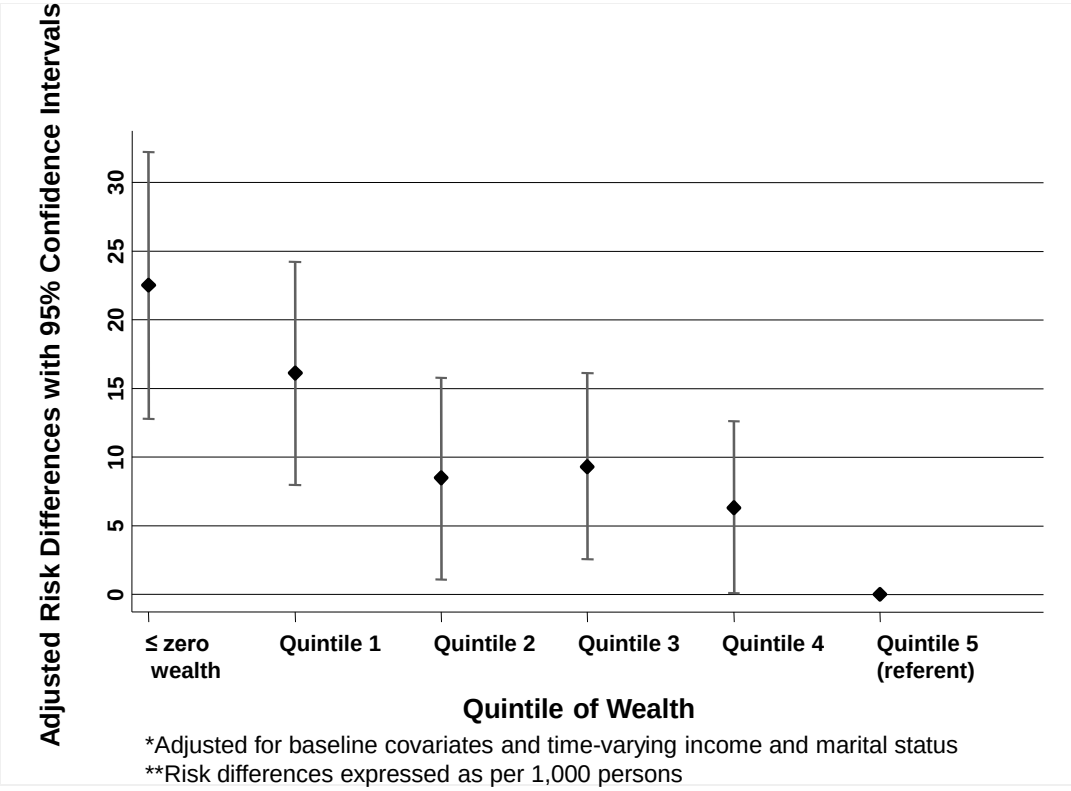
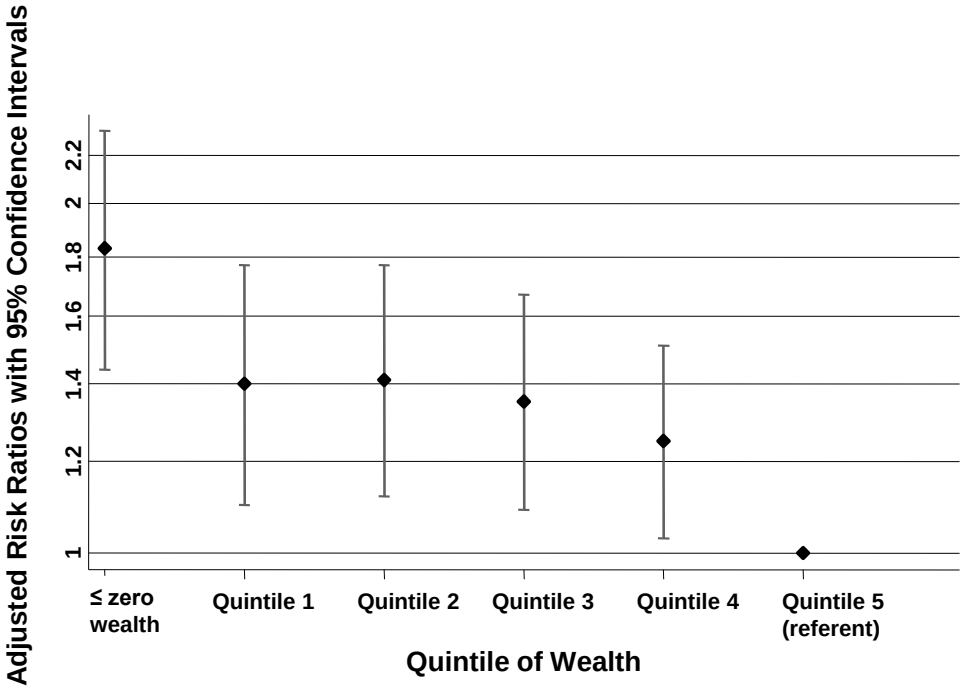
