

A GLOBAL PERSPECTIVE ON AGING, ADULT HEALTH, AND INEQUALITY

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## ABSTRACT

### A GLOBAL PERSPECTIVE ON AGING AND INEQUALITY

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In many developing countries, the share of the population living in the adult ages is increasing. Despite these demographic shifts, there are still many gaps in the research on aging and adult health in developing countries. My first chapter uses data on Indonesia to study socioeconomic differences in adult mortality. I find that the size of socioeconomic differences is much smaller in Indonesia than in many HICs and not explained by behavioral risk factors. My results suggest that mortality inequality in middle-income countries may follow a trajectory that is distinct from the current and historical experiences of HICs. One surprising finding from my first chapter is that high blood pressure is very high in Indonesia and strongly predictive of mortality. My second chapter builds on these findings by examining the etiology of high blood pressure in Indonesia. Using fixed-effects panel data methods with 17 years of longitudinal data in Indonesia, I find that changes in weight are related to changes in blood pressure across the entire distribution of BMI. My findings reveal that changes in weight among lean individuals can still have consequences for blood pressure and that conventional risk factors for high blood pressure may not be sensitive indicators of disease in developing contexts. Underlying the entire study of individual aging is the question of why some individuals engage in behaviors that are known to negatively affect health. My third chapter uses data on U.S. twins to investigate the degree to which multiple adult health behaviors can be explained by a single set of characteristics. Our paper combines approaches from economics and behavioral genetics to determine the contribution of schooling, genetic endowments, and environments to unhealthy behaviors among U.S. adults. We find that most health-related behaviors in adulthood are largely idiosyncratic and likely not caused by single factors. The results from the three chapters suggest that greater attention needs to be given to context-specific determinants of behavior, health, and mortality. As countries around the world continue to age, understanding why differences in aging exist across and within populations can provide new insights to promote healthy aging globally.

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## PREFACE

In many developed and developing countries, the share of the population living in the adult ages is increasing. For example, between 1990 and 2015, the share of the population above the age of 15 grew from 78.4% to 81.0% in the United States and from 63.6% to 72.3% in Indonesia. As populations around the world age and life expectancy increases, the overall burden of health and mortality will shift from infectious diseases, which are most common among children, to chronic non-communicable diseases, which occur most often in adulthood. The combined consequences of population aging and the epidemiological transition makes health and mortality in adulthood more important for overall population health. Despite these demographic and epidemiological shifts, there are still many gaps in the research on aging and adult health. The goal of my dissertation is address some of these gaps by providing three new contributions to the study of aging globally.

Although life expectancy is improving globally, patterns of disease and health behaviors still vary substantially across countries and regions. For example, cardiovascular diseases are decreasing in many developed countries but have become extremely prevalent in larger developing countries like India and Indonesia. Similarly, cancers and neurodegenerative diseases are increasing sharply in developed countries but remain low in most developing countries. Even within countries at similar levels of national income, some conditions, like obesity, vary substantially. Importantly, the within-country social patterning of these diseases and risk factors also vary across contexts: in high-income countries, risk factors like smoking and obesity are more pronounced among the poor than the rich; in contrast, these gradients are reversed or non-existent in many aging middle-income countries like Indonesia.

Since these diseases and behaviors have different implications for mortality, insights about the extent and causes of mortality inequality from high-income countries (HICs) may not translate to aging middle-income countries. However, to date, the majority of the literature on adult mortality is focused on high-income countries. My first chapter

addresses this gap in the literature using data from Indonesia—the third largest middle-income country. Using longitudinal information on adults over the age of 30, I find that the size of socioeconomic differences in adult mortality is much smaller in Indonesia than in many HICs. The small SES differences relative to developed countries may be driven by the distribution of risk factors across SES groups. In developed countries, risk factors for NCDs such as obesity, tobacco use, and hypertension are more pronounced among the poor and are implicated as primary reasons for SES differences in longevity. In contrast, SES gradients in risk factors in Indonesia are very different than in developed countries: high blood pressure and tobacco use are high across all SES groups and obesity is slightly higher among the upper SES groups. Consequently, I find that these risk factors do not explain why upper-SES groups experience lower mortality in adulthood. Instead, the moderate differences across SES groups may be driven by access to health resources and services. Overall, my results suggest that mortality inequality in middle-income countries may follow a trajectory that is distinct from the current and historical experiences of HICs.

As adult mortality in middle-income countries increases as a share of overall mortality, identifying the determinants of adult health is important for setting research and policy priorities. One surprising finding from my first chapter is that high blood pressure is very high in Indonesia and strongly predictive of mortality. My second chapter builds on these findings by examining the etiology of high blood pressure in Indonesia. While a large literature from HICs finds that obesity is a main risk factor for high blood pressure, few people in Indonesia, and many other low- to middle-income countries (LMICs), are clinically obese. Given this combination of high blood pressure and low obesity, an important question is whether weight is related to blood pressure among leaner individuals. Using fixed-effects panel data methods with 17 years of longitudinal data in Indonesia, I find that changes in weight are related to changes in blood pressure across the entire distribution of BMI. My findings reveal that changes in weight among lean individuals can still have consequences for blood pressure and that conventional risk factors for high blood pressure may not be sensitive indicators of disease in developing contexts.

Underlying the entire study of individual aging is the question of why some individuals engage in behaviors that are known to negatively affect health. While many potential causes of health-related behaviors have been identified—such as schooling, genetics, and environments—little is known on how much of the variation across multiple behaviors is due to a common set of causes. My third chapter, co-authored with Jere R. Behrman and Hans-Peter Kohler, uses data on U.S. twins to investigate the degree to which multiple adult health behaviors can be explained by a single set of characteristics. Our paper combines approaches from economics and behavioral genetics to determine the contribution of schooling, genetic endowments, and environments to unhealthy behaviors – or the outcomes of such behaviors such as BMI and waist circumference – among U.S. adults. We find that most health-related behaviors in adulthood are largely idiosyncratic and likely not caused by single factors, whether that is schooling, genetics, or environments. The one prominent exception to this pattern is the relationship between smoking and unhealthy drinking: although the environmental correlation between these two is modest, our results suggest that a common aspect of the childhood and adolescent environment is consistent with variation in both behaviors.

My dissertation contributes to the literature on aging globally by expanding the study of mortality inequality and disease etiology into middle-income countries and investigating the underlying causes of health behaviors in adulthood. The results from the three chapters suggest that greater attention needs to be given to context-specific determinants of behavior, health, and mortality. As countries around the world continue to age, understanding why differences in aging exist across and within populations can provide new insights to promote healthy aging globally.

# CHAPTER 1 : Socioeconomic Differences in Adult Mortality in a Developing Country: Evidence from Indonesian Adults

## 1.1. Abstract

In developed countries, studies of socioeconomic status (SES) differences in adult mortality almost unanimously find that higher SES groups have lower mortality and consequently higher life expectancy. Despite the large body of work in developed countries, there is little research on adult mortality differences in developing countries, where research on mortality has historically focused on children. Given the large contextual differences between developed and developing countries, insights on the extent and causes of adult mortality differences from developed countries may not translate to developing countries. Using a large national data set from Indonesia — the fourth most populous country and the third most populous developing country — I combine information across a wide range of adult ages to provide new estimates of SES differences in adult mortality. Second, I use biomarker and anthropometric data to estimate the contribution of major risk factors for adult mortality (hypertension, unhealthy weight, and tobacco use) to observed mortality differences. I find that mortality differences in Indonesia are complex, and depend on sex and the type of SES measure used. For both rural and urban men, there are modest differences in adult mortality across an asset-based wealth index, but not across expenditure quartiles. In contrast to men, I find little evidence of inequality for women. Second, I find that risk factors for adult mortality (tobacco use, obesity, and hypertension) are high across all SES groups and do not explain differences in adult mortality across SES quartiles. Overall, my results suggest that higher consumption or wealth is not always associated with higher life expectancies in a developing country. Constrained health resources or the high prevalence of tobacco use and hypertension across all groups may overshadow SES differences.

## 1.2. Introduction

Across developed countries, more educated and affluent individuals tend to live longer than the poor [10, 12, 13, 18, 23, 28, 33]. For example, men in the United States (US) with less than a high school education have a life expectancy at age 25 that is 10.2 years less than US men with at least a college degree, while US women with less than a high school education have a 9.7 year lower life expectancy compared to women with at least a college degree [23]. Similarly, a recent study finds around a 10-year difference in life expectancy at age 40 between the top and bottom quartiles of income for US men and around a 6-year difference for women [10]. These studies have fueled recent debates among researchers and policy makers about the root causes of mortality inequality and how to address them.

Despite the large body of work in developed countries, there is little research on the association between socioeconomic status (SES) and adult mortality in developing countries, where research on mortality has historically focused on children. There are reasons to suspect that the association between SES and adult mortality in developing countries may be more or less pronounced. First, many developed countries have public safety nets for poorer individuals, which may insulate poorer and older individuals against excess mortality [45]. In developing countries, the lack of strong health safety nets for poorer individuals may lead to more pronounced inequality between the rich and poor, since only richer individuals would have access to healthcare resources. Second, a large portion of the relationship between SES and adult mortality in developed countries is driven by causes of death linked to unhealthy behaviors (mainly smoking, excess weight, and heavy alcohol consumption), which are more common among the poor than the rich [33]. In contrast, in developing countries, higher SES groups may face the greatest burden from lifestyle diseases such as heart disease, diabetes, and hypertension [3, 14]. This patterning of health risks suggests that the relationship between SES and adult mortality may be smaller in developing countries than in developed countries, since the rich in developing countries may lower their life expectancy through behavioral causes of death. Third, the quality of healthcare in developing

countries is often poor [15, 16]. SES differences in adult mortality in developing countries may be less pronounced if a generally low quality of health care and infrastructure limits the ability of higher SES individuals to seek and purchase better health. Given the large contextual differences between developed and developing countries, insights on the extent and causes of mortality inequality from developed countries may not translate to developing countries.

My study provides new evidence on the relationship between SES and adult mortality in developing countries. Prior literature on SES differences in adult mortality within developing countries has focused on specific age groups or used methodological approaches that do not readily translate into summary measures of longevity like life expectancy [4, 25, 38]. Many previous studies have also been based on smaller countries that are just beginning the epidemiological transition [9]. Using a large national data set from Indonesia — the fourth most populous country and the third most populous developing country — I combine information across a wide range of adult ages to provide new estimates of SES differences in adult mortality for a country where adult diseases are the leading causes of death [51]. I also take advantage of measured biomarker and anthropometric data and information on health behaviors to estimate the contribution of major risk factors for adult mortality (hypertension, unhealthy weight, and tobacco use) to SES differences in adult mortality.

### 1.3. Background

#### *1.3.1. SES, Adult Health, and Mortality in Developing Countries*

The relationship between SES and adult mortality has historically been studied by comparing mortality across countries or regions at different levels of national income and development [24, 40]. As individual level data sources have become available, a small literature on SES differences in adult mortality within developing countries has emerged. The results from these studies are mixed, with the size of the relationship varying across countries and measures of SES. For example, studies find evidence of an association between SES and mortality for adults over the age of 20 in rural Zambia and older adults (greater than age

65) in China and Indonesia; in contrast, other studies find no evidence of wealth differences in adult mortality in Tanzania or rural Kenya [9, 25, 30, 38]. Even within countries, the SES-mortality relationship varies across measures of SES: using data on South African adults, Ardington and Gasealahwe (2014) find that adults with higher levels of wealth are less likely to die between survey waves; however, they fail to find a relationship when SES is measured based on household per capita expenditure. Although these studies have greatly expanded the literature on SES and adult mortality in developing countries, they are limited by either a focus on specific age groups (such as adults over the age of 65) or by measures of mortality that are difficult to compare across countries and time periods. Estimating the association between SES and mortality across a wider range of adult ages with a standardized measure such as life expectancy at age 30 (a common measure used in studies of longevity in developed countries) would allow for greater comparability of the size of SES differences across contexts and time.

Beyond mortality, many studies examine SES differences in adult health within developing countries. In general, these studies find that higher SES individuals are more likely to have greater levels of non-communicable diseases (NCDs) and risk factors for these diseases compared to lower SES individuals; however, the size of these differences is often small with many exceptions to the pattern of inequality by SES. For example, two major reviews of the relationship between socioeconomic status and obesity in developing countries find mixed relationships: while higher SES individuals tend to be more obese in low income countries, levels of obesity tend to be similar across SES groups for low-middle and middle income countries [17, 34]. Similarly, while more educated individuals tend to smoke less compared to less educated individuals, the absolute differences across education groups is small in most countries [14]. The relationship between SES and adult health may also vary by region: a study using data from nine rural INDEPTH Health and Demography Surveillance System sites in Bangladesh, India, Indonesia, Thailand, and Vietnam finds greater clustering of NCD risk factors (including tobacco use, poor diet, physical inactivity, high blood pressure, and high body mass index) among individuals with higher levels of edu-



cation [1]. Despite the large literature on the relationship between SES and adult health, the contribution of health conditions to SES differences in adult mortality in developing countries remains unknown.

### *1.3.2. Indonesia*

Indonesia presents an important context to study the association between SES and adult mortality. Indonesia is the third most populous developing country in the world with a 2015 population of 254,564,000 that is projected to grow to 313,648,000 by 2100 [48]. Indonesia's population is also aging and adult causes of death are now the leading cause of death: the proportion of the population between ages 15 and 65 grew from 64.6% in 2000 to 67.1% in 2015 while stroke, ischemic heart disease, and diabetes have taken over as the top three leading causes of death as of 2012 [49, 51]. Finally, inequality in Indonesia is present across both geographic and socioeconomic dimensions. Geographically, Indonesia is an archipelago that consists of 13,466 islands that span a distance of 3,182 miles between the Indian and Pacific Oceans. Within the archipelago, 54% of individuals live on the island of Java while only around half the population overall lives in urban areas as of 2010 [5]. Economically, income inequality is growing rapidly in Indonesia with a rise in the Gini index from 29.7 in 2000 to 35.6 in 2010 [49].

SES differences in health in Indonesia also mirror the larger trends found in many developing countries. For example, obesity and unhealthy weight are more common among the more educated and wealthier segments of the population, while tobacco use is slightly more common among the lower education and wealth groups [36, 39, 43, 46]. Similarly, high SES individuals are more likely to have unhealthy levels of NCD risk factors, including diabetes and hypercholesterolemia [27]. In terms of functional limitations, more educated individuals and individuals who self-report higher SES are less likely to be disabled compared to lower SES groups [35, 37]. Studies have also found evidence that more educated individuals may be more resilient to shocks and more likely to seek treatment for illnesses [21, 26]. Since the overall patterning of SES differences in adult health in Indonesia is very

similar to the patterns observed in developing countries at similar levels of development, insights drawn from an analysis of Indonesia are potentially relevant to a broader set of developing countries.

Based on the existing literature, the relationship between SES and adult mortality in developing countries is ambiguous. By using data from Indonesia, standardized measures of adult mortality over a wide range of adult ages, and measured biomarker and anthropometric data, the goals of this study are: (1) to provide new evidence on the association between SES and adult life expectancy; and (2) estimate the contribution of health conditions and risk factors to differences across SES groups.

## 1.4. Data and Methods

### *1.4.1. Data*

Data are from the 2007 and 2015 waves of the Indonesian Family Life Survey (IFLS), a longitudinal nationally representative survey of 13,535 households [47]. The IFLS surveyed households from 13 of Indonesia’s 27 provinces-the remaining 14 provinces were not sampled due to political violence and the high cost of surveying more remote regions of the country. The 13 selected provinces contain the majority of the country’s population, making the IFLS representative of 83% of the population of Indonesia. For each of the selected households, the most knowledgeable household member provided basic information on every household member (such as age, sex, and educational attainment) and information on household consumption, expenditures, and assets (such as a home, car, or livestock). In addition, trained assessors collected anthropometric data (height, weight, blood pressure, waist circumference) for a subsample of individuals in the household. If an individual present in the 2007 survey died between 2007 and 2015, the IFLS interviewed a household member in 2014 about the deceased and asked them to provide information on the month of death and year of death.

#### *1.4.2. Main Variables*

The primary outcome is adult mortality. Specifically, age at death information was constructed based on self-reported birth date and household member reported date of death. In addition to mortality, I examined the contribution of adult risk factors to differences in mortality between SES groups. Specifically, I examined three of the four leading risk factors for adult mortality: obesity, hypertension, and tobacco use (the IFLS does not collect data on the fourth risk factor, excess alcohol consumption) [29]. Based on standard World Health Organization cutoffs, individuals were classified as obese if they had a body mass index greater than or equal to 30; individuals were classified as hypertensive if they had a systolic blood pressure greater than or equal to 140 mmHg and/or a diastolic blood pressure greater than or equal to 90 mmHg [50, 52]. Individuals were classified as ever users of tobacco if they answered yes to the question: “Have you ever had a tobacco habit?” Finally, individuals were classified as living in an urban or rural area based on Indonesian census classifications.

The SES of individuals was measured using two common approaches: consumption or expenditure quartiles and an asset-based wealth quartile. The individual in the household most knowledgeable about expenses was asked to report monthly expenditure on a range of goods and services. Total household income per capita was then calculated as the sum of all the expenditure aggregates divided by the number of household members. I then classified individuals into quartiles of consumption; this procedure was done separately for individuals living in rural and urban areas to adjust for differences in consumption patterns and the cost of living between urban and rural areas.

While consumption/expenditure data provide information on a household’s current living standards, many researchers and organizations, including the Demographic and Health Surveys (DHS), measure SES using housing- and asset-based wealth indices [20, 44]. In contrast to consumption, which is generally viewed as a short-term measure, housing conditions and assets are thought to reflect long term SES. Using both consumption and a

wealth index may provide greater insight into which dimensions of SES are more predictive of mortality. To create a wealth index, I followed the same procedure used by the DHS and classified individuals into wealth quartiles based on a wealth index that is created using principle components analysis and information on asset ownership and housing characteristics (Details of this process are presented in Appendix A).

### *1.4.3. Statistical Analyses*

While the association between SES and age-specific mortality rates could be examined directly, interpreting the consequences of differences in mortality rates over a large range of ages is challenging. As an alternative, organizations such as the World Bank, United Nations, and World Health Organization measure mortality using summary measures of mortality that are based on the period life table. I followed this approach and present SES differences in two commonly used measures of adult mortality: life expectancy at age 30 ( $e_{30}$ ) and the probability of dying between the ages of 30 and 60 ( ${}_{30}q_{30}$ ). While both measures are derived from the period life table,  $e_{30}$  measures mortality over all adult ages while  ${}_{30}q_{30}$  specifically measures mortality in the working, productive, ages. To construct  $e_{30}$  and  ${}_{30}q_{30}$ , I first estimated age-specific mortality rates using a discrete failure time regression model. Next, I used the estimated rates to construct a life table starting at age 30 separately by sex, urban/rural residence, and by SES. Finally, I present  $e_{30}$  and  ${}_{30}q_{30}$  for each group based on the constructed life tables (Details on the estimation are presented in Appendix B).

To understand the contribution of major risk factors to SES differences in adult mortality, I first calculated the age-standardized prevalence of each risk factor across both consumption and wealth quartiles. The goal of this analysis was to describe how levels of each risk factor vary across SES groups. I then estimated the discrete failure time regression mortality model used to estimate the period life tables, additionally adjusting for obesity, hypertension, and tobacco use. The change in the estimated odds of mortality across SES quartiles between the unadjusted and adjusted models reveals the contribution of the three

risk factors to differences in adult mortality across SES quartiles.

#### *1.4.4. Sample Size and Missingness*

In 2007, 18,740 target respondents between the ages of 30 and 80 were interviewed. Of these, individuals, 629 were missing information on household expenditure per capita and additional 186 were missing information on household assets, for a final baseline 2007 sample of 17,925 individuals (95.7% of the eligible sample). The IFLS had exceptional mortality follow-up: by 2015 the mortality status of all of the 17,925 individuals was known, with 1,443 individuals dying between waves. Since information on tobacco use, alcohol consumption, and obesity was only measured for a subset of the total sample, the sample size for the secondary analyses was limited to 16,230 individuals.

All analyses were conducted in STATA 13.

### 1.5. Results

Table 1.1 presents the age, death, and urban-rural distribution of the sample in the year 2007 for men and women separately. For both men and women, the sample is concentrated in the younger ages, with 21% of men and 20% of women falling in the 30-35-year age group. Although deaths occurred at all age groups, the majority of deaths are unsurprisingly clustered at older ages (between ages 60 and 80) for both sexes. Based on these data, life expectancy at age 30 is 43.9 years for women and 41.2 years for men. These estimated life expectancies are close to the World Health Organization published estimates for 2012 (Appendix Figure 1.1). Finally, among both men and women, 53% of individuals live in urban areas.

#### *1.5.1. Female Mortality by Urban/Rural Residence and SES*

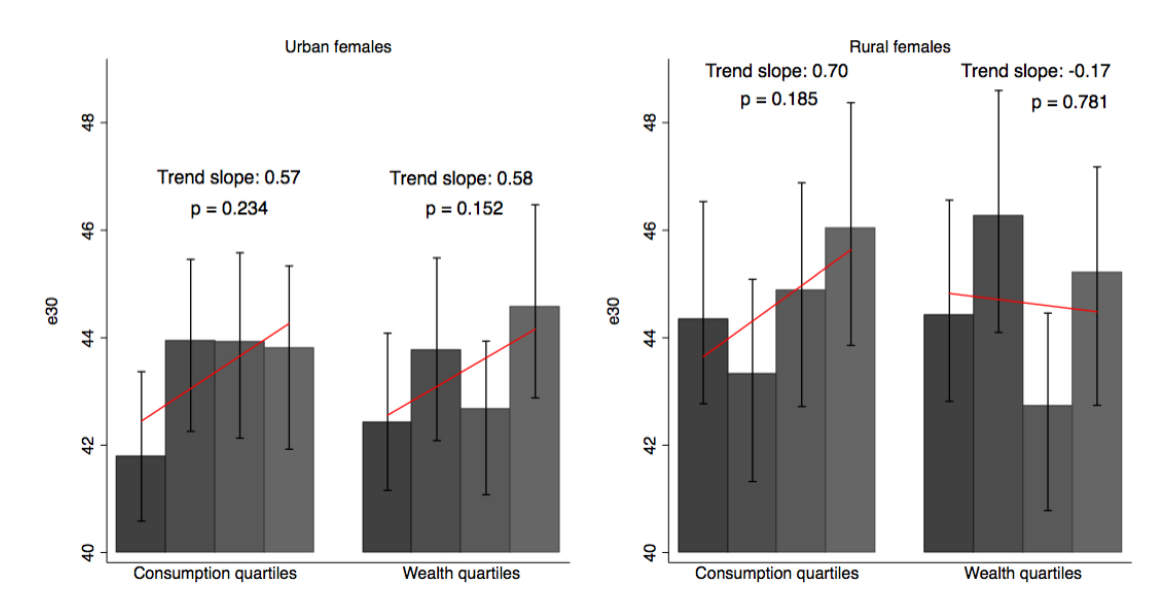
Figure 1.1 shows life expectancy at age 30 across consumption and wealth quartiles for urban and rural women. I find weak evidence of a relationship between SES and life expectancy for women in both urban and rural areas. For example,  $e_{30}$  for urban women increases by an average of 0.57 years per quartile (trend  $p = 0.234$ ) from 41.8 years (95% CI: 40.6, 43.4)

Table 1.1: Age, death, and urban-rural distribution, Indonesian Family Life Survey, 2007

	Females				Males			
	N	%	Deaths	%	N	%	Deaths	%
<b>Age group</b>								
30-35	1788	0.20	23	0.03	1862	0.21	33	0.04
35-40	1545	0.17	29	0.04	1678	0.19	44	0.06
40-45	1305	0.14	43	0.06	1260	0.14	47	0.06
45-50	1170	0.13	52	0.08	1085	0.12	66	0.09
50-55	949	0.10	63	0.09	851	0.10	93	0.12
55-60	679	0.07	74	0.11	659	0.08	86	0.11
60-65	560	0.06	92	0.13	462	0.05	95	0.13
65-70	558	0.06	158	0.23	452	0.05	137	0.18
70-75	368	0.04	113	0.16	283	0.03	114	0.15
75-80	242	0.03	46	0.07	169	0.02	35	0.05
Total	9164	1	693	1	8761	1	750	1
Life expectancy at age 30	43.9	-	-	-	41.2	-	-	-
Urban	4872	0.53	-	-	4627	0.53	-	-

Notes: Age group for deaths are presented as the age of the deceased at the time of the survey in 2007. The actual age of death may fall in the adjacent age group. Life expectancy at age 30 was estimated in the IFLS using life tables created from a discrete-failure time longitudinal regression model with a Gompertz age hazard.