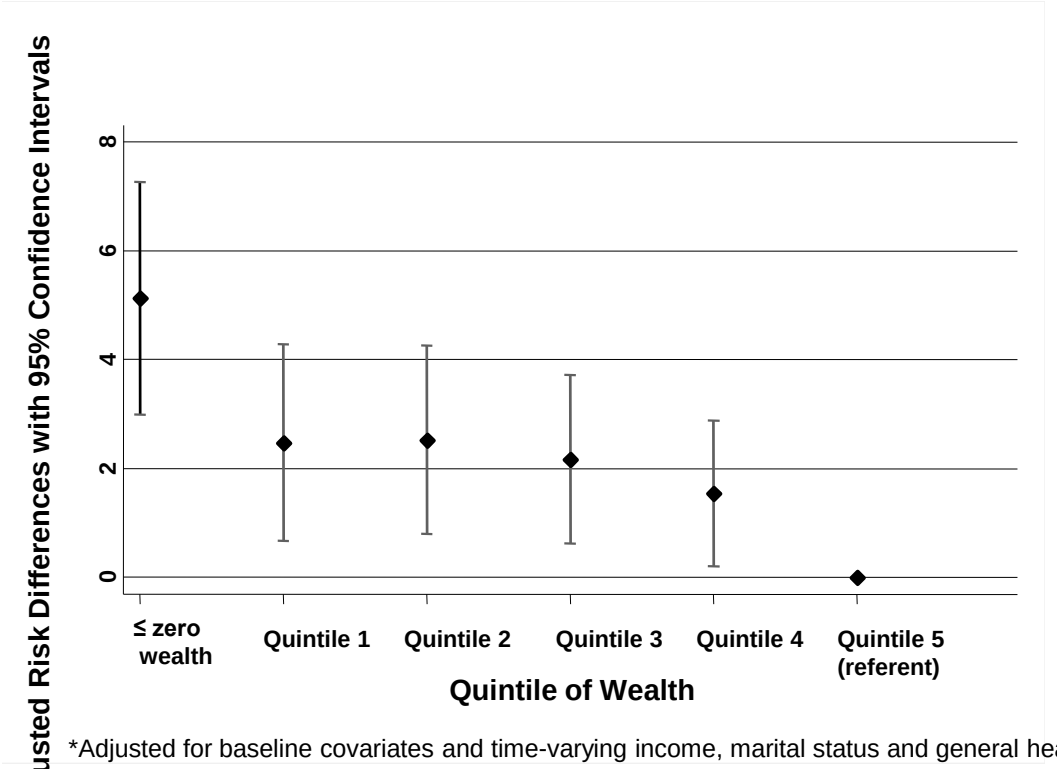


Figure 6. Risk ratios and 95% confidence intervals of mortality by wealth quintile, PSID 1984-2005



*Adjusted for baseline covariates and time-varying income, marital status and general health status

Figure 7. Risk differences and 95% confidence intervals of mortality by wealth quintile, PSID 1984-2005



*Adjusted for baseline covariates and time-varying income, marital status and general health status
**Risk differences expressed as per 1,000 persons

Table 13. Risk ratios and 95% confidence intervals for the association between mortality, general health status and wealth among men, women, whites and non-whites, PSID 1984 - 2005

Self-Reported General Health Status								
	Men	*95% CI*	*Women*	*95% CI*	*White*	*95% CI*	*Non-White*	*95% CI*
≤0 wealth	1.75	1.32, 2.31	1.56	1.18, 2.05	2.01	1.54, 2.62	1.02	0.80,1.31
Quintile 1	1.41	1.08, 1.82	1.62	1.26, 2.09	1.65	1.31, 2.10	1.00	0.78, 1.28
Quintile 2	1.29	1.01, 1.66	1.30	1.01, 1.68	1.38	1.10, 1.72	0.88	0.69, 1.12
Quintile 3	1.19	0.95, 1.50	1.50	1.18, 1.89	1.23	0.99, 1.52	0.91	0.71, 1.16
Quintile 4	1.14	0.92, 1.42	1.19	0.95, 1.49	1.06	0.87, 1.30	0.89	0.69, 1.13
Quintile 5	Referent		Referent		Referent		Referent	
Mortality								
	Men	*95% CI*	*Women*	*95% CI*	*White*	*95% CI*	*Non-White*	*95% CI*
≤0 wealth	1.61	1.15, 2.25	2.29	1.60, 3.26	1.87	1.33, 2.63	1.69	1.17, 2.44
Quintile 1	1.23	0.89, 1.71	1.79	1.27, 2.54	1.77	1.30, 2.40	1.23	0.82, 1.83
Quintile 2	1.11	0.81, 1.53	1.93	1.37, 2.73	1.30	0.94, 1.80	1.08	0.71, 1.64
Quintile 3	1.14	0.86, 1.51	1.84	1.32, 2.55	1.24	0.94, 1.64	1.50	1.02, 2.20
Quintile 4	1.20	0.91, 1.58	1.37	1.02, 1.83	1.12	0.90, 1.40	1.38	0.94, 2.02

Quintile 5	Referent		Referent		Referent		Referent	

Table 14. Risk difference and 95% confidence intervals for the association between mortality, general health status and wealth among men, women, whites and non-whites, PSID 1984 - 2005

Self-Reported General Health Status								
	Men	*95% CI*	*Women*	*95% CI*	*White*	*95% CI*	*Non-White*	*95% CI*
≤0 wealth	29	15, 42	22	9, 34	29	17, 42	4	-13, 22
Quintile 1	15	4, 25	23	12, 34	20	10, 29	1	-15, 18
Quintile 2	11	1, 21	11	1, 21	11	3, 18	-7	-23, 8
Quintile 3	7	-2, 16	18	8, 27	6	0, 13	-5	-21, 11
Quintile 4	5	-3, 13	6	-2, 15	2	-4, 7	-7	-22, 9
Quintile 5	Referent		Referent		Referent		Referent	
Mortality								
	Men	*95% CI*	*Women*	*95% CI*	*White*	*95% CI*	*Non-White*	*95% CI*
≤0 wealth	7	2, 11	5	2, 7	4	1, 7	6	2, 10
Quintile 1	3	-1, 7	3	1, 5	4	1, 6	2	-2, 6
Quintile 2	1	-2, 5	3	1, 5	1	0, 3	1	-3, 5
Quintile 3	2	-2, 5	3	1, 5	1	0, 3	4	0, 9

Quintile 4	2	-1.1, 5	1	0.1, 3	1	-0.5, 2	3	-0.5, 7
Quintile 5	Referent		Referent		Referent		Referent	

*per 1,000 persons

Table 15. Risk ratios, risk differences and their respective 95% confidence intervals for the association between general health status and wealth, PSID 1984 – 2005

	Risk ratio	*95% CI*	*Risk difference**	*95% CI*
≤ 0 wealth	1.54	1.27, 1.88	22	13, 32
Quintile 1	1.41	1.18, 1.69	16	8, 24
Quintile 2	1.21	1.01, 1.45	8	1, 16
Quintile 3	1.25	1.06, 1.47	9	3, 16
Quintile 4	1.17	1.00, 1.37	6	0, 13
Quintile 5	Referent		Referent	

*per 10,000 persons

Table 16. Risk ratios, risk differences and their respective 95% confidence intervals for the association between mortality and wealth, PSID 1984 – 2005