Figure 1.1: Life expectancy at age 30 across consumption and wealth quartile, Female, Indonesian Family Life Survey, N = 9,164, 2007-2015



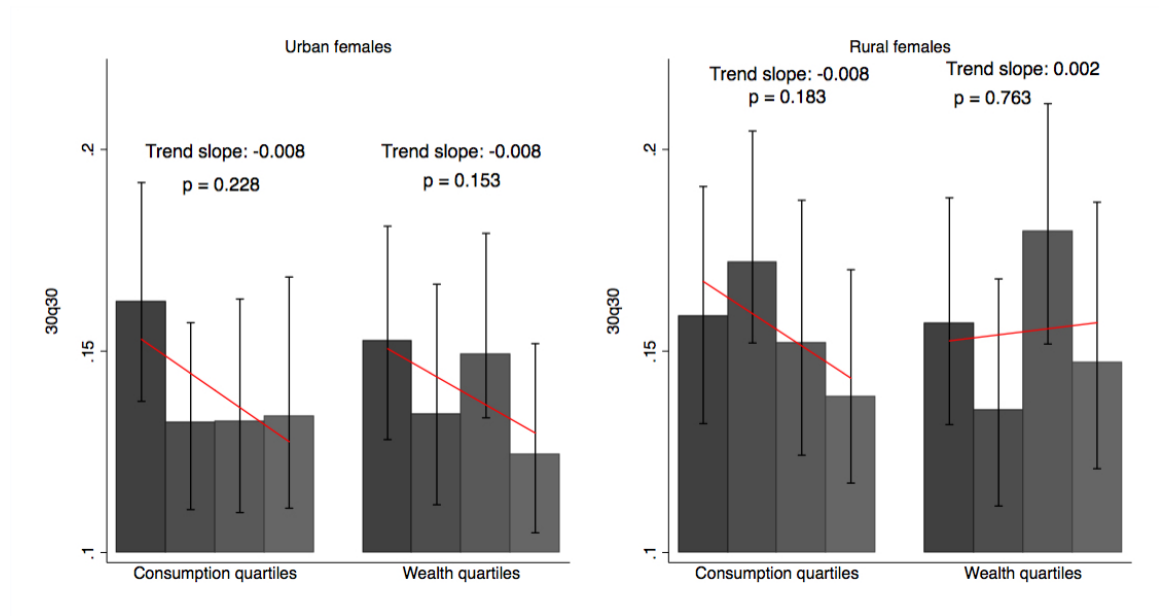
for women in the bottom consumption quartile to 43.8 years (95% CI: 41.9, 45.3) for women in the top quartile. There are similar weak trends across wealth quartiles in urban areas and consumption in rural areas. In contrast, I find virtually no evidence of an association between wealth quartile and life expectancy in rural areas (trend  $p = 0.781$ ).

Figure 1.2 shows trends in  $_{30}q_{30}$  for urban and rural women. The patterns are very similar to those observed for  $e_{30}$ , with marginal evidence of small a downward trend in the probability of dying in the working ages (0.8 percentage point decrease per quartiles) across consumption quartiles for three of the four SES-urban/rural groups (urban consumption  $p = 0.228$ , urban wealth  $p = 0.153$ , rural consumption  $p = 0.183$ ).

### 1.5.2. Male Mortality by Urban/Rural Residence and SES

Figure 1.3 shows  $e_{30}$  for urban and rural men by the two measures of SES. In contrast to women, there is around a three-year difference in life expectancy at age 30 across wealth quartiles, but not consumption quartiles for both urban and rural men. For urban men,  $e_{30}$  increases by an average of 1.1 years per quartile ( $p = 0.014$ ) from 38.7 years (95% CI: 37.4,

Figure 1.2: Probability of dying between ages 30 and 60 across consumption and wealth quartile, Females, Indonesian Family Life Survey, N = 9,164, 2007-2015



40.5) for men in the bottom wealth quartile to 42.1 years (95% CI: 40.3, 44.1) in the top wealth quartile; for rural men,  $e_{30}$  increases by an average of 1.35 years per quartile ( $p = 0.007$ ) from 40.6 years (95% CI: 39.2, 42.5) for men in the bottom wealth quartile to 44.3 years (95% CI: 42.4, 46.6) in the top wealth quartile. In sharp contrast, there is no evidence of a trend in  $e_{30}$  across consumption quartiles (trend  $p$ -value = 0.736 for urban men and 0.311 for rural men).

Figure 1.4 shows  $30q30$  by SES quartiles for urban and rural men. Similar to  $e_{30}$  the gradient in the probability of dying between the ages of 30 and 60 is pronounced and present for both urban and rural men across wealth, but not consumption, quartiles. For example, on average the probability of dying in the working ages decreases by around 2 percentage points per quartile in both urban and rural areas (urban  $p = 0.013$ , rural  $p = 0.006$ ) resulting in a 6 percentage point lower probability of dying for top compared to bottom SES quartile men.

Aside from SES differences, I find evidence that adult mortality is actually slightly higher in urban, compared to rural, areas for both women and men (Figures 1.1-1.4). For



Figure 1.3: Life expectancy at age 30 across consumption and wealth quartile, Males, Indonesian Family Life Survey, N = 8,761, 2007-2015

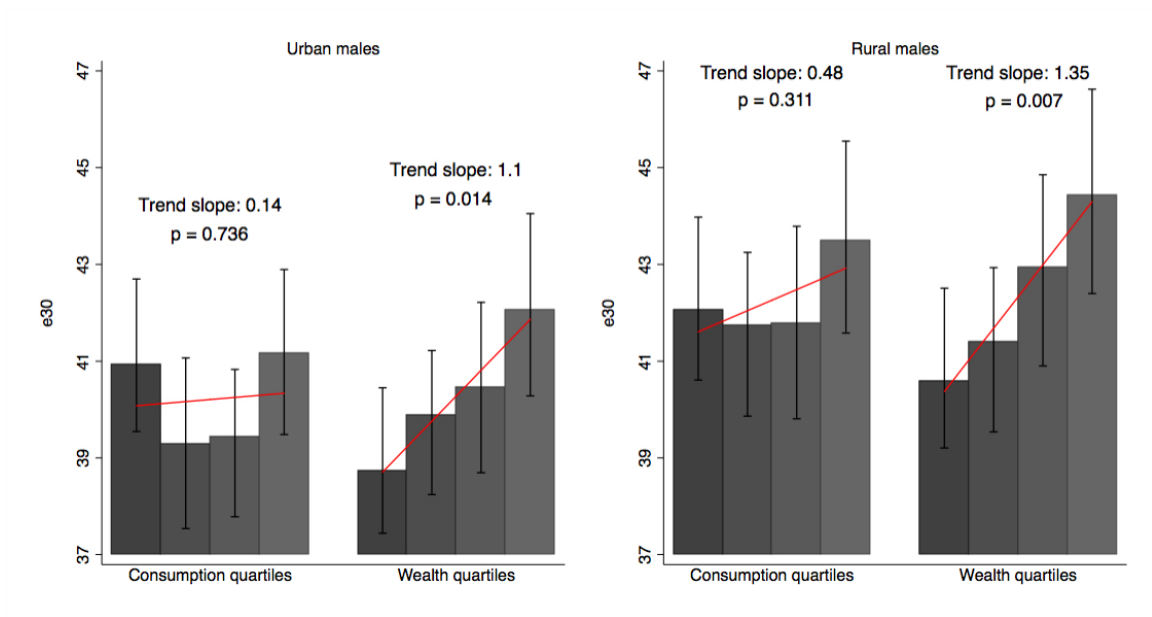
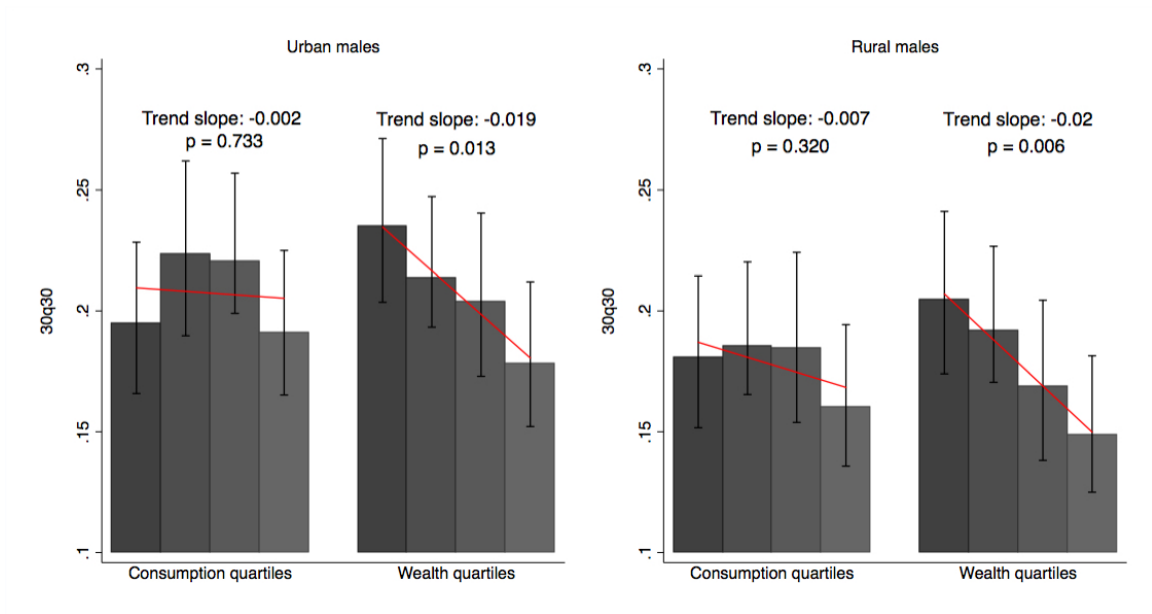


Figure 1.4: Probability of dying between ages 30 and 60 across consumption and wealth quartile, Males, Indonesian Family Life Survey, N = 8,761, 2007-2015



example,  $e_{30}$  is around 2-3 years greater for rural women ( $e_{30}$  ranges between 42 and 44 for urban women but between 41 and 46 for rural women). Similarly,  $e_{30}$  is around 1.5-2 years greater for rural men ( $e_{30}$  ranges between 39 and 42 for urban men but between 41 and 44 for rural men).

### *1.5.3. The Role of Risk Factors*

Table 1.2 presents the age-standardized prevalence estimates of tobacco use, obesity, and hypertension across wealth and consumption quartiles by sex. In general, the patterning of risk factors is varied, with some risk factors being higher among high SES groups while others are higher among low SES groups. For example, tobacco use decreases across wealth quartiles for both men and women: tobacco use moves from 80% (95% CI: 78%, 82%) for men in the bottom wealth quartile to 71% (69%, 73%) for men in the top wealth quartile and from 8.3% (95% CI: 6.8%, 9.3%) for women in the bottom wealth quartile to 4.9% (95% CI: 3.8%, 6.1%) for women in the top wealth quartile. Importantly, although there is evidence of a gradient for tobacco for men, overall levels are extremely high for all SES groups. In contrast to tobacco use, obesity moves in a reverse direction, increasing across both consumption and wealth quartiles for both men and women: for example, obesity moves from 7.3% (95% CI: 6.1%, 8.6%) for women in the bottom consumption quartile to 11% (95% CI: 9.8%, 13%) for women in the top consumption quartile and from 1.4% (95% CI: 0.83%, 1.9%) for men in the bottom consumption quartile to 5.9% (95% CI: 4.8%, 7.0%) for men in the top quartile. Finally, hypertension is very prevalent and largely similar across all sex-SES groups (between 33-41%). The one exception is across consumption quartiles for men, with a higher level of hypertension for men in the top quartile (39%, 95% CI: 37%, 41%) compared to men in the bottom quartile (33%, 95% CI: 30%, 35%).

Table 1.2: Age-standardized prevalence of obesity, hypertension, and tobacco use across consumption and wealth quartiles, Indonesian Family Life Survey, 2007

Females (N = 8,382)				Males (N = 7,763)		
	Obesity	Hypertension	Tobacco use	Obesity	Hypertension	Tobacco use
<b>Consumption Quartiles</b>						
1	0.073 (0.061,0.086)	0.39 (0.37,0.41)	0.054 (0.043,0.064)	0.014 (0.0083,0.019)	0.33 (0.30,0.35)	0.79 (0.77,0.81)
2	0.068 (0.056,0.080)	0.4 (0.38,0.42)	0.056 (0.045,0.067)	0.022 (0.015,0.030)	0.34 (0.31,0.36)	0.75 (0.73,0.77)
3	0.091 (0.078,0.10)	0.4 (0.38,0.42)	0.063 (0.052,0.074)	0.03 (0.022,0.038)	0.36 (0.34,0.39)	0.77 (0.75,0.79)
4	0.11 (0.098,0.13)	0.4 (0.38,0.42)	0.057 (0.046,0.068)	0.059 (0.048,0.070)	0.39 (0.37,0.41)	0.73 (0.71,0.76)
<b>Wealth Quartiles</b>						
1	0.069 (0.058,0.080)	0.41 (0.39,0.43)	0.08 (0.068,0.093)	0.024 (0.016,0.032)	0.35 (0.33,0.38)	0.8 (0.78,0.82)
2	0.081 (0.069,0.093)	0.39 (0.37,0.41)	0.059 (0.048,0.069)	0.022 (0.016,0.029)	0.34 (0.32,0.37)	0.79 (0.77,0.81)
3	0.085 (0.073,0.097)	0.4 (0.38,0.43)	0.052 (0.041,0.062)	0.029 (0.021,0.036)	0.34 (0.32,0.36)	0.77 (0.75,0.79)
4	0.1 (0.090,0.12)	0.39 (0.37,0.41)	0.049 (0.038,0.061)	0.042 (0.033,0.051)	0.37 (0.35,0.39)	0.71 (0.69,0.73)

Notes: Prevalence estimates were estimated using the overall population distribution as the standard. Obesity and hypertension were based on measured height, weight, and blood pressure; tobacco use was self-reported.

Table 1.3 presents regression estimates for the association between SES and adult mortality for women, before and after adjusting for tobacco use, obesity, and hypertension. Consistent with Figure 1, there is weak evidence of a relationship between SES and mortality; furthermore, adjusting for differences in adult risk factors has negligible effects on the size of the estimated associations. For example, the odds ratio of the top consumption quartile relative to the bottom actually becomes stronger, moving from 0.827 (95% CI: 0.581, 1.178) to 0.796 (95% CI: 0.558, 1.135). This pattern is consistent across the rest of the models, with very little change in the estimated SES coefficients after adjusting for obesity, hypertension, and tobacco use.

Table 1.4 presents the same set of regression estimates for men. Similar to women, adjusting for differences in obesity, hypertension, and tobacco use has negligible effects on the estimated associations, with the association becoming stronger in some cases. For example, the odds ratio for mortality for rural men in the top wealth quartile compared to bottom quartile is 0.706 (95% CI: 0.500, 0.998); after adjusting for adult risk factor, the relationship remains stable with an odds ratio of 0.704 (95% CI: 0.498, 0.995). Although the estimated association between wealth and mortality for urban becomes slightly weaker (unadjusted OR: 0.767, 95% CI: 0.548, 1.073; adjusted OR: 0.777, 95% CI: 0.554, 1.091) the overall trend of lower mortality in the higher wealth quartiles remains consistent.

Table 1.3: Association between SES quartiles and mortality adjusting for obesity, hypertension, and tobacco use, Females, Indonesian Family Life Survey, 2007-2014

Outcome: Mortality	Urban				Rural			
	Consumption	Wealth	Consumption	Wealth	Consumption	Wealth	Consumption	Wealth
	Model 1	Model 2	Model 1	Model 2	Model 1	Model 2	Model 1	Model 2
SES Quartiles								
2	0.898 (0.642,1.257)	0.880 (0.629,1.232)	0.853 (0.618,1.175)	0.874 (0.634,1.206)	1.174 (0.835,1.652)	1.157 (0.822,1.628)	0.929 (0.657,1.313)	0.928 (0.656,1.314)
3	0.921 (0.658,1.289)	0.899 (0.642,1.259)	0.903 (0.652,1.251)	0.909 (0.656,1.260)	0.993 (0.696,1.416)	0.972 (0.681,1.388)	1.183 (0.847,1.652)	1.211 (0.866,1.693)
4	0.827 (0.581,1.178)	0.796 (0.558,1.135)	0.585*** (0.393,0.869)	0.593*** (0.399,0.883)	0.961 (0.672,1.375)	0.937 (0.654,1.342)	0.967 (0.669,1.399)	0.997 (0.688,1.446)
Age	1.101*** (1.090,1.113)	1.095*** (1.082,1.107)	1.100*** (1.089,1.112)	1.094*** (1.081,1.106)	1.082*** (1.071,1.094)	1.074*** (1.062,1.086)	1.083*** (1.071,1.094)	1.074*** (1.062,1.086)
Obese		0.998 (0.658,1.515)		1.006 (0.664,1.524)		1.212 (0.683,2.150)		1.185 (0.668,2.102)
Hypertensive		1.652*** (1.246,2.188)		1.632*** (1.231,2.162)		1.546*** (1.182,2.022)		1.554*** (1.188,2.032)
Ever used tobacco		0.747 (0.450,1.239)		0.763 (0.460,1.265)		1.464** (1.034,2.073)		1.479** (1.043,2.098)
Person-year observations	29,856	29,856	29,856	29,856	26,773	26,773	26,773	26,773

\*\*\* p<0.01, \*\* p<0.05, \* p<0.1

Results are presented as odds ratios with 95% confidence intervals in parentheses.

Table 1.4: Association between SES quartiles and mortality adjusting for obesity, hypertension, and tobacco use, Males, Indonesian Family Life Survey, 2007-2014

Outcome: Mortality	Urban				Rural			
	Consumption	Wealth	Consumption	Wealth	Consumption	Wealth	Consumption	Wealth
	Model 1	Model 2	Model 1	Model 2	Model 1	Model 2	Model 1	Model 2
SES Quartiles								
2	1.188 (0.869,1.625)	1.227 (0.896,1.679)	0.875 (0.646,1.187)	0.881 (0.649,1.195)	1.086 (0.789,1.493)	1.084 (0.787,1.492)	0.896 (0.649,1.238)	0.911 (0.659,1.259)
3	1.140 (0.830,1.567)	1.108 (0.806,1.524)	0.909 (0.670,1.235)	0.935 (0.688,1.271)	0.977 (0.702,1.359)	0.973 (0.699,1.356)	0.817 (0.589,1.134)	0.826 (0.595,1.147)
4	1.124 (0.817,1.545)	1.104 (0.801,1.521)	0.767 (0.548,1.073)	0.777 (0.554,1.091)	0.809 (0.572,1.143)	0.775 (0.547,1.098)	0.706** (0.500,0.998)	0.704** (0.498,0.995)
Age	1.095*** (1.084,1.105)	1.089*** (1.079,1.100)	1.094*** (1.084,1.104)	1.088*** (1.078,1.099)	1.093*** (1.082,1.104)	1.086*** (1.075,1.098)	1.092*** (1.082,1.103)	1.086*** (1.075,1.098)
Obese		1.054 (0.583,1.904)		1.071 (0.594,1.933)		1.380 (0.600,3.177)		1.330 (0.580,3.048)
Hypertensive		1.686*** (1.332,2.134)		1.694*** (1.339,2.143)		1.781*** (1.384,2.293)		1.752*** (1.362,2.255)
Ever used tobacco		1.407*** (1.086,1.822)		1.371** (1.058,1.778)		0.839 (0.624,1.128)		0.816 (0.607,1.097)
Person-year observations	26,855	26,855	26,855	26,855	25,354	25,354	25,354	25,354

\*\*\* p<0.01, \*\* p<0.05, \* p<0.1

Results are presented as odds ratios with 95% confidence intervals in parentheses.

One important finding is that across every sex-urban group, hypertension increases the odds of mortality substantially (OR between 1.546 to 1.781, all  $p < 0.01$ ), while tobacco use increases the odds of mortality for rural women (adjusted ORs between 1.464-1.479,  $p < 0.05$  for both) (Tables 1.3 and 1.4).

#### *1.5.4. Robustness*

My conclusions are dependent on the accuracy of the mortality model. In Appendix Figure 1.1, I show the fit of the discrete failure time mortality model against the raw mortality rates and find that the estimated mortality rates track very closely with the observed levels. I also compare the shape and levels of my estimated mortality to the World Bank life table for Indonesia and find that the mortality estimates are extremely close to the World Bank life table (the around 1.5-year difference is likely partially the result of the fact that my estimates and the WHO estimates correspond to different time periods). My estimates of mortality controlling for the risk factors may be biased if the small number of individuals who were dropped between the main and secondary analyses were disproportionately more or less likely to die. In Appendix Figure 1.2, I graph the survival curves and estimated life expectancies at age 30 for the total eligible sample, the main analytic sample, and the sub-sample used to estimate risk factor contributions. I find virtually no difference in the levels of mortality between the eligible and main sample although there is some evidence that mortality for the sub sample is somewhat lower compared to the eligible and main samples. However, this bias would result in conservative estimates of the contribution of risk factors, since the risk factors are evenly distributed across SES quartiles.