	Risk ratio	*95% CI*	*Risk difference**	*95% CI*
≤ 0 wealth	1.83	1.44, 2.31	5	3, 7
Quintile 1	1.40	1.10, 1.77	2	1, 4
Quintile 2	1.41	1.12, 1.77	3	1, 4
Quintile 3	1.35	1.09, 1.67	2	1, 4
Quintile 4	1.25	1.03, 1.51	2	0.2, 3
Quintile 5	Referent			

*per 1000 persons

Chapter 7

Discussion

Summary of findings and study aims

The aims of this dissertation were straightforward and the study questions asked were simple. The first aim of this dissertation was to investigate the association between wealth and the incidence of 3 cardiovascular disease risk factors: smoking, hypertension and obesity on the absolute and relative scales in the time period 1999 - 2005. The results for aim 1 showed a strong association between wealth and obesity incidence, a moderate association between wealth and smoking initiation and a weak association between wealth and hypertension incidence. This was true on both the absolute and relative scales, where both the risk ratios and risk differences revealed similar overall patterns.

Aim 2 of this study was to investigate the overall, gender and race specific association between wealth and all-cause mortality and poor self-reported general health status on the absolute and relative scales in the time period 1984 - 2005. Our study of the association between wealth and these 2 health outcomes found a strong inverse association between wealth and GHS and between wealth and mortality. GHS models stratified by race and gender revealed a similarly strong association for men, women and whites, while stratified mortality models suggested strong relative effects among women and whites.

Strengths

There were several strengths to our study. First, by using data from the PSID, we were assured that wealth was measured in a rigorous and comprehensive fashion (61). As

discussed earlier many epidemiologic studies measured wealth poorly, thus the estimates based on these studies run the risk of serious measurement error (8). Furthermore, many studies that use wealth as a measure of SES focus solely on elderly populations (11, 19-21, 25, 27, 30, 36, 38, 39, 45, 48). This study sought to understand the association between wealth and health in a working age population. Our results indicate that wealth is indeed associated with several different health outcomes implying it may be useful as an additional measure of SES among non-elderly populations. Although the wealth-health association may not be as strong as the income-health association, the conceptual advantages of wealth may outweigh empirical considerations in certain cases. That is researchers should consider their study population as well as the nature of the SES-health relationship when selecting a measure of SES to use in future health studies.

In addition, the longitudinal nature of the data allowed us to explore the question of wealth and health from a life course perspective as well as to attempt to disentangle the direction of the causal relationship between wealth and health. Most previous studies of wealth and the health outcomes studied here have used cross sectional data (7, 7, 11, 16, 16, 19-32), making it impossible to disentangle the direction of causality. A related advantage was our use of marginal structural models as the analytic technique to answer the study question. After satisfying several important assumptions, MSM can have a causal interpretation even in the presence of time-varying confounding. Without overstating the causal nature of the association between wealth and health, we believe this study produced improved estimates relative to past studies (76).

However, it should be noted that the results we obtained from MSM were similar to those from traditional models for the CVD risk factors and for GHS (see Table 17 and Table

18, in the Appendix). This indicates that there was not a substantial amount of time-varying confounding in these health outcomes. We hypothesized that income would be the strongest time-varying confounder. However, given the short time interval in the CVD analysis, it is likely that income trajectories remained relatively stable during this period; thus it is understandable that income was not a strong time-varying confounder for the CVD risk factor. It is well known that the strong confounders for the SES - CVD association are actually time-invariant confounders such as age, race and sex; this was confirmed in our study.

In the mortality analysis, however, there was a considerable amount of time-varying confounding (see Table 18 in the Appendix). In light of the longer follow up in the mortality analysis, more time-varying confounding was anticipated. Furthermore, the differences between traditional and MSMs could reflect the difference in the nature of the mortality outcome compared to the other self-reported ones. That is, time-varying confounders may be more relevant to mortality than to the other outcomes.

Limitations

There are other potential explanations for the observed association between wealth and these health outcomes. Specifically, the weak association between wealth and hypertension was not anticipated. The use of self-reported data about hypertension (and obesity) probably resulted in misclassification. Hypertension was most likely underreported among younger respondents who have yet to be diagnosed with hypertension, but accurately reported among older individuals who were involved in the ongoing management of the condition (68). Several studies have found that women, older and more educated individuals have less misclassification for self-reported hypertension than other groups (68-71). Given

the relationship between wealth, age, education and health insurance status (the least wealthy tend to be younger, less educated and uninsured) we may have differential misclassification of hypertension. In light of this misclassification bias, our data would reveal fewer hypertensives among the less wealthy, thus potentially explaining the attenuated results we see for this outcome. Additional sensitivity analysis would be required to further explore the impact of the misclassification bias. It should be noted, however, that other studies have found a weak association between hypertension and income, thus if wealth is expected to operate in a similar manner to income our result may not be wholly unexpected (65, 66).

The exclusion of baseline cases from the analysis raises questions about selection bias, another potential source of bias in this study. Those with preexisting hypertension are more likely to be older and of lower SES. By excluding them, the effect estimate will likely be biased downward. That is, a sample of healthier, wealthier individuals will create an underestimate of the effect estimate for hypertension (75). Modeling the probability of hypertension instead of the incidence will help address the concern of selection bias. Table 19 in the Appendix presents the model for the probability of hypertension. Although the magnitude of effect is similar between the incidence and the probability models for hypertension (except for the risk ratio for the less than or equal to 0 group), the precision in the probability model is improved. This suggests that the exclusion of baseline cases in the hypertension analysis results in a weaker association between wealth and hypertension.

We should also point out the likely bias in the mortality findings resulting from attrition within the PSID. Respondents who were lost to follow up were included in the analysis and censored at their last time point; however, their mortality status remained unknown. If there was differential mortality between the censored and the uncensored

respondents, (it is feasible that censored individuals had higher mortality rates and were less wealthy) we would observe bias towards the null. That is our results may be underestimating the real mortality risk. PSID conducted an internal examination of the affects of attrition on mortality and found that the mortality status of 11% of a sample of persons lost to follow-up could not be determined and about 3% were actually deceased (85). Overall the problem of attrition is significant in PSID. Of the approximately 18,000 individuals in the sample in 1968 only about 5000 were interviewed in 2001 (92).

The findings related to the other health outcomes, smoking, obesity and general health status were all in the expected direction. It should be noted, however, that the discussion of selection bias resulting from the exclusion of baseline cases applies to these health outcomes as well. Specifically, the discussion in Chapter 5 examines selection bias in smoking more closely. Furthermore, given that height and weight were self-reported, some misclassification of obesity was inevitable. Thus the results presented in Chapter 5 are likely an underestimation of effect.

Although MSM can produce a causal effect estimate, it should be noted that this holds only if certain assumptions are met. The assumption of no unmeasured confounding, a prerequisite for producing causal estimates, is unlikely for this dissertation. For example, health insurance status is arguably a potential confounder in the GHS-wealth and mortality-wealth analyzes. Unfortunately, health insurance data were not collected in early waves of the PSID. Others may argue that our adjustment for baseline health status was not sufficient in the mortality-wealth analysis. Other studies have used less crude measures to control for baseline health (48). Furthermore, causality can only be inferred from observational data if the assumption of consistency holds. That is the researcher can randomize participants to the

exposure, as is done in a randomized control trial. Wealth, similar to other social factors, is not fully assignable. Government policies, however, do play some role in the distribution of wealth in the US, thus it is not wholly unrealistic to conceptualize wealth as at least in part randomizable. Therefore, we believe that the use of MSM in this dissertation improves upon past estimates, but must be seen in light of these caveats.

Mechanisms

The choice of the 3 CVD risk factors as health outcomes for this study was motivated primarily by our interest in the chronic stress pathway. It has been hypothesized that chronic stress and other psychosocial factors trigger a series of biological events, through central nervous system activation of autonomic, neuroendocrine and immune responses (82). Since wealth is a stockpile of financial resources, a lack of wealth (which translates into the absence of a safety net) is ostensibly a cause for financial stress. The absence of wealth over time is thus a cause of chronic stress. The lack of wealth among poor families may result in worries over immediate survival in the event of job loss or some other catastrophe, but among middle class families a lack of wealth, for example, may result in stress surrounding a family's ability to save for higher education for their children. The absence of wealth, therefore, is not stressful for poor families, but may weigh heavily on middle and upper class families as well.