My classification of per capita household expenditure assumes no economies of scale within households; as an alternative, some researchers propose using an “equivalenced” measure that divides total expenditure by the square root of the number of family members; similarly, the creation of the wealth index may be sensitive to the input variables used. Similarly, the results may be sensitive to the categorization of per capita expenditure and the wealth index. However, I find no substantive changes to my conclusions when using

alternative classifications (equivalence per capita expenditure for consumption and an only asset-based wealth index for the wealth quartiles) or quintiles rather than quartiles.

## 1.6. Discussion

Although reducing health inequalities is a major global policy priority, the size of the relationship between SES and adult life expectancy in developing countries is not well known. Using recent data from the Indonesian Family Life Survey, I find that the relationship between SES and adult mortality in Indonesia is complex, and depends on sex and the type of SES measure used. For both rural and urban men, there is a modest association between SES and adult life expectancy across an asset-based wealth index, but not across expenditure quartiles. While surprising, other studies from developing countries have found similar results. For example, Ardington and Gasealahwe (2014) use longitudinal individual level data on adults in South Africa and find an association between assets and two-year mortality among adults over the age of 20; however, they fail to find a relationship between household per capita expenditure and mortality. Similarly, Opuni et al (2011) estimate concentration indices for mortality in Tanzania and find no evidence of income inequality in adult mortality. These findings suggest that differences in consumption alone may not be sufficient to produce differences in mortality for men. The finding of an association across wealth but not consumption also suggests that differences in mortality are not driven by the short-term fluctuations in the ability to purchase better health but rather by longer-term socioeconomic disadvantage. In contrast to men, I only find weak evidence of an SES-mortality relationship for women. These results suggest that greater availability of resources for women does not necessarily translate into better health.

Second, I find that SES differences in three of the four leading risk factors for adult mortality (tobacco use, obesity, and hypertension) do not explain differences in adult mortality across SES quartiles. The patterning of risk factors is inconsistent across SES quartiles: tobacco use is slightly lower among high SES individuals compared to individuals in the lower quartiles; in contrast, the prevalence of obesity increases as SES increases, while hyperten-



sion has similar levels across all groups with some evidence of higher levels of hypertension among higher SES groups. Unsurprisingly, I find that adjusting for differences in these risk factors across SES groups does not explain away the association between SES and mortality—in some cases the association becomes even stronger. This finding is consistent with studies of the prevalence of NCDs and their risk factors in developing countries, which often find either inconsistent associations between SES and NCD risk factors or higher levels of risk factors like obesity among high SES groups relative to low SES groups [2, 3, 14, 32]. Tobacco use, however, may explain the differences in life expectancy between men and women. Given the large overall differences in tobacco use between men and women, non-communicable diseases related to these risk factors (such as heart disease and stroke) are strong candidate explanations for the sex inequalities in life expectancy. This hypothesis is consistent with findings from developed countries that implicate smoking as a primary reason for sex differences in life expectancy [7, 42]. Reducing tobacco use among Indonesian men may provide a promising strategy for improving male life expectancy.

One unexpected finding was that adult mortality is actually higher in urban, compared to rural, areas. Given the large burden of mortality attributable to risk factors such as obesity and tobacco use, urban-rural differences in mortality may be partly driven by the observed difference in risk factors [11]. This difference may also arise if sicker individuals are more likely to move to urban areas to seek healthcare. More research is needed to better understand urban-rural and other geographic differences in mortality within Indonesia.

My findings contrast with the literature on SES and adult life expectancy in developed countries and suggest that insights on the relationship between SES and adult mortality from developed countries may not generalize to developing countries. First, the overall size of the association between SES and adult life expectancy in Indonesia is modest compared to the United States. While recent studies from the United States find between 6-10 year differences in adult life expectancy, differences between the top and bottom quartiles in Indonesia only range from 2-4 years. Furthermore, SES differences in adult mortality are

not always present: I find very little evidence of SES differences for women. Patterning of risk factors may drive the modest to null differences across groups across SES groups. For example, in developed countries, mortality inequality is linked to greater adoption of poor health behaviors and risk factors such as smoking and unhealthy weight among the poor compared to the rich [10]. In contrast, while I observe a small gradient for men in tobacco use, overall levels remain extremely high across all levels of income for men. Similarly, levels of hypertension are high and present for all sex-SES groups. Given the large and significant relationship between hypertension and the odds of mortality for all sex-urban-SES groups, my results suggest that the widespread prevalence of mortality risk factors in Indonesia may result in smaller SES differences in adult mortality. Expansions of mortality inequality in developed countries has also been linked to the introduction of medical technologies and care, since higher SES individuals tend to benefit more from new health technologies [8, 22]. The lack of pronounced inequalities in Indonesia, especially for rural women, may result from a general lower quality or availability of medical services in more remote contexts [6, 31]. Further research measuring health-seeking behavior across SES groups would provide greater insights into the mechanisms behind the observed patterns.

The study has some important limitations. Many studies have shown that individuals in developing countries may misreport their age [19]. While the IFLS attempts to provide a best guess age for each individuals, if poorer individuals were less likely to know their correct age, the estimated difference between poor and rich individuals may be biased downward by the measurement error introduced into the mortality estimates for the poor [41]. While height, weight, and blood pressure was measured by the IFLS, tobacco use was self-reported. The estimated gradients and the contribution of tobacco use to mortality may therefore be biased if some groups misreport their tobacco use. Both socioeconomic status and risk factors were measured for one point in time (the 2007 survey); evidence from other studies has shown that the timing and duration of SES and risk factors plays an important role in the relationship between individual characteristics and mortality. Without information on the duration, my estimates of SES differences and the contribution of each risk factor may

be biased. Further studies would greatly benefit from multiple measurements of individual characteristics. There is potential for reverse causality between SES and health, where poor health leads to low SES; however, this would bias estimated gradients upward and therefore not affect my conclusions. Finally, the results of this study are not causal but rather measure the association between SES and life expectancy. The true causal effects of SES on life expectancy are likely smaller than the estimated association since the same characteristics that determine high SES may also produce better levels of health.

Overall, my results suggest that higher consumption or wealth is not always associated with higher life expectancies in a developing country. Health resource constraints or the high prevalence of tobacco use and hypertension across all groups may overshadow SES differences. As a next step, health policy in developing countries needs to address the high burden of NCD risk factors while also working to identify context specific correlates of longevity.

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## CHAPTER 2 : Body Mass Index and Blood Pressure in a Low-Obesity Context: a Longitudinal Fixed-Effects Study of Indonesian Adults

### 2.1. Abstract

#### **Background**

The prevalence of hypertension is very high in Indonesia and many other developing countries. While obesity is a major risk factor for hypertension, levels of obesity in many developing countries are low. An important question is whether weight is an etiologic factor for blood pressure among leaner individuals.

#### **Methods**

Using longitudinal data on Indonesian adults over a 17-year period, I estimate the relationship between BMI and blood pressure using fixed-effects models. By comparing within-individual changes in BMI and blood pressure, this approach adjusts for both observed and unobserved time-invariant confounders.

#### **Results**

In fully adjusted models, there is a positive relationship between BMI changes and blood pressure changes: a one unit increase in BMI is associated with an 1.7 (95% CI: 1.5,1.9;  $p < 0.001$ ) unit increase in systolic blood pressure for men and an 1.3 (95% CI: 1.2,1.5;  $p < 0.001$ ) unit increase for women. In stratified models, BMI changes are associated with larger blood pressure changes in the lower BMI ranges: a one unit increase in BMI for men is associated with a 2.2 (95% CI: 1.7,2.7;  $p < 0.001$ ) unit increase in blood pressure in the 15-20 BMI range, a 1.3 (95% CI: 1.0,1.6;  $p < 0.001$ ) unit increase in the 20-25 range, and a 1.2 (95% CI: 0.8,1.7;  $p < 0.001$ ) unit increase in the 25-30 range. The estimated patterns are similar for women.

#### **Conclusions**

In Indonesia, changes in BMI are related to changes in blood pressure for both men and women across all age groups and levels of BMI. These results suggest that BMI is still an important risk factor for blood pressure in lean populations.

## 2.2. Introduction

High blood pressure (hypertension) is the leading risk factor for mortality globally [32, 20]. Although hypertension is often believed to be more prevalent in developed countries, rates of hypertension in many developing countries are extremely high. For example, the age standardized prevalence of hypertension is 57.1% in Ghana, 32.3% in India, and 58.2% in Mexico [21]. In fact, recent estimates from the Global Burden of Disease study find a higher age standardized prevalence of hypertension in many developing compared to developed countries [29]. The high burden of hypertension in developing countries has prompted calls for action from health and policy experts globally [25, 16]. As the populations of developing countries continue to age, reducing rates of hypertension will be essential for promoting longevity. Understanding the modifiable causes of high blood pressure can provide needed policy solutions to address this challenge.

Obesity is one of the primary risk factors for hypertension [10, 13, 26]. In developed countries, many studies have found that obese individuals ( $\text{BMI} \geq 30$ ) have a higher risk of hypertension compared to normal weight individuals ( $18 \leq \text{BMI} < 25$ ), even after adjusting for a wide range of potential confounders [10, 3, 14]. In populations like the United States, the common approach of categorizing BMI into obese, overweight, and normal weight provides a useful diagnostic criterion for assessing blood pressure risk since a large fraction of the population is obese [23]. In contrast, the absolute levels of obesity in developing countries tend to be low, with prevalence rates well below 10% in many countries [17]. Given the combination of high blood pressure but low obesity, an important question is whether BMI is related to blood pressure at lower levels of weight. Unfortunately, most studies of BMI and blood pressure in developing countries also categorize BMI into broad categories [2, 21, 24], potentially covering up important relationships between weight and blood pressure in the lower BMI ranges. While some researchers address this limitation by estimating country specific BMI cut-points [28], a small literature using continuous measures finds a near linear relationship between BMI and blood pressure [9, 7, 8, 19, 15].

Evidence from these studies suggests that weight at lower levels of BMI may still be associated blood pressure; however, these results are from cross-sectional surveys and only adjust for observed confounders. If there are unobserved or omitted confounders, the linear relationship observed across individuals may not represent the effect of BMI on blood pressure. Whether the relationship between BMI and blood pressure in developing countries is robust to unobserved confounders remains unclear.

This study aims to fill this important gap in the literature. Using nationally representative longitudinal data over a 17 year period, I estimate the relationship between changes in BMI and changes in blood pressure within individuals in Indonesia. This approach provides a substantial improvement over cross-sectional studies by adjusting for time-invariant observed and unobserved confounders. I also estimate the size of BMI-blood pressure relationship for individuals at different levels of BMI to determine how changes in BMI at lower average levels of BMI compare with changes at higher BMI levels. Indonesia presents a strong context to study the BMI-blood-pressure relationship in developing countries since it is the third largest developing country, has very high levels of blood pressure, low levels of obesity, and is one of the only developing countries with multiple waves of longitudinal, nationally representative, survey data with measured biomarkers. Overall, the results of this study provide new evidence on the etiology of blood pressure in developing country contexts.

## 2.3. Background

### *2.3.1. BMI and blood pressure in developed countries*

Many studies using data from developed countries examine the relationship between BMI and blood pressure. Nearly every study categorizes BMI into standard groups (normal weight, overweight, and obese) and estimates the relative risk of high blood pressure for obese individuals relative to normal weight individuals. For example, two studies using data from the 1988 through 1994 waves of the National Health and Nutrition Examination Survey (NHANES) find that obese individuals are significantly more likely to have high

blood pressure, even after controlling for a wide range of covariates [3, 18]. Studies also examine this relationship from an incident hypertension perspective. Hu et al. (2004) use a large sample of Finnish adults to study the relationship between weight and incident hypertension over an 11-year period . They find that overweight and obese individuals are significantly more likely to become hypertensive over the period compared to normal weight individuals. Importantly this relationship is found at all levels of physical activity, suggesting that the estimates are not confounded by physical activity.

Evidence from randomized-control trials (RCTs) supports the observational relationship between weight and blood pressure: across nearly all RCTs, weight loss has a negative effect on blood pressure. For example, a trial of 2,382 individuals across nine medical centers in the United States finds that on average, a 4-6 kg reduction in weight among overweight individuals results in a 3-5 mmHg reduction in systolic blood pressure [27]. Other randomized control trials have found similar results for samples of obese individuals [31]. Indeed a meta-analysis of 25 RCTs testing the effect of weight reduction on blood pressure finds that on average, a reduction of 5.1 kgs of weight results in a 4.4 mmHg reduction in systolic blood pressure and a 3.6 mmHg reduction in diastolic blood pressure [22]. While these studies establish strong evidence for the effect of weight loss among overweight and obese individuals, none of the trials evaluate the effects in the lower weight ranges. Based on both observational and RCT evidence from developed countries, the relationship between weight change and blood pressure at lower weight ranges remains unclear.

### *2.3.2. BMI and blood pressure in developing countries*

A small set of observational studies from developing countries also examine the relationship between obesity and high blood pressure. For example, Basu et al. (2015) and Lloyd-Sherlock et al. (2014) use cross-sectional data from India, China, Russia, Ghana, South Africa, and Mexico to study correlates of hypertension. They find that in every country, obese individuals are significantly more likely to be hypertensive relative to normal weight individuals, even after controlling for potential socioeconomic confounders . Hussain et al.

(2016) and Qi et al. (2016) find similar results for China and Indonesia .

The evidence from these studies suggests that similar to developed countries, obesity is related to high blood pressure in developing countries. However, one prominent difference between many developed and developing countries is that rates of obesity tend to be lower in most developing countries. Therefore, studying the relationship between obesity and high blood pressure may ignore important determinants of blood pressure for the large subset of the population that is not obese. To address this limitation, a subset of papers from developing countries examine the continuous relationship between BMI and blood pressure rather than categorizing BMI. These studies find a near linear cross-sectional relationship between BMI and blood pressure among populations in Seychelles, Indonesia, and among smaller African study sites [9, 7, 8, 19, 15].

The existing literature from LMICs is limited by the categorization of BMI for many studies and by the cross-sectional approaches for all the studies. While, RCTs from developed countries establish a causal link between weight loss and blood pressure among overweight and obese individuals, there is no equivalent evidence for leaner individuals, especially in developing countries. The goal of this study is to address this important gap in the literature by using fixed-effects methods to study the relationship between within-individual changes and in BMI and blood pressure across the entire distribution of BMI. Since the estimated relationship is biologically mediated, the results are plausibly generalizable to a larger set of lean populations. Therefore, the results of this study can help to better understand the etiology of blood pressure in many contexts.

## 2.4. Data

### *2.4.1. Data*

Data are from the 1997, 2000, 2007, and 2014 waves of the Indonesian Family Life Survey (IFLS). The IFLS is a longitudinal survey of 11,000 households from 13 of Indonesia’s 27 provinces – making the IFLS representative of 83% of the Indonesian population. The IFLS is also one of the few surveys from a developing country with multiple waves of measured

biomarker and anthropometric health data, providing a rare source of directly measured information on how adult health has changed for individuals over time.

To select households, the IFLS first randomly sampled 321 enumeration areas based on Indonesia’s SUSENAS survey. Within each selected EA, between 200-300 households were randomly selected. For each identified household, information on household characteristics, household members, and household expenditure and consumption were provided by the household member most knowledgeable about household affairs. Individuals above the age of 30 were asked to provide information on self-reported health conditions; in addition, trained assessors collected measured blood pressure, height, and weight for each of these individuals. The sample was limited to only those individuals who had measured information in the 1997 wave (adults age 30 and above).

#### *2.4.2. Primary Outcomes*

The main outcomes are systolic and diastolic blood pressure (both measured continuously in mmHg). Blood pressure was measured in the household by a trained nurse using an Omron digital device with individuals in a sitting position. For the 2007 and 2014 waves, two separate measurements of blood pressure were collected; for these waves, I averaged the two measurements.

#### *2.4.3. Primary Exposure*

The primary exposure is measured body mass index (BMI-measured continuously). During the survey, the height and weight of each individual was measured by a trained assessor using a Shorr board and SECA 890 scale. Based on measured height and weight, I calculated each individual’s body mass index as weight in kilograms over height in meters squared.

#### *2.4.4. Other Covariates*

There are a number of important covariates that need to be adjusted for to reduce bias in the relationship between BMI and blood pressure (the following section discusses the role of these variables): age in years (based on self-reported age and/or birth date infor-



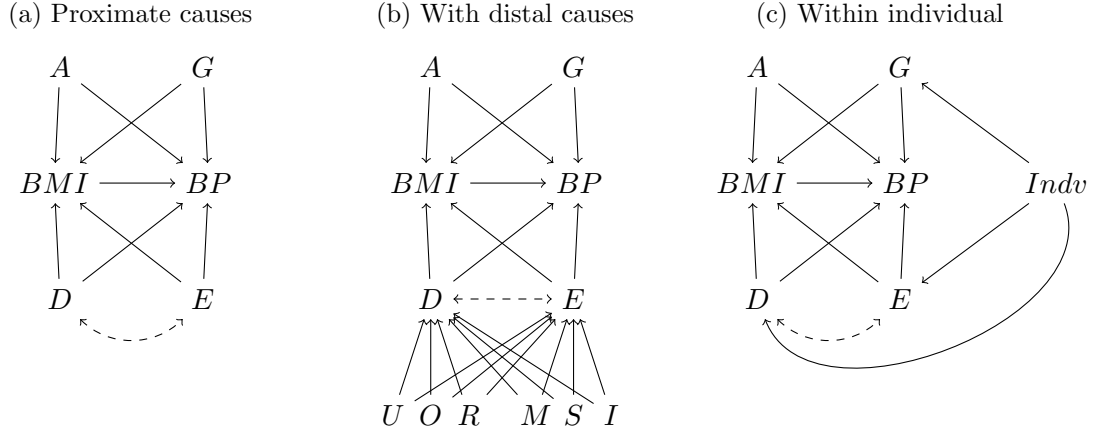
mation); self reported sex; urban or rural residence (based on census classification); survey wave; religion (grouped into Islam, Hindu, Protestant, and other); marital status (grouped into never married, current married, and formerly married); self reported occupation type (grouped into retail, manufacturing, agriculture, service, housewife, retired, and not working); self reported completed schooling (grouped into no schooling, some primary school, primary school or more); and household expenditure per capita (calculated as the sum of reported monthly household expenditure on housing, education, and food, and non-food goods divided by the number of individuals in the household).

#### *2.4.5. Causal Framework*

The causes of hypertension are poorly understood. Indeed, essential hypertension, or hypertension with no clearly identifiable cause, makes up an estimated 95% of hypertension cases [5]. However, both randomized trials and observational studies have identified a few likely causal risk factors for blood pressure: age (in most populations hypertension increases steadily over age), physical activity, diet (this includes both dietary salt consumption as well as specific nutrient deficiencies such as magnesium and potassium), excessive alcohol intake, genetic factors, psychosocial stress, and unhealthy weight [12, 5, 6]. Since, BMI shares many of these same causes, the unadjusted estimate of the relationship between BMI and BP will be a biased estimate of the effect of BMI on blood pressure. These causes, along with the direct effect of BMI on blood pressure can be represented by the graph in Figure 2.1, Panel A.

The two-sided dashed arrow represents the set of common causes of both diet and exercise, including social factors such as where a household lives, their income, or their religion. With valid and reliable measures of diet and exercise, the bias from observable confounders could be eliminated by conditioning on age, diet, and exercise. However, measures of diet and exercise are rare in large surveys and may not be reliable even when measured. One approach to reduce bias from poor or missing diet and exercise data is by conditioning on the common social causes of both diet and exercise, such as urban/rural residence, occupa-

Figure 2.1: Causal relationship between body mass index and blood pressure.



A: Age, G: Genetic Factors, D: Diet, P: Physical Activity, U: Urban/rural, O: Occupation, R: Religion, M: Marital Status, S: Schooling, I: Income

tion, religion, marital status, schooling, and income (Figure 2.1 Panel B). Importantly, this estimate may still be biased by other unobserved common causes of diet and exercise (the remaining dashed line between the two factors) and unobserved genetic factors.

Within-individual, or fixed-effect, models provide a strong way to reduce these sources of bias (Figure 2.1 Panel C). Assuming that genetic confounders do not vary over time, comparing changes in BMI to changes in blood pressure within individuals, would remove genetic confounding. Looking within individuals would also remove confounding from the time-invariant parts of diet and exercise. However, the fixed effects estimates may still be biased if there are time-variant unobserved confounders or measurement errors in the exposure and covariates.

#### 2.4.6. Methods

I first graph the age patterns of blood pressure and BMI to document the relationship between blood pressure, BMI, and age. I then graph the relationship between BMI in and blood pressure by 10 year age groups to visually examine the shape of the association across age-sex groups. Importantly, the results of this graph show the unadjusted association and do not adjust for common causes of BMI and blood pressure that confound the

relationship.

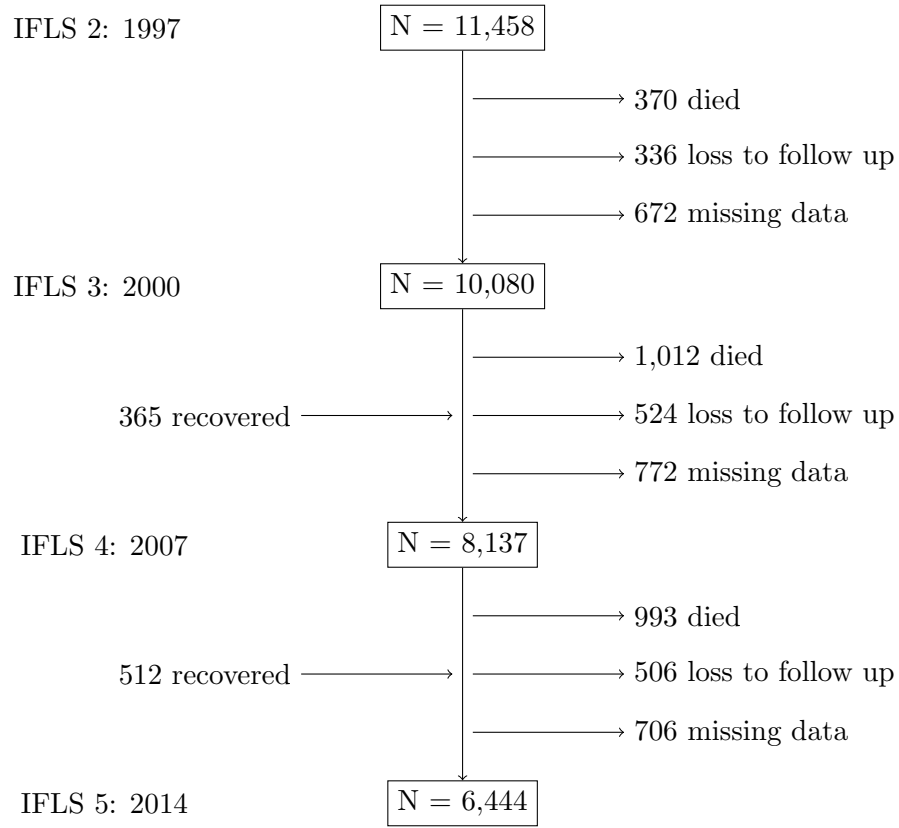
I use three separate models to adjust for potential confounders. First, I estimate an OLS regression that includes a quadratic age specification, a quadratic specification of household expenditure per capita, and dummy variables for each of the categories of schooling, religion, marital status, occupation, period of observation, and urban residence separately for men and women. Second, I use a non-parametric coarsened exact matching approach to group individuals in to strata defined by 5 year age groups, urban residence, schooling, and period of observation. I then estimate the same regression as above including dummy variables for each of the strata, as well as dummy variables for religion, job, and a continuous measure of income per capita. Importantly, both these models only condition on observed variables and may still be biased by unobserved confounders. To adjust for time-invariant confounders, I then estimate a within-individual fixed effects regression. To specifically identify if changes in weight are associated with changes in BMI at lower BMI levels, I stratify the fixed effect analyses by an individual's average BMI over the period, and re-estimate the models for each BMI group.

Since the data are drawn from a subset of the overall panel, appropriate survey weights were not available; however, omitting the survey weights would only affect the results if the probability of selection confounded the BMI-blood pressure relationship. Results are presented for systolic blood pressure since it is the primary source of hypertension in Indonesia; however, the overall conclusions for diastolic pressure are similar (Appendix E).

## 2.5. Results

Figure 2.2 shows the sample size at each wave and the mortality, attrition, and missing data per wave. In 1997, the sample consisted of 11,458 individuals with valid information on all variables. Over the 17 year period, the majority of individuals had valid information for at least two waves (10,519 individuals) with 5,695 individuals having information for all four waves of data. Individuals were not present in specific waves due to mortality, loss to follow up, and missing data, although the magnitude of these sources of attrition varied by

Figure 2.2: Sample selection, mortality, attrition, and missingness of the panel.



Total confirmed deaths = 2,375

Individuals present in at least 2 waves = 10,519

Individuals present in all waves = 5,695

wave.

Table 2.1 presents descriptive characteristics of the sample for the baseline 1997 wave. At baseline, mean levels of blood pressure are high despite normal to low levels of weight. For example, 34.3% of men and 37.3% of women were hypertensive at baseline; in sharp contrast, levels of obesity were extremely low for both men and women at only 1.0% for men and 4.3% for women. On average the sample started off middled aged (48.0 years for men and 47.3 years for women). The vast majority of individuals were married at baseline (93.9% for men and 74.0% for women) with Islam as the primary religion (87.7% of men and 87.1%

of women). Patterns of occupations differed for men and women. For men, agricultural work was the most common job type (35.4%) followed by other forms of employment (18.5%) and service sector work (13.7%). For women, house care was the most common occupation (34.8%), followed by agriculture (18.4%), and retail work (18.0%). Schooling levels also differed by sex: more than half of men had primary schooling or more (51.6%) compared to only 38.2% for women. A greater fraction of women also had no schooling compared to men (32.4% for women compared to 15.5% for men). Finally, the sample was slightly more concentrated in rural areas with 44.5% of men and 46.4% of women living in urban areas.

Figure 2.3 graphs the age-patterns of systolic blood pressure and hypertension by sex with 95% confidence intervals. For both men and women, systolic blood pressure increases steeply with age, although the growth is more pronounced for women than for men. For example, mean systolic blood pressure starts around 120 mmHg for both men and women at age 30 and increases to around 150 mmHg for men and around 160 mmHg for women. The prevalence of hypertension increases similarly, starting at below 20% for both men and women at age 30 and ending at around 60% for men and 80% for women in the oldest age group.

Figure 2.4 shows age patterns for BMI and obesity by sex with 95% confidence intervals. Weight displays a very different age pattern compared to blood pressure, peaking in the middle ages, then declining into the older ages. Women have a higher level of BMI and obesity compared to men at all ages. Despite the pronounced age-pattern, the mean levels of BMI stay well below the obesity threshold of  $\text{BMI} \geq 30$ , with obesity prevalence rates around 2% for men and between 2-9% for women.

Figure 2.5 graphs the smoothed relationship between BMI and systolic blood pressure for each 10-year age group. At every age, there is a near linear relationship between BMI and blood pressure, with no evidence of strong non-linearities above the obesity cut off. For example, for men and women between the ages of 50 and 60, systolic blood pressure

Table 2.1: Descriptive characteristics of the sample at baseline, Indonesian Family Life Survey, 1997, N = 11,458.

	Men (N = 5,183)		Women (N = 6,275)	
	Mean or %	SD or N	Mean or %	SD or N
<b>Blood Pressure</b>				
Systolic BP	131.7	22.2	132.9	27.6
Diastolic BP	80.9	12.1	80.9	13.5
Hypertensive	34.3	1777	37.3	2340
<b>Weight</b>				
BMI	21.2	3.1	22.3	3.9
Obese	1.0	53	4.3	270
<b>Sociodemographic Characteristics</b>				
Age	48.0	13.3	47.3	13.5
Marital Status				
Never married	2.2	114	2.6	163
Was married	3.9	204	23.4	1466
Currently married	93.9	4865	74.0	4646
Religion				
Islam	87.7	4544	87.1	5464
Hindu	5.5	284	5.1	318
Protestant	3.8	197	4.7	293
Other	3.0	158	3.2	200
Completed schooling				
No schooling	15.5	805	32.4	2034
Some schooling	32.9	1706	29.4	1843
Primary or more	51.6	2672	38.2	2398
Primary Job				
Retail	11.8	614	18.0	1127
Housewife only	0.5	24	34.8	2181
Retired	7.6	395	8.0	501
Agriculture	35.4	1836	18.4	1153
Manufacturing	10.0	520	7.3	457
Service	13.7	710	7.6	477
Not working	2.5	127	0.8	53
Other	18.5	957	5.2	326
Per capita expenditure	134276.4	328162.0	144040.2	480129.0
Urban	44.5	2309	46.4	2913

Notes: Hypertension was classified as a systolic blood pressure  $\geq 140$  mmHg or diastolic blood pressure  $\geq 90$  mmHg. Obesity was classified as a BMI  $\geq 30$ .

Figure 2.3: Age patterns of blood pressure and hypertension by sex, Indonesian Family Life Survey, 1997-2014. Data were pooled over the four survey years. N = 33,119. Errors bars show 95% confidence interval.

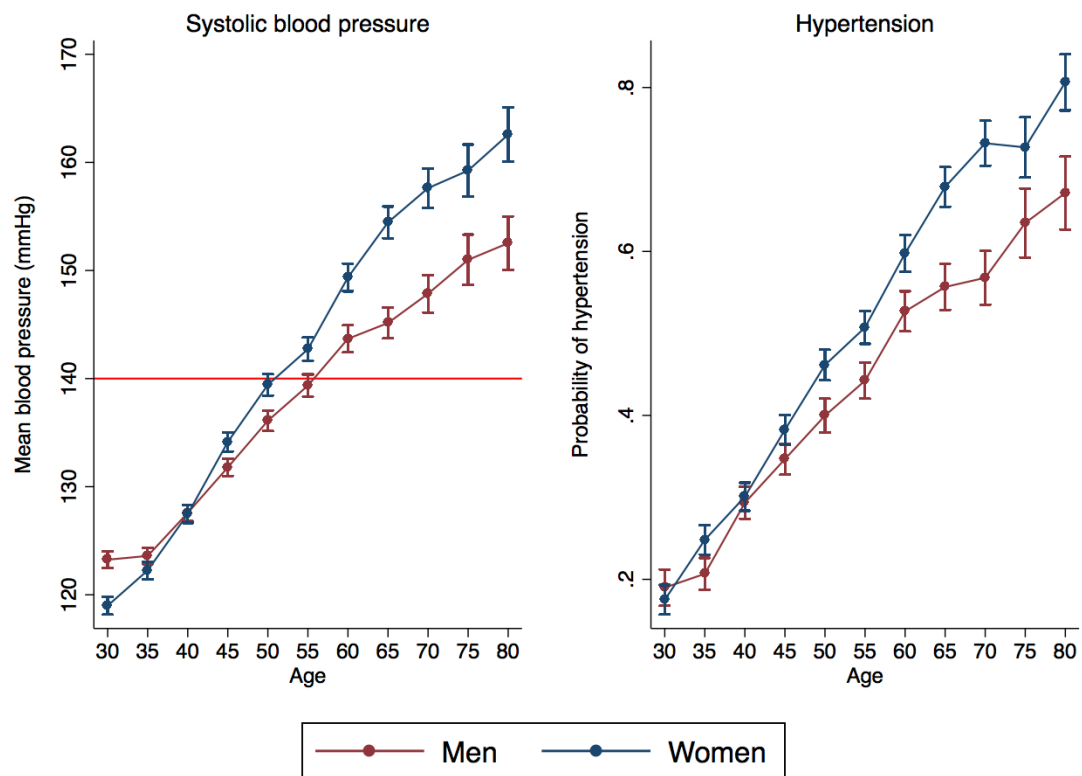


Figure 2.4: Age patterns of body mass index and obesity by sex, Indonesian Family Life Survey, 1997-2014. Data were pooled over the four survey years. N = 33,119. Errors bars show 95% confidence interval.

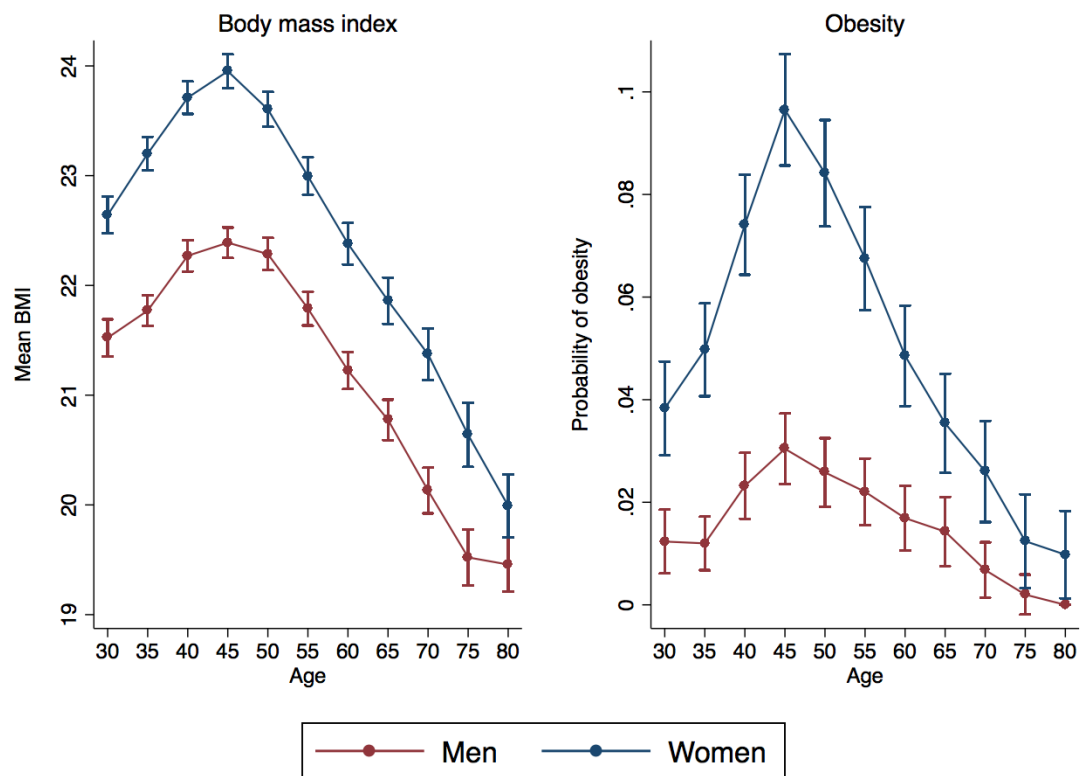
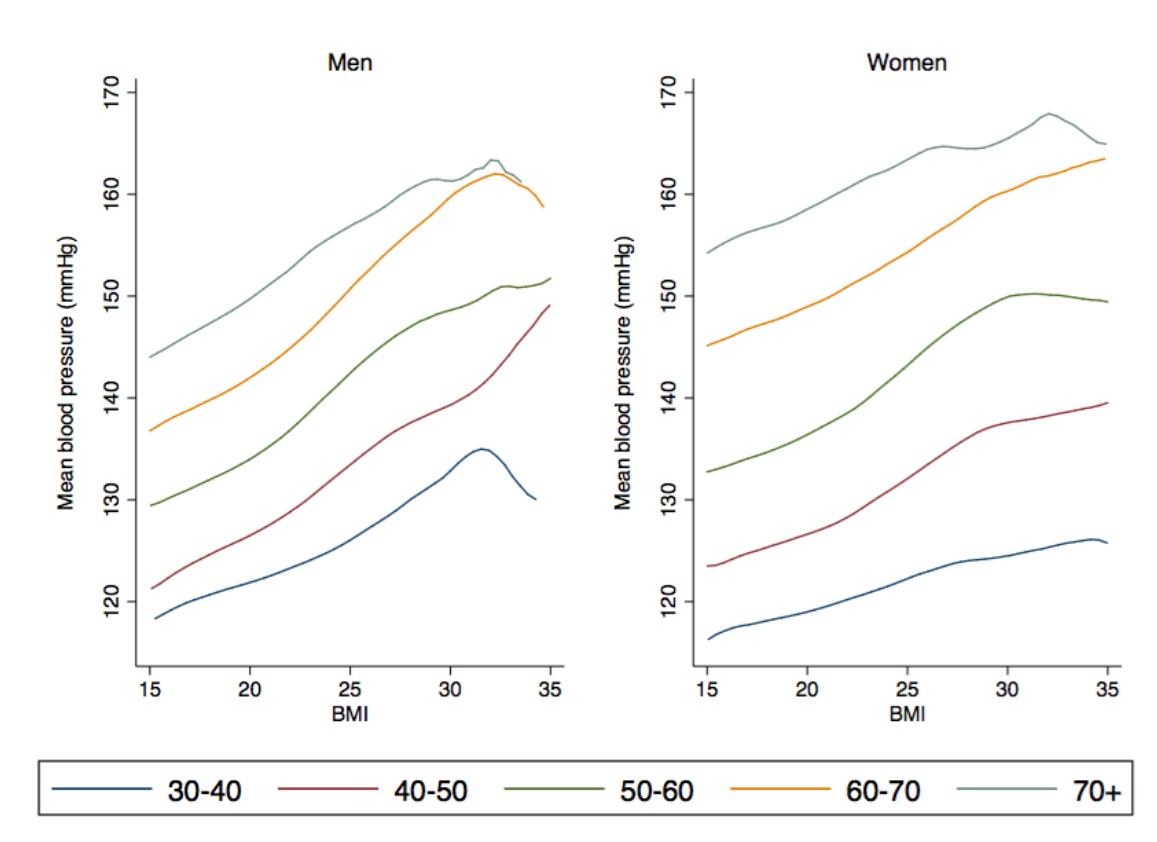




Figure 2.5: Relationship between body mass index and systolic blood pressure by sex and 10 year age groups, Indonesian Family Life Survey, 1997-2014. Data were pooled over the four survey years. Results are smoothed using a 2 unit moving average. N = 33,119



increases almost linearly from a BMI of 15 to a BMI of 35. This linear relationship is similar at each group despite the strong increase in blood pressure over age.

Table 2.3 shows the estimated relationship between BMI and systolic blood pressure adjusting for potential confounders using three different models. There is a pronounced relationship between BMI and systolic blood pressure that is very similar in size across the three model specifications. Within each model type, the size of the association is larger for men than for women. For example, based on the fixed-effects model, a one unit increase in BMI is associated with an 1.7 (95% CI: 1.5,1.9;  $p < 0.001$ ) unit increase in systolic blood pressure for men and an 1.3 (95% CI: 1.2,1.5;  $p < 0.001$ ) unit increase for women.

Table 2.2: Estimated relationships between BMI and blood pressure, Indonesian Family Life Survey, 1997-2014.

	Men		Men		Women		Women	
	Pooled OLS	Pooled OLS + CEM	Fixed Effects	Pooled OLS	Pooled OLS + CEM	Fixed Effects	Pooled OLS	Fixed Effects
Body mass index	1.8 (1.6 - 1.9)	1.8 (1.7 - 1.9)	1.7 (1.5 - 1.9)	1.3 (1.1 - 1.4)	1.3 (1.2 - 1.3)	1.3 (1.2 - 1.5)	1.3	1.3
Observations	16,121	16,121	16,121	19,998	19,998	19,998	19,998	19,998
R-squared	0.21	0.08	0.28	0.25	0.05	0.33	0.05	0.33
p-value	< 0.001	< 0.001	< 0.001	< 0.001	< 0.001	< 0.001	< 0.001	< 0.001
Overall Mean	135.4	135.4	135.4	137.8	137.8	137.8	137.8	137.8

Standard errors are clustered by individual. 95% CI in parentheses.

Notes: The pooled OLS and fixed effects models include the following covariates: age (quadratic), period of observation, urban, schooling, religion, marital status, primary job, and per capita expenditure (quadratic). The OLS + CEM Model includes dummies for strata defined by 5 year age groups, urban residence, schooling, and period of observation in addition to covariates for religion, marital status, primary job, and per capita expenditure (quadratic).

In Table 2.4, I stratify the fixed-effects models by the mean BMI to determine how the association between BMI and blood pressures varies across the distribution of BMI. I find that when looking within individuals, changes in BMI are actually associated with larger changes in blood pressure in the lower BMI ranges. For example, a one unit increase in BMI for men is associated with a 2.2 (95% CI: 1.7,2.7;  $p < 0.001$ ) unit increase in blood pressure in the 15-20 BMI range, a 1.3 (95% CI: 1.0,1.6;  $p < 0.001$ ) unit increase in the 20-25 range, and a 1.2 (95% CI: 0.8,1.7;  $p < 0.001$ ) unit increase in the 25-30 range. There are similar changes in the estimated associations across BMI ranges for women, although the absolute size of the coefficients is smaller.

## 2.6. Discussion

The prevalence of hypertension in the adult ages is extremely high in Indonesia and many other developing countries. While many studies have found that obesity is a strong predictor of high blood pressure, only a small fraction of the adult population in Indonesia is actually obese. The results from existing studies on BMI and blood pressure in developing countries are also cross-sectional, and may not represent the effect of BMI changes on blood pressure. The goal of this study was to determine if changes in weight are related to changes in blood pressure at lower levels of BMI—where the majority of adults in Indonesia are. Using nationally representative longitudinal data on Indonesians over a 14 year period, I find a strong linear relationship between changes in BMI and changes in blood pressure for both men and women. This relationship remains unchanged even after adjusting for time-invariant observed and unobserved confounders using fixed effects models. While other studies have not looked at changes within individuals, this finding is consistent with cross sectional studies from both developed and developing countries [28, 9, 7, 8, 19]. When stratified over different segments of the BMI distribution, the size of the relationship is actually slightly larger in the lower BMI ranges (between 15-20). Cross-sectional studies of African diaspora populations find a similar plateauing of the relationship at higher bmi levels [4]. These findings suggest that changes in weight at even low levels of average BMI may produce large and meaningful changes in blood pressure.

Table 2.3: Estimated relationships between BMI and blood pressure stratified by mean BMI, Indonesian Family Life Survey, 1997-2014.

	Mean BMI	Mean BMI	Mean BMI	Mean BMI
Men	15-20	20-25	25-30	30-35
Body mass index	2.2 (1.7 - 2.7)	1.3 (1.0 - 1.6)	1.2 (0.8 - 1.7)	1.4 (-1.2 - 3.9)
Observations	5,529	8,140	2,273	179
R-squared	0.24	0.29	0.33	0.37
p-value	< 0.001	< 0.001	< 0.001	0.07
Women				
Body mass index	2.0 (1.5 - 2.5)	1.1 (0.8 - 1.3)	1.0 (0.7 - 1.3)	0.9 (0.3 - 1.6)
Observations	4,975	9,314	4,785	924
R-squared	0.28	0.34	0.35	0.40
p-value	< 0.001	< 0.001	< 0.001	< 0.001

Standard errors are clustered by individual. 95% CI in parentheses.

Notes: Models include the following covariates: age (quadratic), period of observation, urban, schooling, religion, marital status, primary job, and per capita expenditure (quadratic).

Although the relationship between BMI and blood pressure is strong for both men and women, the size of the relationship is smaller for women than for men. This finding is consistent with the larger literature on weight and blood pressure [10, 3, 14, 28, 9, 7, 8, 19, 2, 21]. While this difference could reflect sex differences in the biological mechanisms mediating the BMI and blood pressure relationship, this finding could also be driven by confounding from physical activity. If the relationship between BMI and physical activity is stronger for men than for women, the larger relationship between BMI and blood pressure for men could reflect greater bias due to confounding. Indeed, in sensitivity analyses for the waves of data with physical activity data (Appendix B), I find a stronger association between BMI and physical activity for men than for women; however, adjusting for physical activity did not attenuate the BMI blood pressure relationship.

There are some important considerations when using BMI as a measure of adiposity. First, many studies argue that BMI may not be a valid measure of adiposity and suggest the use of alternative measures such as waist circumference [18, 30]. Second, studies have found that changes in BMI at lower levels of BMI may reflect fundamentally different physiological processes than BMI changes at high levels—where changes at lower levels reflect changes in lean, rather than fat, mass [11]. To address both these concerns, in Appendix C, I show the relationship between BMI and waist circumference as well as the results using waist circumference (waist circumference was only measured in three of the four waves). I find a very strong linear relationship between BMI and waist circumference and similar results when using waist circumferences. These results suggest that my results are robust to alternative measures of adiposity. Still, changes in both waist circumference and BMI at lower levels may still reflect different forms of weight change compared to changes at higher levels—where.

This study has some important limitations. First, the fixed-effects models rely on within-individual changes over time. If within-individual weight change is rare, the estimated effects from these models would only apply to a narrow subset of the population. In Appendix A, I graph the within-individual variation in BMI and find that most indi-

vidual experience changes of around  $\pm 5$  units around their overall average, suggesting that the within-individual estimates are identified for a large portion of the sample. If weight loss within the sample was due to illness, there is potential for a false association between low BMI and poor health. However, I find the lowest levels of blood pressure in at the low BMI range, suggesting that this source of bias is unlikely. Confounding from physical activity and diet may bias the estimated effects. Unfortunately, detailed physical activity and dietary data are not available in the IFLS for all waves. However, if both diet and physical activity are determined by social factors such as religion, urban/rural residence, and income, bias from these sources of confounding will be reduced in the multivariate models. Additionally, the fixed effects models also reduce bias from the parts of diet and physical activity that are stable over the life-course. Further, in Appendix B I re-estimate the models for the subset of waves with physical activity data and find almost no changes to the effect sizes, suggesting that physical activity is not confounding the relationship. Studies have found that blood pressure measured from arm devices may produce artificially high blood pressure values [1]. Although this error may inflate the overall levels of blood pressure, it would not bias the BMI-blood-pressure relationship unless the measurement error was correlated with BMI. My analysis does not identify individuals that are currently taking anti-hypertensive medication, since only a subset of the waves have information on medication use. This limitation would not bias the estimated effects unless medication use interacted with the BMI-blood-pressure relationship. Since the data are drawn from a subset of the overall panel, appropriate survey weights were not available; however, omitting the survey weights would only affect the results if the probability of selection confounded the BMI-blood pressure relationship. My results were also robust to potential collider bias from common outcomes of BMI and blood pressure (Appendix D).