The biological pathways associated with the chronic stress hypothesis may be especially germane to hypertension and other cardiovascular functions (83). In addition, smoking and weight gain may be mechanisms to cope with chronic stress for some individuals. Thus they fall on the causal pathway from a lack of financial resources to cardiovascular disease. Although they are not health outcomes per se, smoking and obesity

are important risk factors for a host of diseases and they are important in cultivating healthy environments and lifestyles.

In addition, poor physical and social environments can encourage health damaging exposures (77). For example, the lack of economic resources associated with having little wealth may limit an individual's access to health care, quality housing and nutritious foods among other things. A lack of economic and social resources can also result in insufficient investment in "human, physical, social and health infrastructure" which may be detrimental to the health of populations (78). Assuming that less wealthy individuals live in less wealthy communities, insufficient infrastructure may take the form of fewer or less convenient public transportation options, a lack of parks and unsafe streets, and more liquor and convenient stores. These deficiencies can then lead to increased isolation, a more sedentary lifestyle and poor diets, which directly result in higher rates of smoking and obesity among less wealthy individuals (79, 80). Other research has found that low SES individuals may have less social support, higher job strain and less job control. All these factors have been associated with higher rates of smoking and obesity (81).

Poor general health status may result from several of the above mentioned pathways. For example, a lack of economic resources may result in poor access to health care which may in turn result in poor health status. The general health status variable may also be capturing a host of other health problems which result from poor physical and social environments. Given its lack of specificity, several potential pathways could result in poor health status.

The cumulative disadvantage literature seems most relevant to a discussion of wealth and mortality. If posits that inequalities in SES worsen over the life course, resulting in

worse health outcomes for low SES individuals. Therefore the effects of low SES over a lifetime are more likely to be seen with an outcome like mortality than a more proximal one. The other health outcomes, smoking, obesity, hypertension and GHS, all show stronger effects among the poor. Since these factors contribute to mortality, the pathways through which they function may also be acting on mortality.

Policy Implications

Most government policy to date has focused on improving the income of poor families. Programs such as Temporary Assistance for Needy Families (TANF), food stamps, section VIII housing subsidies and the earned income tax credit are all directed towards helping poor families augment resources for consumption. About 40% of low income families own homes and have other assets, such as savings accounts. Thus policies that focus on increasing wealth may further assist low income families to find financial stability.

Many current policies discourage low income families from asset building. For example, the tax exemption for mortgage interest on homes is targeted mainly at middle and upper income families. Low income families who pay little to no income tax do not benefit from it. In addition, for those families who receive housing subsidies, purchasing a home would result in the loss of this benefit thus further discouraging low income families to buy a home. Furthermore, many current income assistance programs, such as TANF and food stamps, have asset restrictions. Families wishing to save money may lose these benefits if their savings were beyond a certain amount; yet another disincentive for poor families to build assets.

In recent years, the advent of individual development accounts (IDA) is a possible avenue to assist low income families with asset building. IDAs are restricted savings

accounts, where withdrawals must be used for specific purposes such as home ownership, education or to start a business. Upon withdrawal (for a sanctioned purpose) funds are matched by state and federal governments. As of 2003, on average funds were being matched with $2 for each dollar invested by the account holder for a maximum of $13,000 eligible for matching (93). As of 2007, 37 states plus the District of Columbia and Puerto Rico had enacted an IDA programs (94). Although IDAs are a relatively new asset building tool, thus far an important lesson learned from IDAs is that low income families can indeed save money.

Future directions

The study of wealth's impact on health still remains relatively unexplored. In addition to the health outcomes presented here, several other health outcomes would be worthy of exploring. Specifically CVD outcomes such as stroke and heart attacks as well as CVD-specific mortality would be of interest within the context of the chronic stress pathway. Several large studies with good wealth measures, PSID included, have expressed interest in collecting biomarker data. The association between well measured clinical health outcomes and wealth (from the PSID or other sources) could prove a useful addition to this literature.