Despite these limitations, this study has a number of strengths. My study is one of the first to use long-term longitudinal data to estimate the effect of BMI changes on blood pressure changes, removing bias from unobserved time-invariant confounders. My results are also based on rare, nationally representative, measured health data for one

of the largest developing countries. Overall, I find strong evidence that in a low-obesity developing country context, changes in weight are related to changes in blood pressure at most levels of BMI.

In sum, I find that in a low-obesity developing country context, changes in weight are related to changes in blood pressure at most levels of BMI. These results have important implications for future levels of hypertension in Indonesia: even if obesity is low and increasing slowly over time, rising mean levels of BMI or increases in the lower range of the BMI distribution may result in a rising population prevalence of hypertension. Given that levels of hypertension are already high in Indonesia, further increases in the population prevalence of hypertension may have substantial consequences for morbidity and mortality. Policy interventions to minimize rising population levels of weight and treat existing cases of hypertension are essential for improving overall population health in Indonesia and other developing countries.

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## CHAPTER 3 : Limited Common Origin of Multiple Adult Health-related Behaviors: Evidence from U.S. Twins

Co-authored with Jere R. Behrman and Hans-Peter Kohler

### 3.1. Abstract

Health-related behaviors are significant contributors to morbidity and mortality in the United States, yet evidence on the underlying causes of the vast within-population variation in behaviors is mixed. While many potential causes of health-related behaviors have been identified—such as schooling, genetics, and environments—little is known on how much of the variation across multiple behaviors is due to a common set of causes. We use three separate datasets on U.S. twins to investigate the degree to which multiple health-related behaviors correlate and can be explained by a common set of factors. We find that aside from smoking and drinking, most behaviors are not strongly correlated among individuals. Based on the results of both within-identical-twins regressions and multivariate behavioral genetics models, we find some evidence that schooling may be related to smoking but not to the covariation between multiple behaviors. Similarly, we find that a large fraction of the variance in each of the behaviors is consistent with genetic factors; however, we do not find strong evidence that a single common set of genes explains variation in multiple behaviors. We find, however, that a large portion of the correlation between smoking and heavy drinking is consistent with common, mostly childhood, environments. This suggests that the initiation and patterns of these two behaviors might arise from a common childhood origin. Research and policy to identify and modify this source may provide a strong way to reduce the population health burden of smoking and heavy drinking.

### 3.2. Introduction

Health-related behaviors, such as smoking and heavy drinking, are responsible for a large portion of global morbidity and mortality. For example, smoking, heavy drinking, and obesity were associated with 38% of United States mortality in 1993 and almost 50% in 2000 [37, 41]. Health-related behaviors have also been implicated as reasons for international differences in life expectancy: smoking and obesity may explain why the United States has lower life expectancy compared to other Western countries and why life expectancy in the former Soviet Union countries has stagnated relative to other European countries [49, 51].

An important question for understanding trends and variation in health outcomes is whether multiple health-related behaviors are determined by a common cause or if behaviors each have unique underlying determinants. In many studies, socioeconomic status, usually measured as either schooling or household income, is posited as a cause of health-related behaviors. On first glance, the evidence is compelling: higher levels of schooling are overwhelmingly associated with healthier behaviors across many domains and may potentially explain why more-schooled people tend to be in better health [12]. Despite these associations, a more recent literature using data on identical twins has tried to determine if these associations are causal, or if schooling is determined by unobserved characteristics that also determine health-related behaviors. The findings from these studies suggest that while schooling is associated with better health-related behaviors, schooling may not be a cause of these behaviors [2, 7, 6].

Genetics are also commonly cited as causes for health-related behaviors. Studies have found that a substantial part of the variation in smoking, physical exercise, and body mass index (BMI) can be attributed to genetic differences within populations [5, 30, 60, 61]. Also, many aspects of the childhood environment have been associated with physical activity patterns [5], smoking behavior [22], and obesity across a wide range of adult ages [45]. While these studies have provided substantial evidence to suggest that genetics and child-

hood environments play an important role in the development of health-related behaviors in adulthood, the relationship between a common set of genetic endowments, childhood environments, and variation across multiple behaviors remains unclear.

In this paper we use data on U.S. twins to investigate the degree to which multiple health-related behaviors can be explained by a single set of characteristics. Our paper combines approaches from economics and behavioral genetics to determine the contribution of schooling, genetic endowments, and environments to unhealthy behaviors – or the outcomes of such behaviors such as BMI – among U.S. adults. As the health and mortality profile of high- and increasingly also low-to middle-income countries shifts further towards chronic, behavior-related, conditions, understanding the origins of health-related behaviors can help to formulate effective policies and interventions to improve population health.

### 3.3. Background

Given the substantial associations between health-related behaviors, morbidity, and mortality, a large literature has focused on why people engage in behaviors that are widely known to negatively affect health. Underlying much of this literature is the belief that specific factors, such as genetics, personality, or schooling, are common underlying determinants of a broad range of individual health-related behaviors. In the following sections, we briefly review evidence from health, economics, and behavioral genetic studies on the causes of health-related behaviors.

Economic studies of the underlying behavioral causes of health are heavily influenced by Grossman’s model of health capital. In this model, more-educated people are more likely to make better choices regarding health inputs, including health-related behaviors, given available resources (allocative efficiency), and are better at producing health from a given set of inputs (productive efficiency) [23]. Similar theories suggest that more educated people may also have more available resources to invest in health [35]. Descriptive studies of health behaviors are very consistent with these theories, since higher levels of schooling are strongly associated with healthier behaviors across many domains. For example, col-

lege graduates are less likely to smoke, less likely to be obese, less likely to drink heavily, and less likely to be physically inactive compared to high school dropouts. They are also more likely to receive mammograms, colorectal screenings, and use sunscreen [12]. Cutler and Lleras-Muney attempt to unpack these strong associations by examining the potential mechanisms behind the large education gradient in health-related behaviors. They find that around 30% of the educational gradient in health-related behaviors is explained by income, health insurance, and family background, and around 30% from knowledge and cognitive ability [15]. While this study made a substantial contribution towards understanding the sources of educational differences in health-related behaviors, the study design was limited by an inability to identify whether the education health relationship is causal. In a recent paper, Heckman, Humphries, and Veramendi use a dynamic structural model of educational choice and find evidence that education may have a causal effect on health [28]. An emerging literature using data on identical twins has also tried to determine if these associations are causal, or if schooling is determined by unobserved characteristics that also determine health-related behaviors. These studies essentially assume that identical twins share the unobserved characteristics (such as parental background, genetic dispositions, the shared mostly childhood environment) that simultaneously influence schooling and health outcomes and bias estimates of the education health relationship in conventional analyses [33]. By using within-MZ-twins estimates, the cross-sectional associations between schooling and health are purged of bias from these unobserved factors. The findings from these studies suggest that while schooling is associated with better health-related behaviors, schooling may not be a cause of health-related behaviors [2, 7, 6]. Similarly, Cutler and Glaeser try to confirm empirically Grossman’s model by arguing that if health-related behaviors are determined by individual investments in future health, different health-related behaviors should be correlated within individuals. Using data from the Behavioral Risk Factor Surveillance System, they find weak correlations between the health-related behaviors of individuals—such as obesity and smoking, and smoking and receiving mammograms for women—implying that the factors that determine health-related behaviors vary across be-

havioral domains (e.g. the factors that lead individuals to smoke do not necessarily lead individuals to be physically inactive) [14].

Variation in health-related behaviors has also been examined from a behavioral genetics perspective. Under this paradigm, health-related behaviors are additively determined by genetic endowments, common (shared by sibling) environments, and individual idiosyncratic environments. Many behavioral genetic studies of health find that a large fraction of the within-population variance in health-related behaviors is consistent with variation in genetic factors. For example, a study using Dutch twins pairs reports that smoking initiation has a heritability of 44%—implying that, subject to the assumptions of the behavioral genetics model, 44% of the variation in smoking initiation is associated with genetic differences within the population [60]. This same study finds that 51% of the variation in the initiation of smoking is associated with the shared, mostly childhood, environment between twins. This approach has been applied to a range of behaviors: in a meta-analysis of the heritability of alcohol abuse and dependence, Walters reports that around 12% of the variation in alcohol abuse is associated with genetic variation in the population [61]. Genetics are also thought to play an important role in unhealthy weight—a literature review of many behavioral genetic studies finds that genetic factors are associated with between 50% and 90% of the variation in BMI [40]. These studies thus suggest that genetic and childhood environmental heterogeneity is an important correlate of health-related behaviors. Importantly, the size of the association between genetic factors and health-related behaviors may also interact with other behaviors. For example, Mustelin et al. find that higher levels of physical activity reduce the association between genetic factors and BMI [42]. Boardman et al., find that the composition of the smoker population in the United States became increasingly genetically “vulnerable” to smoking as the overall population of smokers decreased [8]. The results from these studies suggest that genetics may become more correlated with health-related behaviors as the populations of individuals that engage in those behaviors becomes more select.

Many studies in behavioral genetics have also used data on twins to explore the co-



variation between multiple health-related behaviors [19, 25, 30, 34, 57]. For example, Eisen et al. examine the relationships between smoking and weight and alcohol and weight, by comparing the within-twins differences in smoking and drinking to within-twins differences in weight. They find that current smokers tend to weigh less compared to former and never smokers but find no relationship between alcohol consumption and weight [19]. Other twins studies have also found similar results [34]. The behavioral genetics literature on the covariation between tobacco and alcohol use is less consistent, with some studies finding a large genetic correlation between the two behaviors [57], while other studies find negligible genetic correlation [30], and others significant shared environmental correlations [25]. The variation in the results of these studies suggests that greater investigation is needed into the covariation between health-related behaviors, especially the genetic and environmental contributions to multiple behaviors.

A more recent field in genetic research uses data from the DNA of individuals with and without a certain phenotype, such as high blood pressure, to try and identify genetic variants that are correlated with phenotypes. These genome wide association studies (GWAS) can also estimate how much of the observed heritability of traits is explained by common sets of genes. Although this field is still growing, genetic variants responsible for a significant fraction of the variance of many health-related behaviors have already been identified. For example, identified genetic variants explain 18.6% of the variation in BMI, 5.6% of the variation in cigarettes smoked per day, and 15.1% of the variation total cholesterol [62]. Based on these variances, GWAS also allows for estimates of genetic correlation between traits. The results from these analyses suggest the presence of genetic correlation between some health-related behaviors and outcomes, such as BMI and cigarettes smoked per day ( $r = 0.287$ ) [11].

Finally, a mostly descriptive literature in the health sciences has found that many aspects of the childhood environment are correlated with health-related behaviors in adulthood. A common correlate of many health-related behaviors is childhood socioeconomic status, usually measured through parental education. For example, Gilman et al. find that

higher childhood socioeconomic status is negatively correlated with the risk of becoming a regular smoker and the likelihood of smoking cessation [22]. In a review of studies, Parsons et al. report similar correlates of adult obesity, identifying higher parental weight, lower childhood SES, and certain household structures as common predictors of obesity in adulthood [45]. These correlations may be the result of many mechanisms. Some studies suggest that behaviors established in childhood are more likely to persist into adulthood. For example, a cohort study of individuals from Finland finds that being physically active in childhood is a strong predictor of physical activity in adulthood [56]. The effects of childhood SES on adult behaviors may also operate through parental knowledge and resources, although some studies find a persistent relationship between childhood and adulthood behaviors even after adjusting for parental income or SES [48]. One prominent potential mechanism is known as the “fetal origins” hypothesis and posits that children exposed to poor in utero environments are more likely to have high blood pressure, obesity, and develop a range of cardiovascular diseases as adults [3, 4] (Barker, 1990, 1995). Therefore, poor childhood SES may impact adult health outcomes by negatively affecting fetal health through pathways such as poor neonatal nutrition.

Research in multiple disciplines has identified many potential causes of health-related behaviors in adulthood. While studies have shown relationships between schooling, genetics, environments, and various health-related behaviors, the extent to which these factors determine multiple behaviors remains an open question. We use three datasets on U.S. twins to provide new evidence on the degree to which multiple health-related behaviors can be explained by an underlying common set of determinants. Our focus is limited to smoking, drinking, unhealthy weight, and physical activity, since these health-related behaviors are associated with the greatest burden of adult morbidity and mortality [37, 41]. We find that aside from smoking and drinking, most behaviors are not strongly correlated among individuals. However, smoking and drinking are among the two largest behavioral risk factors for poor health, so a correlation between these two important health-related behaviors may have large implications for population health. While we find some evidence

that schooling may be related to smoking, schooling is not a strong candidate explanation for the covariation between multiple behaviors. Similarly, we find that a large fraction of the variance in each of the behaviors is consistent with genetic factors; however, we do not find strong evidence that a single common set of genes explains variation in multiple behaviors. We find, however, that a large portion of the correlation between smoking and heavy drinking is consistent with common, likely mostly in childhood, environments—suggesting that the initiation and patterns of these two behaviors might arise from a common childhood origin.

### 3.4. Data

Our analyses use three separate sources of data on American twins: the National Longitudinal Study of Adolescent to Adult Health (Add Health), the National Survey of Midlife Development in the United States (MIDUS), and the Socioeconomic Survey of Twins of the Minnesota Twin Registry (MTR).

#### *3.4.1. Description of the data sources*

Add Health is a nationally representative longitudinal survey that first surveyed children in grades 7 through 12 in 1994 and 1995, with follow-up surveys in 1996, 2001, and 2008. Beginning in the first wave, the Add Health followed a sibling subsample that included both identical (MZ) and fraternal (DZ) twins. Since the focus of this paper is on adults, we use data on the twin sample from the fourth wave of data collection, when the individuals in the cohort were between the ages of 25 and 32.

MIDUS is a longitudinal survey of the non-institutionalized population of the United States between the ages of 25 and 74. The first wave of data collection was in 1995 with a follow-up survey between 2006 and 2009. For this paper, we focus specifically on the twin subsample, pooling data from both survey years. Finally, we use data from the Socioeconomic Survey of Twins of the Minnesota Twin Registry (MTR). The MTR is a registry of all twins born between 1936 and 1955 in Minnesota. Our data are from the Socioeconomic Survey of Twins, a mail-based survey of same-sex MZ and DZ twins conducted in

1994.

Different procedures were used to identify zygosity across the three datasets. Zygosity in the Add Health data was initially self-reported by the twins but was later confirmed by DNA testing. In the MIDUS data, twins were given a separate survey and asked to self-report their zygosity as either monozygotic or dizygotic. Finally, the zygosity of individuals in the MTR sample was based on analysis of blood enzymes, serum proteins, fingerprint ridgecount, and other biological comparisons. For all three surveys we only consider MZ and same-sex DZ twins, since opposite-sex DZ twins reduce the tenability of the “shared environments” assumption of behavioral genetics models (many behavioral genetic studies also drop opposite sex pairs [25, 30]).

### *3.4.2. Schooling*

While socioeconomic status is reflected over multiple measures, such as income, occupation, and schooling, we limit our focus to schooling for the following reasons. First, measures such as income have been shown to fluctuate over the life course. Income and occupation may also be inversely related with health, where individuals with poor adult health and health-related behaviors earn less money and are less likely to be employed [55]. For both these reasons, income and occupation may not be stable measures of socioeconomic status. In contrast, schooling is preferred as a measure of socioeconomic status in many studies since it is established relatively early in life, and for most people, remains unchanged over the life course [20].

For all three datasets individuals categorically reported their highest level of completed schooling. Based on these responses, we created a continuous measure of grades of schooling by assigning grades of schooling to each of the completed categories. The categories were assigned as follows.

Add Health: Eighth grade or less (8 grades), some high school (10 grades), high school graduate (12 grades), some vocational/technical training (12.5 grades), completed vocational/technical training (13 grades), some college (14 grades), completed college (16

grades), some graduate school (17 grades), completed master’s degree (18 grades), some graduate training beyond a master’s degree (20 grades), completed a doctoral degree (22 grades), some post baccalaureate professional education (18 grades), completed post baccalaureate professional education (20 grades).

MIDUS: No school/some grade school (3 grades), eighth grade/junior high school (7 grades), some high school (10 grades), GED (10 grades), graduated from high school (12 grades), 1–2 years of college (13 grades), graduated from a 2-year college (14 grades), 3 or more years of college (15 grades), graduated from a 4- or 5-year college (16 grades), some graduate school (17 grades), master’s degree (18 grades), doctoral degree (21 grades). MTR: No schooling or completed grades up through secondary school graduation (actual grades as reported), GED (11 grades), vocational degree (13 grades), associate degree or some college (14 grades), bachelor degree (16 grades), masters degree (18 grades), doctoral degree (21 grades).

### *3.4.3. Health-related behaviors*

We created two binary variables for smoking and drinking to capture both initiation and quantity consumed. For smoking, we created a variable for ever smoker if an individual reported ever regularly smoking and variable for heavy smoker if an individual reported currently smoking a pack per day or more. Similarly, we created a variable for ever drinker if an individual ever reported consuming alcohol and a variable for heavy drinker if an individual reported currently drinking four or more drinks per sitting on average (unfortunately, the MTR did not ask about drinks per day, rather they asked the number of days an individual drank per week so for heavy drinking is defined in terms of drinking more on more than four days per week). We preferred drinks per day rather than the number of days an individual drank, since this measure may better capture harmful binge drinking patterns [59].

Measurements of physical activity varied slightly across datasets. For Add Health, we measured physical activity by the number of times per week an individual reported

engaging in vigorous physical activity. This was constructed based on a series of questions on different types of physical activity: we first categorized these questions as light, moderate, and vigorous activity based on their MET score [1], then translated the number of times an individual performed each type of activity into the total number of times they engaged in vigorous activity. In the MIDUS, we used a continuous variable of the average number of days per month that an individual reported engaging in vigorous activity (this variable was top coded at 14 days in the MIDUS data). Finally, we do not have measurements of physical activity in the MTR since individuals were not asked about their activity patterns. Due to the difficulty in measuring diet, we proxied the combined effects of diet and physical activity as unhealthy weight—measured by BMI for all three datasets.

#### *3.4.4. Validity and reliability of the outcome measures*

Although we were not able to directly assess the reliability or validity of our outcomes, we use standard measurements with extensively documented reliability and validity. Based on a meta-analysis of the validity of self-reported smoking, Patrick et al. find that across studies, self-reported smoking tracks closely with biomarker measures of tobacco use [46]. Self-reported smoking has also been shown to be reliable, with a greater reliability for ever-smoking ( $\kappa = 0.82$ ) compared to categories such as light or heavy smoker ( $\kappa = 0.6$ ) [10, 32]. Retrospective quantity smoked has also been found to agree with cigarette sales [27]. Retrospective alcohol information has shown moderate to high reliability: one study estimates a  $\kappa$  between 0.26 and 0.54 while another finds that retrospective alcohol accounts for 86% of the variability in current alcohol consumption [16, 26]. Although the validity of self-reported alcohol is harder to assess, a large meta-analysis concludes that self-reported alcohol is a generally valid measure [39]. For self-reported physical activity, studies of the test-retest reliability find that reliability and validity is generally high, but more so for vigorous than moderate activity [54]. For example, a study of Latinos finds a correlation of  $r > 0.4$  between self-reported vigorous activity and measured activity [50]. Finally, BMI was directly measured for two of the three datasets; in the MTR data, BMI was calculated based on self-reported height and weight. For this dataset, BMI might be underestimated

due to height underreporting for men and weight underreporting for women [38]. There is a general question on whether BMI is a valid measure of body fat; studies find that the validity of BMI as a measure of fat is moderate in the middle ranges and high at higher levels of BMI [18, 52]. Overall, our measures are generally regarded as valid and reliable but it is still important to note potential errors introduced by self-reports, especially for physical activity and alcohol behavior (for the within-MZ twins models, reporting error would only bias the estimates if one twin misreports differently than the other).

#### *3.4.5. Missing values and sample size*

For Add Health, the total wave 4 twin sample consisted of 396 complete MZ or same-sex DZ twin pairs. 22 twin pairs (5.6%) were dropped for missing information for one or both members of the twinship for a final sample of 373 twin pairs (206 MZ twin pairs and 167 DZ twin pairs). The total MIDUS twin sample for waves 1 and 2 pooled consisted of 1085 complete twin pairs. 332 twin pairs (30.6%) were dropped for missing information on the key covariates for one or both members of the twinship for a final sample size of 753 twin pairs (416 MZ twin pairs and 337 same-sex DZ twin pairs). Finally, the MTR had an initial twin sample of 1399 complete twin pairs. 246 twin pairs (17.6%) were dropped for missing information on the key covariates for a final sample of 1153 twin pairs (647 MZ twin pairs and 506 same-sex DZ twin pairs).

### 3.5. Methods

If health-related behaviors are determined by a common set of determinants, we would expect them to correlate within individuals. Therefore, we first estimated a simple correlation table of each of the health-related behaviors for each of the datasets.

#### *3.5.1. Within-MZ twins models*

Our next goal was to determine if schooling is a common cause of multiple health-related behaviors. While a simple regression of health-related behaviors on schooling would quantify the association between schooling and each health-related behavior, both schooling

and health-related behaviors may be determined by unobserved characteristics (such as unobserved dimensions of parental and family background, genetic dispositions, and the childhood environment). By comparing differences in schooling and health-related behaviors, within-MZ twins regressions can net out confounding from these unobserved factors, since identical twins have identical genes at birth, the same parental and family characteristics, and largely the same childhood environment. The plausibility of these estimates depends on the size of the within-twins differences in both schooling and each outcome; in Appendix Figs. 1–3 we graph the within-twins distributions and find a wide range of differences across twin pairs. For example, for a health-related behavior  $y_i$  for individual  $i$ , the regression of  $y_i$  on schooling would be:

$$y_i = \beta_0 + \beta_1 \text{schooling}_i + \beta_2 \text{age}_i + \beta_3 \text{male}_i + \gamma z_i + \epsilon_i \quad (3.1)$$

where  $z_i$  are the unobserved parental, family, genetic, and child environmental characteristics discussed above. The  $\beta_1$  is the association between schooling and behavior  $y$ , but it is not the causal effect, since both schooling and behavior  $y$  are affected by  $z$ . By comparing the within-MZ twins difference in both schooling and health-related behaviors, we can instead estimate the following regression for twinship  $j$ :

$$(y_{1j} - y_{2j}) = \beta_1(\text{schooling}_{1j} - \text{schooling}_{2j}) + \gamma(z_{1j} - z_{2j}) + (\epsilon_{1j} - \epsilon_{2j}) \quad (3.2)$$

Since MZ twins have identical genes at birth, parental and family backgrounds, and childhood environments,  $z_{1j} - z_{2j}$  cancels out, removing the confounding from these unobserved factors.

These models have a few potential problems. First, we have to assume that the source of the within-MZ twins difference in schooling is unrelated to the within-MZ difference in each health-related behavior. If, for example, the same shock caused one twin to discontinue schooling before their cotwin and make them smoke, the within-MZ estimate would falsely attribute the smoking difference between twins to the schooling difference, rather than the

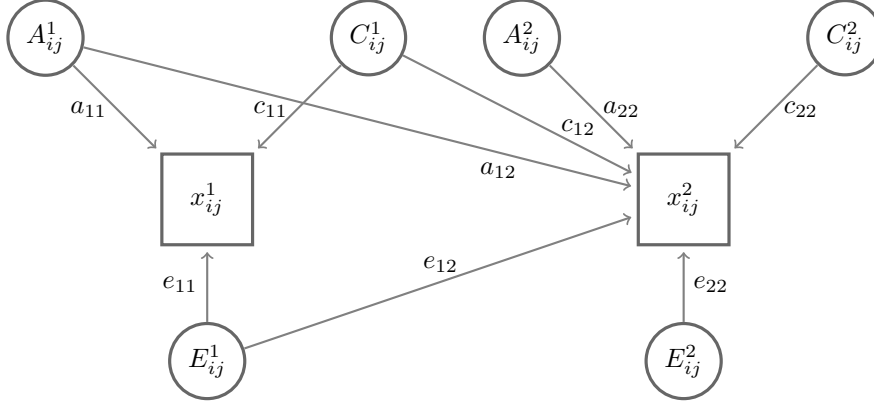


true unobserved shock. Therefore, if this assumption is violated, the within-MZ estimates becomes a bound on the true on the true causal estimate [33]. In addition, if there is measurement error in schooling, the degree of error would be increased for the within-MZ twins regression, biasing the estimated effect towards zero [9]. While these sources of bias may be important, both produce predictable bounds on the true causal estimate [33]. Despite these limitations, the within-MZ regressions provide a robust approach for controlling for unobserved characteristics that may confound the schooling and health-related behavior relationship. We therefore estimated a regression of the form (2) for each of the health-related behaviors.

### 3.5.2. Behavioral genetics models

While the economics literature has focused on the effects of schooling on health and health-related behaviors, behavioral genetics has focused on the role of genetics and environments. In many behavioral genetics studies, observed characteristics like health-related behaviors are expressed as the result of additive genetic endowments ( $A$ ), the shared environment between twins ( $C$ ), and individual environmental factors ( $E$ ). Each health-related behavior can be the result of its own  $A$ ,  $C$ , and  $E$ , or the  $A$ ,  $C$ ,  $E$  factors that also determine other behaviors. The degree to which multiple health-related behaviors are determined by a common set of genetic, shared environment, and individual environmental factors can then be determined by seeing how much of the variance in multiple behaviors is due to a common subset of  $A$ ,  $C$ ,  $E$  factors and how much variation is due to behavior-specific factors. This is the intuition behind the multivariate ACE model, which can be represented by the path diagrams in Fig. 3.1 (the figure is shown for only two health-related behaviors for clarity, but this approach generalizes to any number of behaviors). Here,  $x_{ij}^1$  and  $x_{ij}^2$  are two observed behaviors for individual  $i$  in twin pair  $j$  and all the  $A_{ij}^k$ ,  $C_{ij}^k$ , and  $E_{ij}^k$  are the behavior specific factors. As the diagram shows, each behavior can be the result of its own  $A$ ,  $C$ , and  $E$  factor (paths  $a_{11}$ ,  $c_{11}$ ,  $e_{11}$ ,  $a_{22}$ ,  $c_{22}$ , and  $e_{22}$ ) and the  $A$ ,  $C$ ,  $E$  factors of the other behaviors (paths  $a_{12}$ ,  $c_{12}$ , and  $e_{12}$ ).

Figure 3.1: Path diagrams for the multivariate ACE model



One important conceptual issue arises in the measurement of smoking and drinking. In many behavioral genetic studies of smoking and drinking, researchers assume that every individual has an underlying latent “propensity” for smoking and drinking. Categories such as ever smoker/ever drinker and heavy smoker/heavy drinker simply classify individuals that fall above some threshold on the latent propensities. We follow this approach by combining ever and heavy use into one categorical variable, and then use this model to estimate smoking and drinking as continuous latent propensities.

Using information on both MZ and DZ twins and assuming that MZ twins share identical genetic endowments and common environments while DZ twins share identical common environments and on average 50% of their genetic endowments, we can represent the correlations between all the behaviors as a function of the  $a$ ,  $c$ , and  $e$  path coefficients. This has the advantage of then letting us determine how much of the correlation between the behaviors is due to common genetic factors ( $A$ ), common shared environments between twins ( $C$ ), and common individual idiosyncratic environments ( $E$ ) by looking at the correlations generated by just the subset of the  $a$ ,  $c$ , and  $e$  path coefficients respectively. For more details on the estimation of these models see: [43].

We determine the role of a common set of genetic, shared environment, and individual environmental factors by using the model presented in Fig. 1 to first estimate the cor-

relation between behaviors as a function of all the path coefficients. We then decompose these correlations into the contribution of genetic endowments, shared environments, and individual environments. Large factor-specific contributions to the correlations would imply that a common set of factors is influencing multiple behaviors.

In many twins studies, researchers fit alternative models that assume some factors have no influence (AE, CE, and E models)—in Appendix Table 3.1 we compare the fit of these sub-models to the standard ACE model and find that the ACE provides the best statistical fit for two of the three datasets. Although the AE model provides the best statistical fit for one dataset, our theoretical question revolves around the role of shared environments, so we did not want to constrain this factor to be 0. Similarly, we do not estimate models with genetic dominance effects since they cannot be identified simultaneously with the shared environment parameters unless one is willing to assume an absence of additive genetic effects (an assumption that is generally not plausible).

The behavioral genetics models also make a number of important assumptions that have implications for the results. First, the models assume that the means and variances of each behavior are equal across MZ and DZ twins. In Appendix Table 3.2 we present the proportions, means, and standard deviations across all the variables and find that the levels for most variables are similar across zygosity. Still, there are differences in heavy smoking and heavy drinking across zygosity that may lead to error in the model estimation. Second, the models as presented here assume no gene-environment interactions. This is an important assumption and can potentially bias the genetic contributions if the size of the genetic contribution varies based on environmental interactions [42]. Third, the models assume that the influence of the shared environment is equivalent for both MZ and DZ twins. If, for example, parents were more likely to treat MZ twins similarly compared to DZ twins, the size of the A contributions would be biased upward, leading to inflated estimates of the role of genetics. The models also assume that there is no assortative mating in the population. If individuals with similar health-related behaviors were more likely to have children, the estimated C contributions would be biased upward. Finally, measurement

error in the outcomes can lead to inflated estimates of E while biasing the A and C estimates downward. This bias would lead to conservative estimates of the contribution of genetics and shared environments. Although these assumptions are important to consider, the behavioral genetic models still provide a strong way to assess the relationship between genetic and environmental factors and adult health-related behaviors.

### 3.6. Results

Table 3.1 presents a descriptive overview of the three twins samples. The MIDUS and MTR samples are on average middle aged (47.07 years old for MTR and 47.53 for MIDUS) while individuals in the Add Health are slightly younger (28.93 years). All three samples have a greater share of women compared to men—this difference is especially pronounced for the MTR sample (65.13% female). Most of our analyses focus specifically on differences within twins pairs and would not be biased by the sex composition of the samples. Across all four of the identified health-related behaviors, we observe a common pattern: large fractions of individuals have ever smoked or drank with a much smaller number of individuals currently consuming heavy quantities. For example, between 30% and 40% of individuals in all three samples reported ever smoking; in contrast, the fraction that currently heavy smoke is only between 5% and 14%. Similar patterns are observed for drinking: over 70% of individuals reported ever drinking in all three samples but only around 20% currently consume four or more drinks per sitting (based on the Add Health and MIDUS samples. Although average levels of vigorous physical activity are fairly low (2.44 times per week among the Add Health sample and 6.37 times per month in the MIDUS sample), both measures have large standard deviations, implying a wide distribution in physical activity behavior. Based on the standard Centers for Disease Control and Prevention cutoffs for BMI, the samples are on average slightly overweight.

Table 3.1: Descriptive characteristics of the Add Health, MIDUS, and Minnesota Twins samples

	Add Health Twins			MIDUS Twins			MTR Twins		
	N = 756			N = 1,474			N = 2,344		
	Mean or n	SD	or %	Mean or n	SD	or %	Mean or n	SD	or %
Age	28.93	1.62		47.53	12.31		47.07	5.62	
<i>Sex</i>									
Male	362	48.53		630	41.83		804	34.87	
Female	384	51.47		876	58.17		1,502	65.13	
<i>Zygosity</i>									
MZ	412	55.23		832	55.25		1,294	56.11	
DZ	334	44.77		674	44.75		1,012	43.89	
Ever smoker	292	39.14		824	54.71		947	41.07	
Heavy smoker	41	5.5		212	14.08		292	12.66	
Ever drinker	577	77.35		1,147	76.16		1,628	70.6	
Heavy drinker	147	19.17		330	21.91		136	5.9	
Vigorous activity per month	-	-		6.37	5.36		-	-	
Vigorous activity per week	2.44	2.56		-	-		-	-	
BMI	28.07	7.29		28.59	5.10		25.82	4.64	

Notes: Data are shown for the total number of people (the number of twin pairs is the total sample size divided by 2). The Minnesota twins did not contain questions on drinks per sitting (heavy drinking in the Minnesota Twins is measured as drinking more than 3 days per week) or physical activity. Two different measures of vigorous activity are presented since the Add Health and MIDUS surveys asked physical activity over different recall periods.

Figs. 3.2–3.4 graph the correlation matrix of the selected health-related behaviors for all three samples. The below diagonal elements are the scatterplots of the behaviors against one another while the above diagonal elements are the correlation coefficients. Across all three samples, the most striking initial result is the lack of correlation among many of the behaviors. For example, heavy smoking and physical activity has a correlation of -0.083 in the Add Health sample and a correlation of -0.077 in the MIDUS sample—implying that individuals that smoke heavily are only very slightly less likely to engage in physical activity. Similarly, the correlation of heavy drinking and BMI is -0.038 in the Add Health sample, 0.014 in the MIDUS sample and -0.032 in the MTR sample. These correlations indicate that individuals who drink heavily are not more likely to have higher levels of unhealthy weight. On first glance, these results suggest that a single factor (whether it is personality, schooling, environments, or genetics) is unlikely to be a strong cause of multiple health-related behaviors since the behaviors themselves do not correlate highly. This general lack of correlation between the health-related behaviors is consistent for almost every pairwise comparison except for one: smoking and drinking. We find a large correlation between ever smoking and heavy drinking in two datasets (0.20 in the Add Health, 0.23 in the MIDUS) and between ever smoking and ever drinking in the MTR data ( $r = 0.25$ ). In the following section, we investigate the role of schooling, genetics, and the childhood and adolescent environment in explaining the covariation between health-related behaviors, paying special attention to smoking and drinking.

In Tables 3.2–3.4, we show the results from the OLS and within-twins fixed-effect regressions of each health-related behavior on years of schooling. Focusing on just the OLS regressions, we find the commonly reported conclusion of an association between schooling and better health-related behaviors. In the Add Health sample, a one-year increase in schooling is associated with a lower probability of ever smoking, a lower probability of heavy smoking, an increase in the times an individual engages in vigorous activity per week, and a lower BMI. This pattern of associations between schooling and health-related behaviors is largely similar in the other two samples: in the MIDUS sample schooling is

Figure 3.2: Correlation matrix and scatter plots for the selected health behaviors, Add Health Twins, N = 746

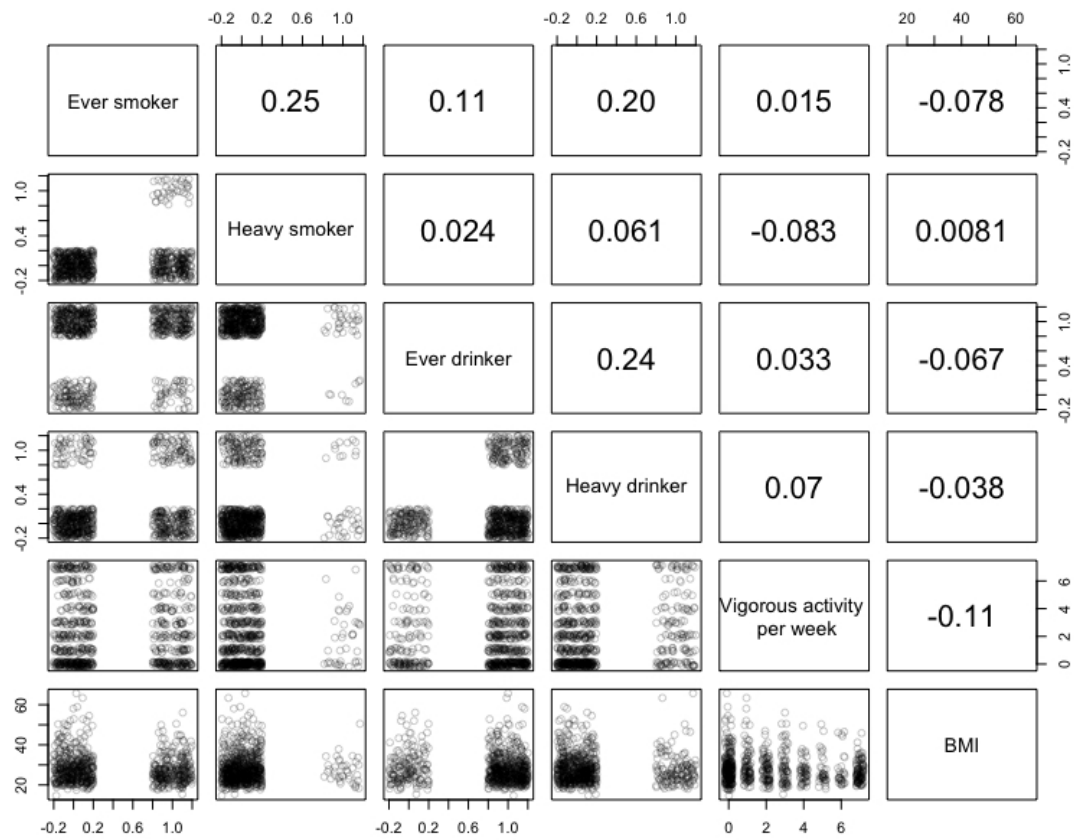


Figure 3.3: Correlation matrix and scatter plots for the selected health behaviors, MIDUS Twins, N = 1,506

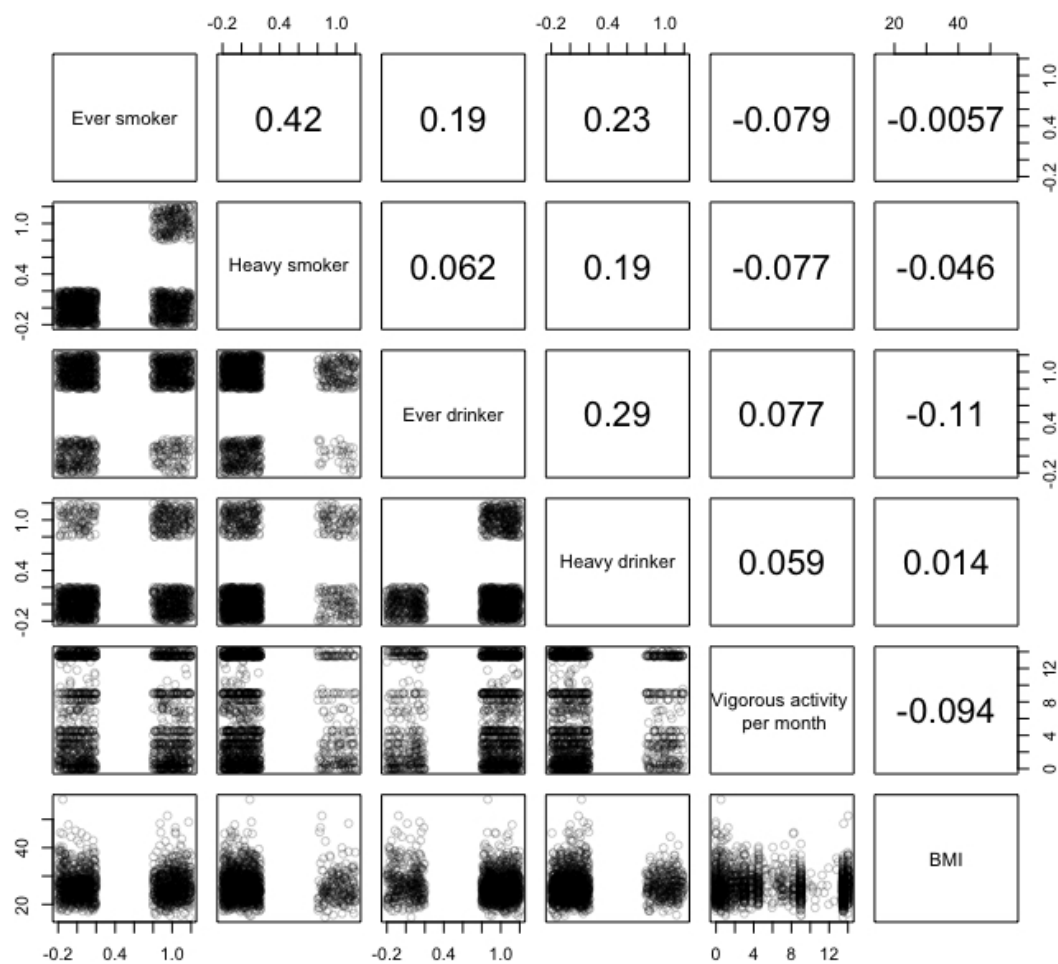
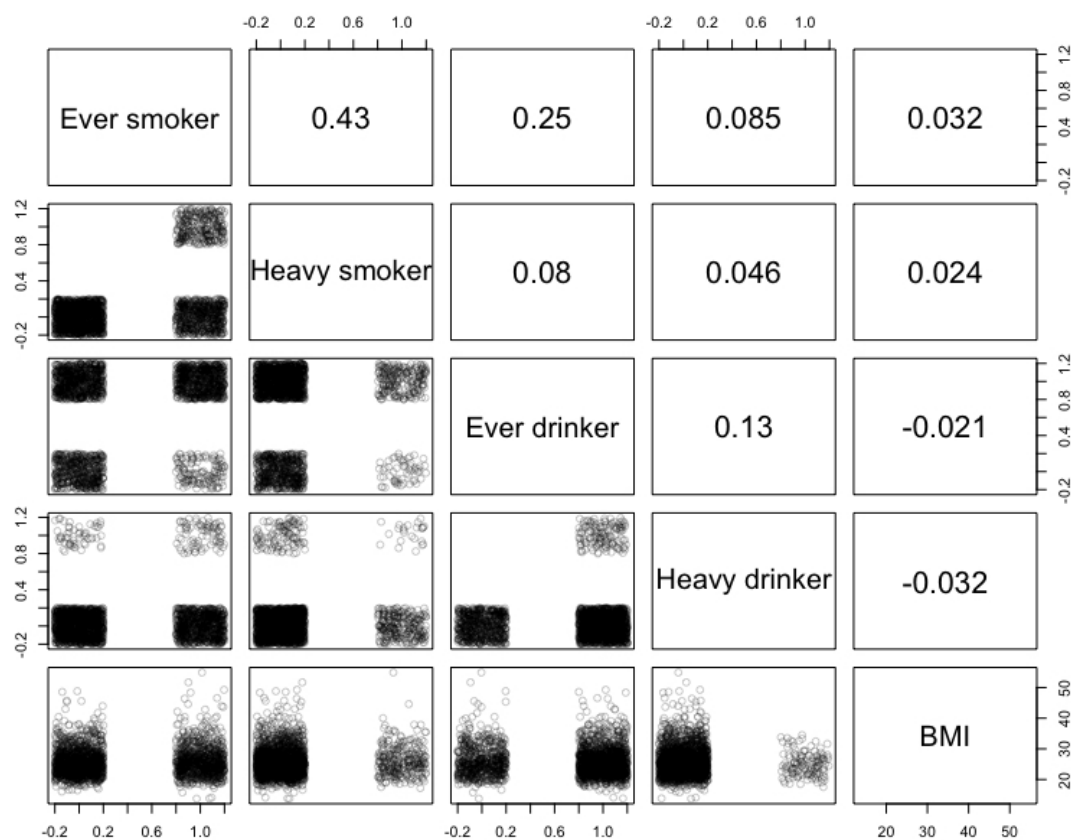




Figure 3.4: Correlation matrix and scatter plots for the selected health behaviors, MTR Twins, N = 2,306



associated with less smoking, less heavy drinking, more vigorous activity per week, and a lower BMI. While these results indicate an association between schooling and health-related behaviors, an important question is whether these associations are robust to unobserved characteristics.

Table 3.2: Estimated OLS and within-MZ twin regressions of smoking, drinking, physical activity, and unhealthy weight on schooling, Add Health Twins, N = 412

	Ever smoker		Heavy smoker		Ever drinker		Heavy drinker		Vigorous act per week		BMI	
	OLS	FE	OLS	FE	OLS	FE	OLS	FE	OLS	FE	OLS	FE
Years of schooling	-0.053*** (0.010)	-0.043* (0.018)	-0.014** (0.005)	-0.006 (0.004)	0.035*** (0.009)	0.007 (0.016)	-0.005 (0.008)	0.008 (0.013)	0.134* (0.059)	0.056 (0.112)	-0.502** (0.163)	0.041 (0.108)
Age	0.034+ (0.018)	0.062 (0.165)	0.022** (0.008)	-0.053 (0.054)	-0.018 (0.016)	-0.051 (0.162)	-0.013 (0.014)	-0.301+ (0.170)	0.037 (0.089)	1.039 (1.009)	-0.191 (0.277)	-3.506 (2.869)
Male	0.028 (0.055)		0.033 (0.024)		0.128** (0.046)		0.125** (0.044)		0.580* (0.286)		1.560+ (0.932)	
R-squared	0.081	0.032	0.050	0.003	0.064	0.001	0.027	0.017	0.032	0.007	0.040	0.028

Standard errors are clustered by twinship. Linear probability models were estimated for dichotomous outcomes.