In addition to assessing other health outcomes, certain populations may benefit more for the study of wealth and health than others. As indicated by past research, the elderly and unemployed are populations in which the use of wealth would be useful. For example, from these results we see little benefit from using wealth in minority populations. Given the very low levels of wealth among black and Hispanic families, wealth may not be a useful measure of SES in studies that focus on these groups.

Furthermore, in light of the tremendous inequality in wealth in America the study of wealth inequality may yield robust and interesting findings. Several measures of wealth inequality could be ascertained. Similar to the income inequality literature, the Gini coefficient or Robin Hood Index could be used to ascertain wealth inequality. Compared to the research looking at income inequality and health, wealth inequality may yield far more decisive results. In addition, recent work on the concept of relative deprivation could prove to be well suited for a measure like wealth (95).

Conclusions

Future health research may benefit from the use of wealth as an additional measure of SES. As discussed earlier, wealth has many advantages relative to other SES measures. If health research can incorporate an effective yet concise way to measure wealth, the limitations of using wealth in health research may be more easily overcome. Forthcoming work by Cubbin and colleagues, hopes to present one way to effectively measure wealth in health research (13). Alternatively, improving the health measures included in social science surveys that already measure wealth effectively would be an alternate source of data for epidemiologists to use. In addition, wealth may provide better control for SES than income, education or occupation. This is critical in ensuring that future health research effectively controls for the powerful confounding effects of SES when exploring additional exposure-disease relationships. Poor control for SES may lead to overestimated results and misleading conclusions.

Appendix

Table 17. Risk ratios and 95% confidence intervals from traditional models (non-MSM) for CVD outcomes, PSID 1999 - 2005

	Obesity			**Smoking**			**Hypertension**		
	risk ratio	**Lower 95% CI**	**Upper 95% CI**	**risk ratio**	**Lower 95% CI**	**Upper 95% CI**	**risk ratio**	**Lower 95% CI**	**Upper 95% CI**
≤ zero wealth	1.71	1.37	2.14	2.10	1.46	3.01	1.17	0.96	1.42
Quintile 1	1.74	1.39	2.17	1.58	1.09	2.30	1.25	1.02	1.52
Quintile 2	1.51	1.22	1.85	1.59	1.11	2.27	1.24	1.03	1.48
Quintile 3	1.54	1.27	1.87	1.42	1.00	2.01	1.17	0.99	1.39
Quintile 4	1.36	1.13	1.64	1.30	0.93	1.82	1.17	1.01	1.36
Quintile 5	Referent			Referent			Referent		

Table 18. Risk ratios and 95% confidence intervals from traditional models (non-MSM) for general health status and mortality, PSID 1984 - 2005

	General Health Status			Mortality		
	Risk ratio	Lower 95% CI	Upper 95% CI	Risk ratio	Lower 95% CI	Upper 95% CI
≤ 0 wealth	1.70	1.44	1.99	1.32	1.07	1.63
Quintile 1	1.48	1.27	1.73	1.07	0.87	1.31
Quintile 2	1.25	1.08	1.45	1.16	0.97	1.40
Quintile 3	1.23	1.07	1.41	1.08	0.91	1.28
Quintile 4	1.18	1.04	1.34	1.00	0.85	1.16
Quintile 5	Referent			Referent		

Table 19. Prevalence and incidence models for hypertension, PSID 1999-2005

	Prevalence model				Incidence model			
	Risk ratio	Lower 95% CI	Upper 95% CI	Confidence Limit Ratio	Risk ratio	Lower 95% CI	Upper 95% CI	Confidence Limit Ratio
≤ 0 wealth	1.26	1.14	1.40	1.23	1.13	0.91	1.40	1.54
Quintile 1	1.21	1.10	1.34	1.22	1.23	0.99	1.52	1.54
Quintile 2	1.19	1.08	1.30	1.21	1.22	1.00	1.49	1.49
Quintile 3	1.16	1.06	1.26	1.19	1.16	0.96	1.39	1.45
Quintile 4	1.07	1.00	1.15	1.16	1.07	0.92	1.26	1.37
Quintile 5	Referent				Referent			

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