\*\*\* p<0.001, \*\* p<0.01, \* p<0.05, + p<0.1

Table 3.3: Estimated OLS and within-MZ twin regression of smoking, drinking, physical activity, and BMI on schooling, MIDUS Twins, N = 832

	Ever smoker		Heavy smoker		Ever drinker		Heavy drinker		Vigorous act per week		BMI	
	OLS	FE	OLS	FE	OLS	FE	OLS	FE	OLS	FE	OLS	FE
Years of schooling	-0.044*** (0.007)	-0.014 (0.010)	-0.030*** (0.006)	-0.020* (0.009)	0.011+ (0.006)	0.007 (0.011)	-0.020*** (0.005)	-0.002 (0.008)	0.230** (0.079)	0.160 (0.141)	-0.229** (0.079)	-0.060 (0.081)
Age	0.002 (0.002)	-0.028 (0.064)	-0.002 (0.001)	-0.005 (0.060)	-0.008*** (0.001)	0.002 (0.070)	-0.007*** (0.001)	-0.074 (0.064)	-0.096*** (0.016)	-0.823 (0.825)	0.030+ (0.018)	0.309 (0.616)
Male	0.094* (0.042)		0.021 (0.028)		0.071* (0.035)		0.258*** (0.033)		1.180** (0.401)		0.946* (0.416)	

Standard errors are clustered by twinship. Linear probability models were estimated for dichotomous outcomes.

\*\*\* p<0.001, \*\* p<0.01, \* p<0.05, + p<0.1

Table 3.4: Estimated OLS and within-MZ twin regression of smoking, drinking, and BMI on schooling, MTR, N = 1,294

	Ever smoker		Heavy smoker		Ever drinker		Heavy drinker		BMI	
	OLS	FE	OLS	FE	OLS	FE	OLS	FE	OLS	FE
Years of schooling	-0.040*** (0.005)	-0.016 (0.010)	-0.017*** (0.003)	-0.001 (0.007)	-0.001 (0.005)	-0.003 (0.007)	0.004+ (0.002)	0.007 (0.005)	-0.160** (0.054)	0.041 (0.061)
Age	-0.008** (0.003)		-0.004* (0.002)		0.000 (0.003)		-0.003* (0.001)		-0.096*** (0.029)	
Male	0.159*** (0.033)		0.034 (0.021)		0.197*** (0.029)		0.043** (0.015)		1.336*** (0.308)	
R-squared	0.073	0.005	0.025	0.000	0.042	0.000	0.021	0.003	0.038	0.001

Standard errors are clustered by township. Linear probability models were estimated for dichotomous outcomes.

\*\*\* p<0.001, \*\* p<0.01, \* p<0.05, + p<0.1

Tables 3.2–3.4 also report the within-MZ twins regressions, providing a more robust evaluation of the schooling-health-related-behavior relationship (for the Add Health and MIDUS samples both twins were not interviewed on the same day. This resulted in a one-year difference in age between the twins for a minority of cases, leading to an estimated coefficient for age even for the within-MZ models). The within-MZ results display a much different overall pattern compared to the standard OLS results. For most of the significant OLS associations, the within-twins estimates are substantially smaller in magnitude and most lose statistical significance. For example, the relationship between schooling and heavy smoking moves from -0.014 to -0.006 in the Add Health sample, from -0.030 to -0.020 in the MIDUS sample, and from -0.017 to -0.001 in the MTR sample (for the MIDUS sample the within-MZ effect is still significant). Similarly, the coefficient for the BMI outcomes moves from -0.502 to 0.041 in the Add Health, from -0.229 to -0.060 in the MIDUS, and from -0.160 to 0.041 in the MTR sample. Not every relationship diminishes or loses statistical significance. In the MIDUS sample, the OLS and within-MZ coefficients are significant for heavy smoking and in the Add Health sample the OLS and within-MZ estimates are both significant for ever smoking, suggesting that schooling may be related to smoking behavior.

While the results from the schooling regressions (Tables 3.2–3.4) suggest that schooling may be related to some health-related behaviors, we find almost no support for the hypothesis that schooling affects all four of the behaviors examined. Focusing specifically on smoking and drinking, the two most correlated health-related behaviors, we find that the schooling effect is much larger in magnitude for smoking than for drinking in all of the three samples (where the schooling-drinking effect is extremely close to zero). These results suggest that schooling is unlikely to be an important common cause of both behaviors.

In Tables 3.5–3.7 we move towards investigating the role of genetics and the childhood environment as potential causes of health-related behaviors. For each table, we present the implied correlation matrix calculated through the behavioral genetics model, and the genetic, shared environment, and individual environment specific contributions to the es-

timated correlations. These second two matrices estimate the portion of the correlation between the behaviors that arise from a common set of genes or shared environments. The diagonals of the genetic, environmental, and individual matrices represent the fraction of variance in each behavior that is consistent with genetic endowments and environmental factors.

Table 3.5: Estimated correlation matrix with genetic, shared environment, and individual environment contributions, Add Health Twins, N = 746

	Estimated Correlation Matrix				Genetic Contributions			
	Smoking	Drinking	Vig. act.	BMI	Smoking	Drinking	Vig. act.	BMI
Smoking	1.00				0.29			
Drinking	0.22	1.00			0.03	0.11		
Vig. act.	0.01	0.05	1.00		0.03	0.08	0.27	
BMI	-0.08	-0.07	-0.11	1.00	0.02	-0.12	-0.12	0.77
	Shared Environmental Contributions				Individual Environment Contributions			
	Smoking	Drinking	Vig. act.	BMI	Smoking	Drinking	Vig. act.	BMI
Smoking	0.34				0.37			
Drinking	0.10	0.30			0.09	0.59		
Vig. act.	-0.04	0.01	0.04		0.02	-0.04	0.69	
BMI	-0.07	0.04	0.04	0.06	-0.03	0.01	-0.03	0.17

Notes: Smoking and drinking are measured as latent propensities. Vigorous activity is measured as times per week.

Table 3.6: Estimated correlation matrix with genetic, shared environment, and individual environment contributions, MIDUS Twins, N = 1,506

	Estimated Correlation Matrix				Genetic Contributions			
	Smoking	Drinking	Vig. act.	BMI	Smoking	Drinking	Vig. act.	BMI
Smoking	1.00				0.27			
Drinking	0.33	1.00			0.14	0.33		
Vig. act.	-0.12	0.10	1.00		-0.03	0.21	0.25	
BMI	-0.03	-0.08	-0.09	1.00	-0.03	0.02	-0.02	0.64
	Shared Environmental Contributions				Individual Environment Contributions			
	Smoking	Drinking	Vig. act.	BMI	Smoking	Drinking	Vig. act.	BMI
Smoking	0.47				0.26			
Drinking	0.17	0.39			0.02	0.28		
Vig. act.	-0.07	-0.09	0.03		-0.02	-0.02	0.72	
BMI	0.04	-0.10	0.02	0.06	-0.04	0.00	-0.09	0.30

Notes: Smoking and drinking are measured as latent propensities. Vigorous activity is measured as times per month.



Table 3.7: Estimated correlation matrix with genetic, shared environment, and individual environment contributions, MTR Twins, N = 2,306

	Estimated Correlation Matrix			Genetic Contributions		
	Smoking	Drinking	BMI	Smoking	Drinking	BMI
Smoking	1.00			0.15		
Drinking	0.33	1.00		0.06	0.49	
BMI	0.04	-0.04	1.00	0.13	-0.02	0.68
	Shared Environment Contributions			Individual Environment contributions		
	Smoking	Drinking	BMI	Smoking	Drinking	BMI
Smoking	0.41			0.44		
Drinking	0.18	0.14		0.09	0.37	
BMI	-0.03	-0.01	0.00	-0.06	-0.01	0.32

Notes: Smoking and drinking are measured as latent propensities

Across all three samples, we find that genetic endowments are consistent with a large fraction of the variance in many of the health-related behaviors. For smoking, genetic endowments are consistent with 29% of the variance among the Add Health twins, 27% among the MIDUS twins, and 15% of the variance among the MTR twins. Similarly, genetic endowments are consistent with a large fraction of the variance in BMI: 77% in Add Health, 64% in MIDUS, and 68% in MTR. The role of the shared, mostly childhood, environment is less pronounced for BMI and physical activity across the datasets. For example, the shared environment is consistent with 6% of the variance in BMI and 4% of vigorous activity for the Add Health sample. We observe a relatively similar pattern in the MIDUS data, with 6% of the variance in BMI and 3% of the variance in vigorous activity consistent with shared environmental factors. However, the results suggest that the childhood and adolescent environment plays an important role in smoking and drinking behavior in adulthood. One of the more surprising findings is that across all three samples and all behaviors, a large fraction of the variation in each of the behaviors is due to individual idiosyncratic environments. While this term also captures measurement and specification errors, these results suggest that despite the potential role of schooling, genetics, and environments in explaining portions of the variation and covariation in these four behaviors, much of the variance is idiosyncratic and behavior specific.

The off-diagonal elements of the matrixes measure the correlation between behaviors consistent with a common set of genetic endowments or environments. As mentioned previously, the one pairwise comparison with a large correlation coefficient is smoking and drinking. For all three samples, we find that a large portion of this correlation is consistent with a common environmental factor (environmental contribution is 0.10 in the Add Health sample, 0.17 in the MIDUS sample, and 0.18 in the MTR sample).

For the other pairwise comparisons, the role of a common set of genetic endowments and environments is inconsistent across the three samples. For example, we find that a common set of genetics is consistent with the covariation in smoking and drinking among the MIDUS twins (contribution = 0.14), but this contribution is not present in the Add

Health or MTR data. We also find a moderate genetic correlation between cigarette smoking and BMI in the MTR sample (contribution = 0.13) that is not present in the other two samples. The inconsistent correlations across the datasets for most of the pairwise comparisons of behaviors is not surprising, since many of these behaviors do not have strong overall correlations.

### *3.6.1. Robustness*

We conducted a number of robustness checks. First, our results were consistent when using continuous measures of smoking and drinking. Our results were also consistent when looking at just moderate physical activity and a measure that combined both moderate and vigorous physical activity. As mentioned previously, the within-MZ regressions may be biased towards zero if there is measurement error in schooling. Although only available in the MTR dataset, we used co-twin reported schooling as an instrument for an individual's schooling and estimated instrumental variable regressions to reduce bias from measurement error. We find that measurement error in the MTR dataset does not affect our conclusions, with the coefficient actually becoming smaller for some outcomes (Appendix Table 3.3)

## 3.7. Discussion

Health-related behaviors are significant contributors to morbidity and mortality in the United States, yet evidence on the underlying causes of the vast within-population variation in behaviors is mixed. While many potential causes of health-related behaviors have been identified—such as schooling, genetics, and environments—the magnitude of the variation across multiple behaviors that is due to a common set of causes remains an open question. Using three data sources on U.S. twins, we do not find evidence that schooling, or a common set of genetic endowments or environments are a common cause of most health-related behaviors. Smoking and excessive alcohol consumption is the main exception: we find evidence that variation in both adult smoking and drinking is consistent with a common shared environment between twins (mostly the childhood environment). Overall, the

results of our study suggest that the causes for health-related behaviors in adulthood are largely idiosyncratic.

Our first primary conclusion is that across all three samples, the key health-related behaviors investigated in this paper do not correlate as strongly as we, and probably many others, would have expected. While theories on the causes of health-related behaviors across many disciplines imply that many behaviors have a common underlying cause, and should therefore correlate, the patterns in our data are not consistent with this expectation. Individuals that smoke are not substantially less likely to be physically active or more likely to have unhealthy weight. Similarly, we observe very weak correlations between physical activity and unhealthy weight, and unhealthy drinking and physical activity. These findings suggest that individuals selectively engage in some unhealthy behaviors but not necessarily multiple behaviors. While perhaps surprising and counter-intuitive, this conclusion is consistent with research on the correlation between health behaviors using the Behavioral Risk Factor Surveillance System in the United States [14]. The one main exception to the lack of correlation across health-related behaviors is the relationship between smoking and drinking (drinks per sitting or day): across all three of the samples, we find that individuals who smoke more are also more likely to drink more per sitting. This finding has precedent in the literature, with many studies documenting an association between the two behaviors [17, 24, 53]. Despite the lack of correlation between many behaviors, the presence of a correlation between smoking and drinking is important, since smoking and heavy drinking are the two health-related behaviors associated with the largest burden of morbidity and mortality [37, 41]. Interventions aimed at the cause of this correlation may provide a strong way to improve population health.

Our second main conclusion is that the relationship between schooling and health-related behaviors is unlikely to be causal: while we initially find many strong associations between schooling and the health-related behaviors, most of these associations attenuate and become non-significant after controlling for unobserved differences shared between MZ twins. Schooling also seems an unlikely explanation for the relationship between smoking

and drinking: while the size of the relationship between schooling and smoking is relatively large and consistent across datasets, this coefficient is very small for drinking—in some cases, the coefficient even suggests opposite associations, where more schooling makes an individual more likely to drink heavily. The results imply that schooling is questionable as a common cause of both smoking and drinking. Although these results may be surprising, they are consistent with prior studies that use within MZ-twins designs, including [2, 7, 6, 21, 33]. These papers generally find that the cross-sectional associations between schooling and health largely overstate the potential relationship—in many cases, the relationship becomes very small in magnitude and loses statistical significance. The estimates from this paper differ from studies of the effect of schooling that use natural experiments and instrumental variables [13, 36]. Although most of these studies find that schooling has a plausibly causal effect on health, these results are only identified for very specific margins of the population, and thus are usually not generalizable to larger populations. Due to the wide range of within-twins differences in schooling and health-related behaviors, our results are identified for a larger subset of the population and come closer to estimating an average treatment effect (In Appendix Figs. 1–3 we show the distributions of within-twins differences in schooling and each of the behaviors—these graphs highlight the wide range of differences on which the within-MZ twins models are estimated over).

Finally, based on the results of the behavioral genetic analyses, we find that the greatest portion of variance for each health-related behavior is related to behavior-specific factors, suggesting that the causes of health-related behaviors are largely idiosyncratic. We also find that genetic endowments are consistent with significant portions of the variance in most of the behaviors. These two results have been found in other behavioral genetic studies on the heritability of individual behaviors [5, 40, 60, 61]—these studies find small contributions from environments, reasonably large genetic contributions, and large individual environment contributions. However, we find that genetic endowments are not consistent with the covariation between the behaviors. The lack of support for a common set of genes that causes multiple unhealthy behaviors may arise if the elevated risk of mortality for individ-

uals with these gene expressions resulted in selective genetic pressure over time—effectively selecting out such sets of genes. Despite the idiosyncratic origins of the health-related behaviors, we find consistent evidence that the correlation between smoking and unhealthy drinking is associated with a common environmental factor: a large part of the correlation between smoking and unhealthy drinking is consistent with a common source of the shared, mostly childhood, environment between twins. This finding suggests that modifying the childhood environment may provide a plausible policy solution to reduce both smoking and unhealthy drinking behavior in adulthood.

In interpreting the results of this study, it is important to address some limitations of our study design. In order for the within-MZ estimates to be causal, we have to assume that the cause of the within-twins difference in schooling was unrelated to the within-twins difference in behaviors, except through schooling, though the violation of this condition produces predictable bounds on the causal estimates (see: Kohler et al., 2011). Furthermore, the outcome variable for one twin cannot depend on the outcome variable for another twin beyond their joint dependence on genetic endowments and childhood environments, although the violation of this condition produces predictable biases that have been discussed extensively elsewhere (see: Kohler et al., 2011). For our estimates of the variance attributable to common environments, we also assume that the common environments of MZ twins are the same as the common environment of DZ twins. However, this assumption only applies to the behavioral genetics models and is not needed for the within-MZ twins estimates. After controlling for any unobserved difference between twins through the within-twins estimates, we assume that the population of twins is representative of the larger American population and that the underlying causes of schooling and health-related behaviors are the same for twins as for the American population. The samples are overwhelming white, and the results estimated might not be generalizable to the unique childhood contexts experienced by other race/ethnic groups in the United States or in other societies if there are interactive race/ethnic effects. Twins studies in general have been criticized for several reasons. For example, studies have found that MZs are not perfectly identical genetically, especially when

considering epigenetic processes [47]. Although such considerations mean that the control for unobservable factors afforded by MZs is less than it would be if they also controlled for epigenetic processes, they do not negate the substantial advantages of twin controls over uncontrolled population-based studies that simply ignore genetic processes and unobserved childhood family background characteristics in exploring associations between risks and outcomes. Similarly, the validity of the so-called equal environment assumption, which holds that MZs share no more common environmental experiences than DZs, has been questioned [29]. Nevertheless, this hypothesis is testable and has generally been supported in the literature [31]. Moreover it is not relevant for the within-MZ estimates. Yet another criticism holds that modern genomic methods and detailed biological understanding of genomics have caused twins-based methods to become antiquated. However, considering that Genome Wide Association Studies (GWAS) often identify only very small single-gene effects on health and behaviors, twins and related study designs continue to be relevant to obtain a comprehensive assessment of the genetic and social determinants of health and health-related behaviors [58]. Finally, researchers have questioned whether twins samples are representative of the populations from which they were drawn. Once again, this hypothesis is testable, and studies have generally reported little or no differences between twins and singleton populations with the exception of birth weights. For example, a recent study that performed MRI brain scans found no significant differences between twins and unrelated, age- and sex-matched singletons in several brain structures [44]. Moreover within-twins estimates control for the additive effect of whatever might be distinctive about being a twin. There is a threat that the smaller coefficients and larger standard errors of the within-twins estimates is due to magnifying of measurement error (Bound and Solon, 1999). While the MTR data ask about co-twin data, allowing for the possibility of instrumenting, the other datasets did not permit this. While this is an important consideration, the results from instrumental variable regression for the MTR sample suggest that measurement error is not driving our results (Appendix Table 3). The MIDUS and MTR samples had a large degree of individuals dropped for incomplete data. In Appendix Table 4, we show the mean levels

of the main variables for those included and excluded and find that most of the variables are similar with differences across smoking and sex. However, these differences would only bias our result if the estimated relationships displayed interaction effects with the unbalanced variables. Importantly, our results may still be biased if those excluded were different from the included sample in unobserved ways that related to both schooling and health-related behaviors. Similarly, if individuals were missing due to premature mortality resulting from multiple poor health-related behaviors, we may underestimate the covariation between poor behaviors, since those with the greatest correlation would be dropped. Given the average ages of the samples, however, the role of selective mortality is likely minor.

Despite these limitations, our study is one of the first to explicitly examine the role of schooling, genetic endowments, and environments as common causes of multiple health-related behaviors. By presenting analyses common to both economics and behavioral genetics, we are able to provide a rich examination of the relationship between multiple health-related behaviors and their causes. We find that most health-related behaviors in adulthood are largely idiosyncratic and likely not caused by single factors, whether that is schooling, genetics, or environments. Our results suggest that programs that categorically target all health-related behaviors in adulthood may not produce changes across all behavioral domains—policies to improve health-related behaviors might be most effective if targeted at specific behaviors. Similarly, research on the causes of health-related behaviors should consider each behavior uniquely. The one prominent exception to this pattern is the relationship between smoking and unhealthy drinking: although the environmental correlation between these two is modest, our results suggest that a common aspect of the childhood and adolescent environment is consistent with variation in both behaviors. Research and policy to identify and modify this source may provide a strong way to reduce the population health burden of smoking and heavy drinking.



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## APPENDIX

### Chapter 1 Appendices

#### *Appendix A: Creating the Wealth Index*

To create a wealth index, I follow the general procedure used by the Demographic and Health Surveys (DHS). I used the following variables as inputs into the index:

1. Ownership of the following assets
  - a. House or land for living
  - b. Other building
  - c. Other land
  - d. Poultry
  - e. Livestock
  - f. Hard-stemmed plant used for business
  - g. Vehicles
  - h. Household appliances
  - i. Savings, certificates of deposits/stocks
  - j. Jewelry
  - k. Receivables
  - l. Household furniture and utensils
  - m. Other assets
2. Roof material
  - a. Concrete
  - b. Wood
  - c. Metal plates
  - d. Shingles
  - e. Asbestos
  - f. Foliage/palm leaves/grass/bamboo
3. Wall material
  - a. Masonry
  - b. Lumber/board/plywood



- c. Bamboo/woven/mat
- 4. Floor Material
  - a Ceramic/marble/granite/stone
  - b Tiles
  - c Cement/bricks
  - d Lumber/board
  - e Bamboo
  - f Dirt

To generate the wealth index, I first converted each of the variables into 0/1 dummies and then conducted principle components analysis on each of the entire set of variables (see results below). Based on the approach used by the DHS and in prior studies (see Filmer and Pritchett 2001), I took the first principle component to represent wealth and then created an index as the weighted sum of each of the dichotomous variables, with the weights coming from the value of the first principle component. Individuals were then classified into wealth quartiles based on the estimated wealth index. This process was conducted separately for urban and rural households to capture living standard differences.

#### *Appendix B: Period Life Table Estimation*

##### *Data*

In 2007 and 2008 the IFLS visited the households of the participants of the previous waves. For each target household, a full household roster was collected with basic demographic information for all individuals. In 2014, the tracking status of all target individuals was ascertained. If an individual had died, a relative knowledgeable about the deceased would provide an exit interview with date of death information. Based on date of birth and date of death information, I created an age at survey in 2007 and age at death variable. For individuals who did not die, I right censored the sample at January 1st, 2014, and created an age at survey exit variable. I then converted the data into a person-age format. For example, if an individual was 40 years old in 2007 and 47 years old on January 1st, 2014, this

individual would contribute observations for ages 40 through 46—they would not contribute an observation for 47 since they did not complete the year at the time of survey. Similarly, if an individual was 40 in 2007 and died in 2010 at the age of 43, they would contribute 4 observations: observations for the ages 40, 41, 42 where they would be marked as alive and an observation for the age of 43 where they would be marked as having died.

### *Estimating the Age-specific Probabilities of Dying*

The age-specific probabilities or hazards of mortality were then calculated for each SES group by first estimating the following logistic regression model on the person-age observations separately by sex and urban/rural residence (in a survival analysis framework, this is exactly equivalent to a discrete failure-time model):

$$\ln\left(\frac{p}{1-p}\right) = \beta_0 + \beta_1 * \text{age} + \beta_2 * \text{SES}_2 + \beta_3 * \text{SES}_3 + \beta_4 * \text{SES}_4$$

Here the three SES variables are dummies for the 2nd, 3rd, and 4th quartiles of either consumption/expenditure or wealth (depending on the model). The linear in the logit specification of age is a variant of the Gompertz-Makeham mortality hazard. This mortality hazard has been shown to widely apply to adult mortality above the age of 30. Importantly, in many cases, mortality in the very old adult ages is observed to decelerate and deviate from the Gompertz-Makeham fit. However, demographers have shown that this deceleration is likely not due to a true deceleration of mortality but rather poor data quality and age misreporting in the older ages. To address this problem, many studies predict mortality using a Gompertz-Makeham hazard between the ages of 30 and 80 and then extrapolate this mortality pattern to ages above 80. Indeed, plotting the observed mortality rates against the estimated Gompertz-Makeham hazard in the IFLS showed this exact pattern, with a deviation and declaration from the Gompertz-Makeham fit above age 80. Therefore, I followed the standard procedure and estimated the mortality model for ages 30 to 80. I then used the model to predict the probability of dying for each age between 30 and 100. For example, the age-specific probability of dying between ages 30 and 31,  ${}_1q_{30}$ , for someone

in the second wealth quartile would be:

$${}_1q_{30}^{\text{SES2}} = \frac{e^{\beta_0} e^{\beta_1 * 30} e^{\beta_2}}{1 + e^{\beta_0} e^{\beta_1 * 30} e^{\beta_2}}.$$

These predicted probabilities can be used to construct life tables.

### *Constructing the Summary Measures of Mortality*

After predicting the age-specific probabilities of dying for each SES-sex-urban/rural group, I constructed period life tables starting at age 30 for each group using standard life table procedures. Life expectancy at age 30 ( $e_{30}$ ) was then simply calculated as:

$$e_{30} = \frac{T_{30}}{l_{30}}$$

where  $T_{30}$  is the total number of person-years lived above age 30 in the life table and  $l_{30}$  is the starting size of the life table cohort. The probability of dying between the ages of 30 and 60 ( ${}_{30}q_{30}$ ) was calculated as:

$${}_{30}q_{30} = \frac{l_{60}}{l_{30}}$$

where  $l_{60}$  is the number of survivors to age 60 in the life table.

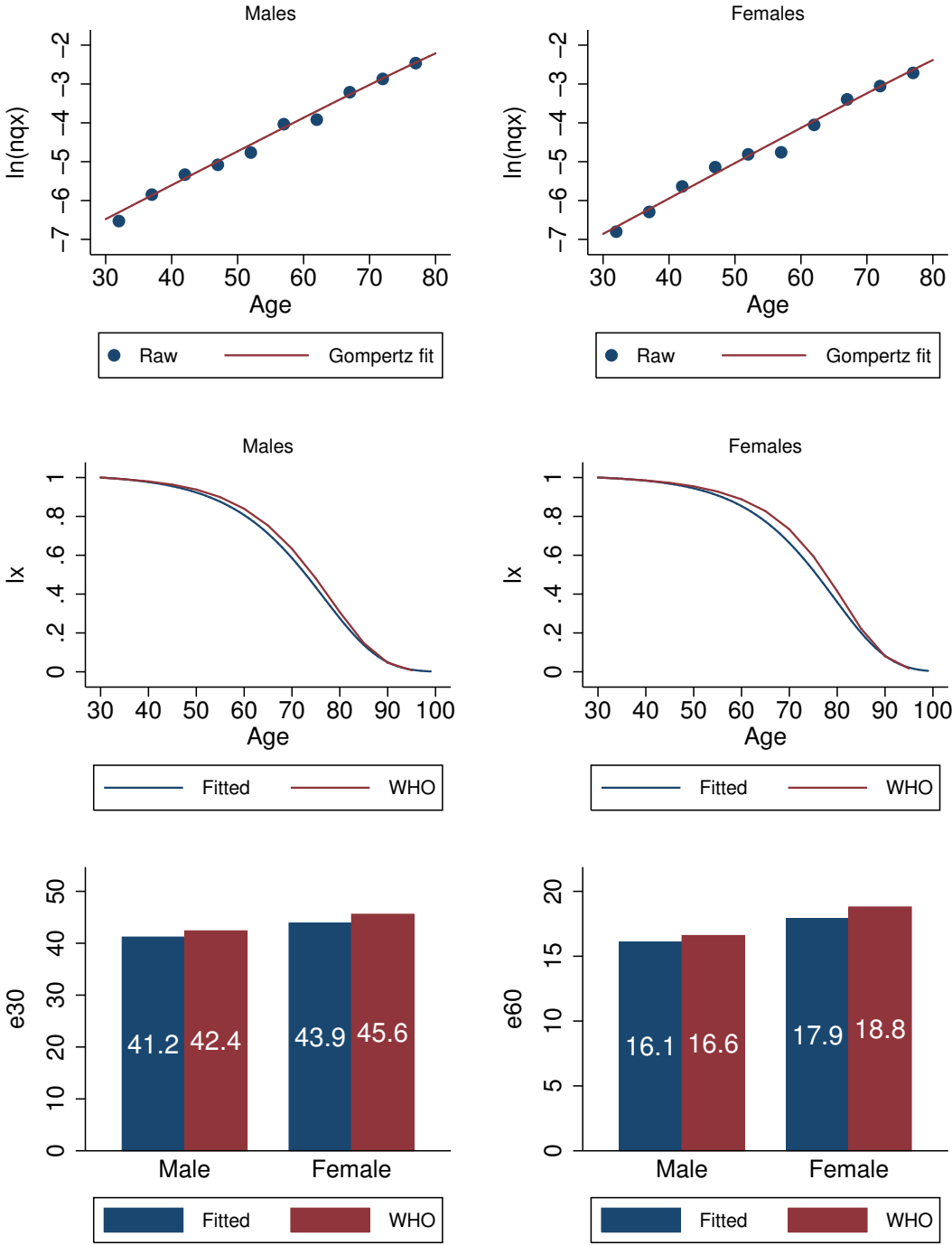
### *Variance and Trend Estimation*

I used a simulation procedure to estimate the variance of each of the estimated measures. This involved the following steps: I first drew 100 samples from the joint distribution of the beta coefficients in the mortality model presented above. I then estimated age-specific mortality probabilities for each of the 100 sets of beta coefficients, then, used these estimated mortality probabilities to construct  $e_{30}$  and  ${}_{30}q_{30}$  for each of the 100 simulated sets of mortality rates. The 95% confidence interval was then estimated as the 5th and 95th percentiles of the empirical distribution of  $e_{30}$  and  ${}_{30}q_{30}$ .

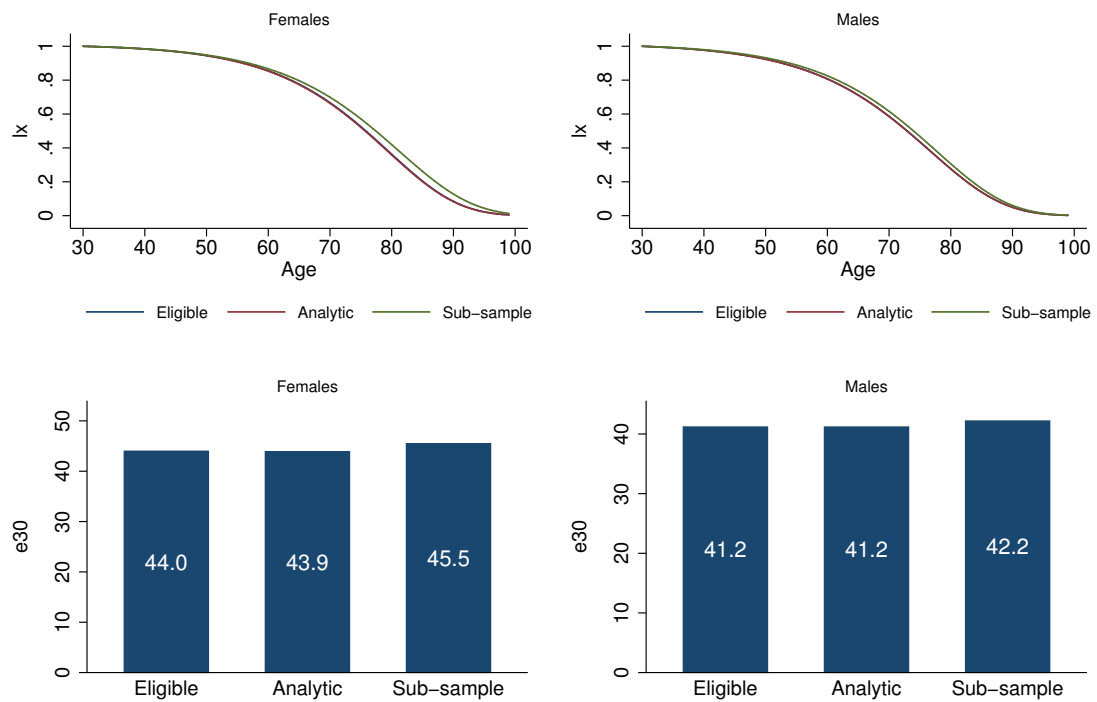
To estimate the trend and trend p-value over SES quartiles I estimated a linear re-

gression of  $e_{30}$  and  ${}_{30}q_{30}$  on a linear SES quartile term for each of the 100 simulated life tables. The estimated trend was then the average beta coefficient over the 100 simulated life tables. The standard error of this average was simply the standard deviation of the beta coefficients across the 100 simulated life tables. The p-value was then calculated using a t-test with the estimated mean and standard error.

Appendix Figure 1.1: Mortality model diagnostics, IFLS, N = 17,925, 2007-2015



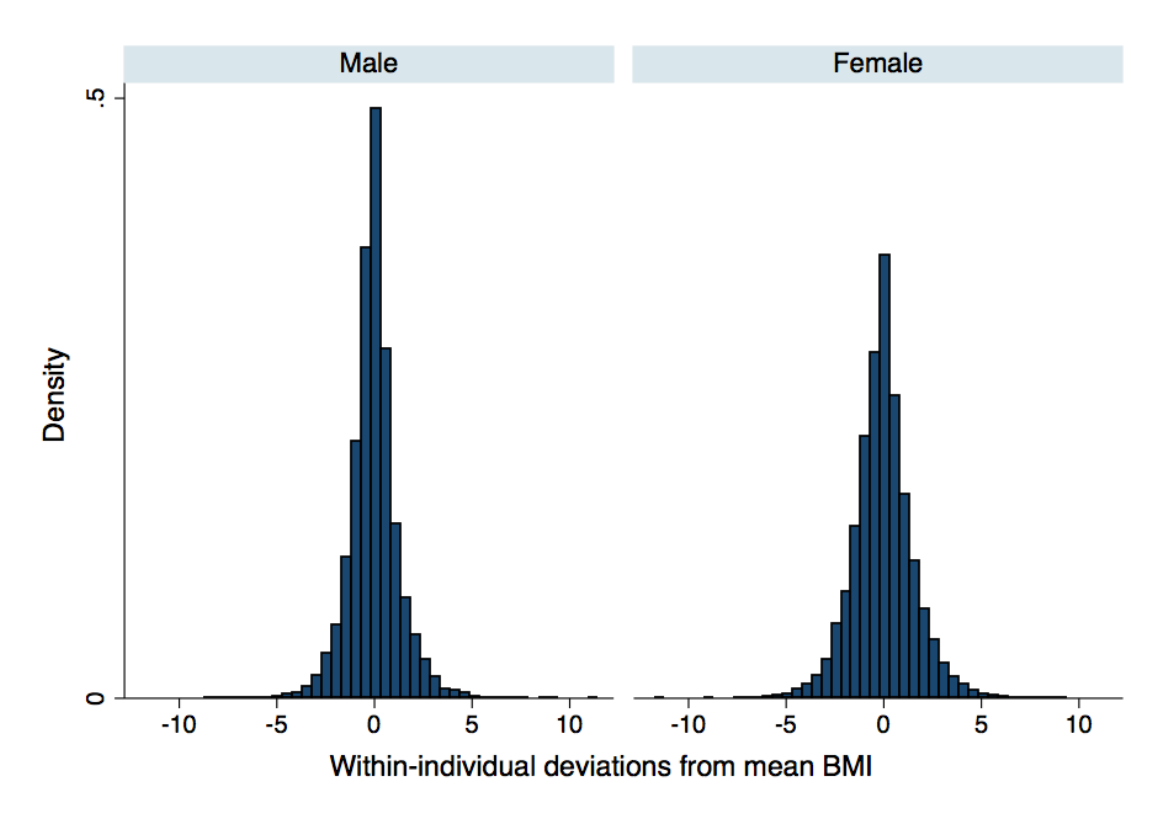
Appendix Figure 1.2: Estimated survival curves and life expectancy at age 30 for the full eligible sample, the analytic sample, and the health risk factor subsamples, Indonesian Family Life Survey, 2007-2015



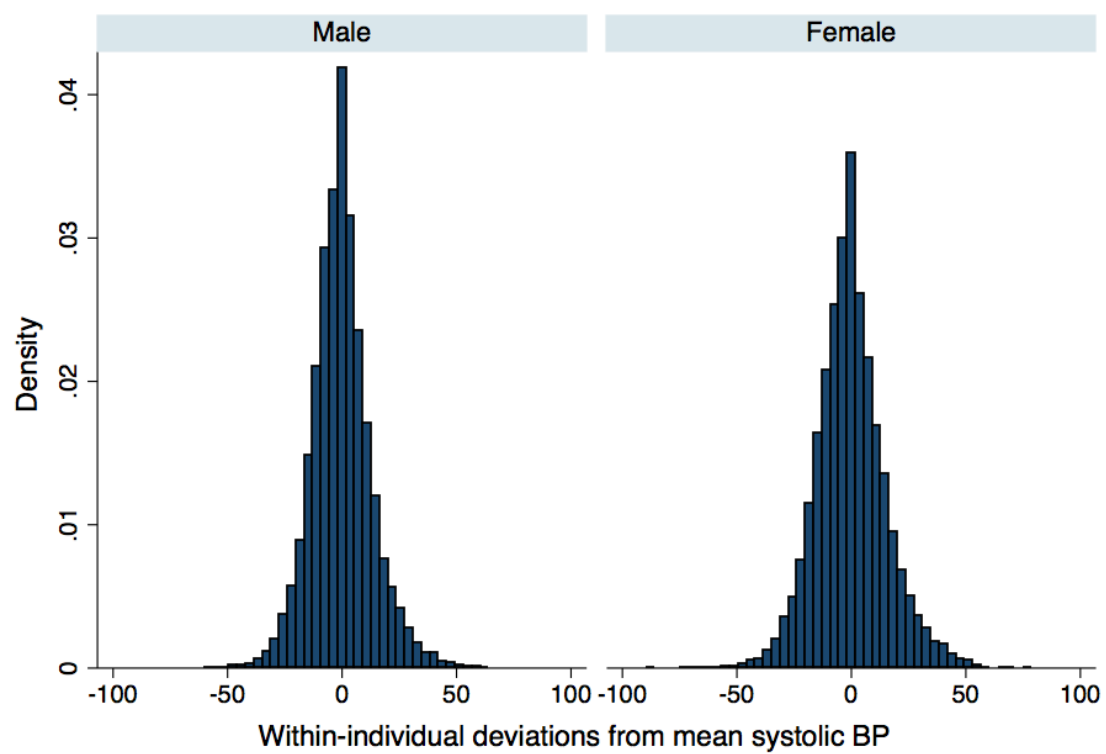
## Chapter 2 Appendices

### *Appendix A: Within-individual variation*

Appendix Figure 2.1: Within individual variation in BMI, 1997-2014, Indonesian Family Life Survey, N = 33,119



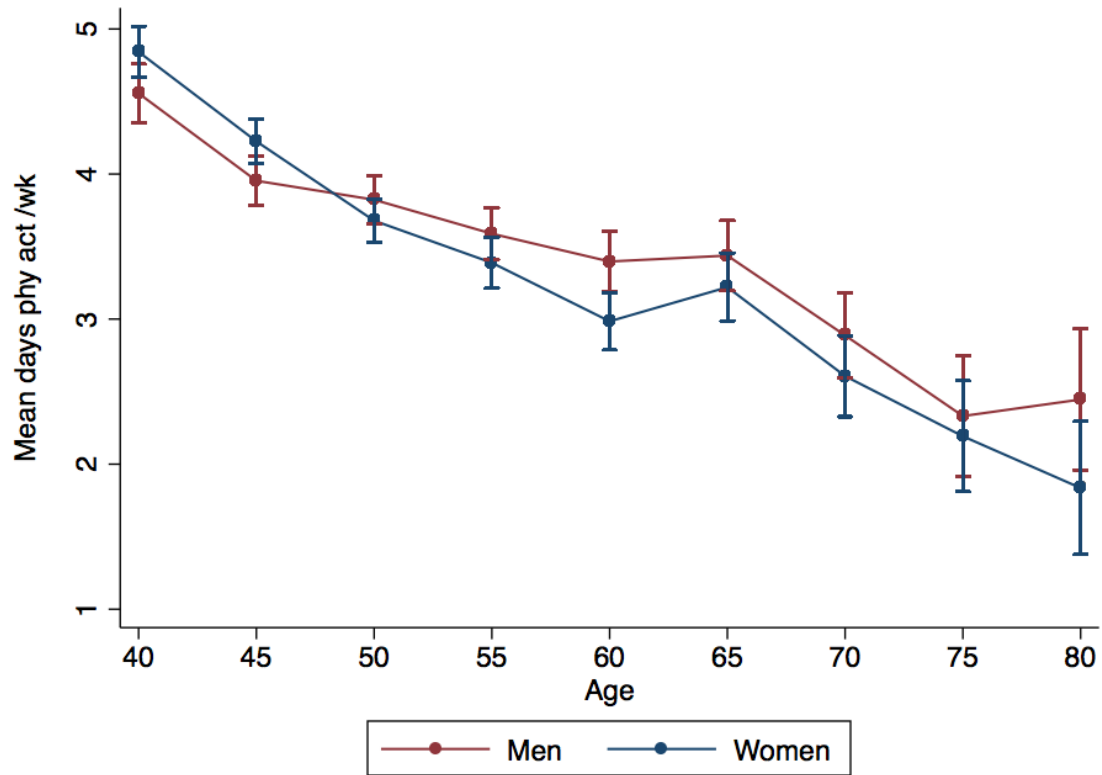
Appendix Figure 2.2: Within individual variation in systolic BP, 1997-2014, Indonesian Family Life Survey, N = 33,199



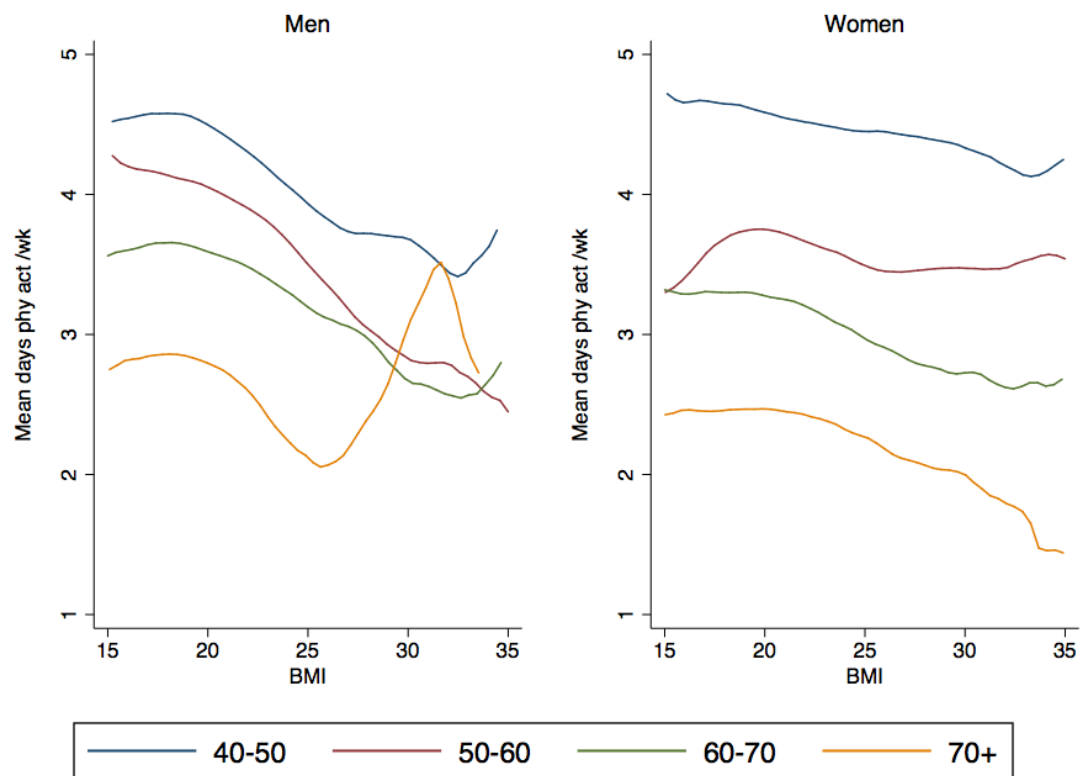


*Appendix B: Results with physical activity*

Appendix Figure 2.3: Age patterns of physical activity by sex, Indonesian Family Life Survey, 2007-2014. Data were pooled over both survey years. N = 13,485



Appendix Figure 2.4: Relationship between body mass index and physical activity by sex and 10 year age groups, Indonesian Family Life Survey, 2007-2014. Data were pooled over both survey years. Results are smoothed using a 2 unit moving average. N = 13,485



Appendix Table 2.1: Estimated relationships between BMI and systolic blood pressure additionally adjusting for physical activity, Indonesian Family Life Survey, 2007-2014

	Men		Men		Women		Women	
	Pooled OLS	Pooled OLS + CEM	Fixed Effects	Pooled OLS	Pooled OLS + CEM	Fixed Effects	Pooled OLS	Fixed Effects
Body mass index	1.769*** (1.581 - 1.956)	1.760*** (1.583 - 1.937)	1.653*** (1.158 - 2.148)	1.318*** (1.152 - 1.483)	1.312*** (1.187 - 1.437)	1.485*** (1.153 - 1.816)		
Observations	5,985	5,985	5,985	7,500	7,500	7,500		
R-squared	0.160	0.088	0.196	0.163	0.060	0.220		
Overall Mean	140.53	140.53	140.53	144.21	144.21	144.21		
Mean physical activity	3.6	3.6	3.6	3.6	3.6	3.6		

Standard errors are clustered by individual. 95% CI in parentheses.

\*\*\* p<0.001, \*\* p<0.01, \* p<0.05, + p<0.1

Notes: The pooled OLS and fixed effects models include the following covariates: age (quadratic), period of observation, urban, schooling, religion, marital status, primary job, per capita expenditure (quadratic), and days of moderate of physical exercise per week (flexible). The OLS + CEM Model includes dummies for strata defined by 5 year age groups, urban residence, schooling, and period of observation in addition to covariates for religion, marital status, primary job, per capita expenditure (quadratic), and days of moderate of physical exercise per week (flexible).

Appendix Table 2.2: Estimated relationships between BMI and systolic blood pressure stratified by mean BMI additionally adjusting for physical activity, Indonesian Family Life Survey, 2007-2014

	Mean BMI	Mean BMI	Mean BMI	Mean BMI
Men	15-20	20-25	25-30	30-35
Body mass index	1.345* (0.176 - 2.513)	2.009*** (1.406 - 2.612)	0.847+ (-0.143 - 1.837)	-1.208 (-4.368 - 1.953)
Observations	1,803	2,894	1,103	185
R-squared	0.164	0.241	0.252	0.411
Women	15-20	20-25	25-30	30-35
Body mass index	1.987** (0.802 - 3.173)	1.184*** (0.692 - 1.677)	1.583*** (1.028 - 2.139)	1.152* (0.204 - 2.099)
Observations	1,520	3,080	2,306	594
R-squared	0.237	0.240	0.237	0.256

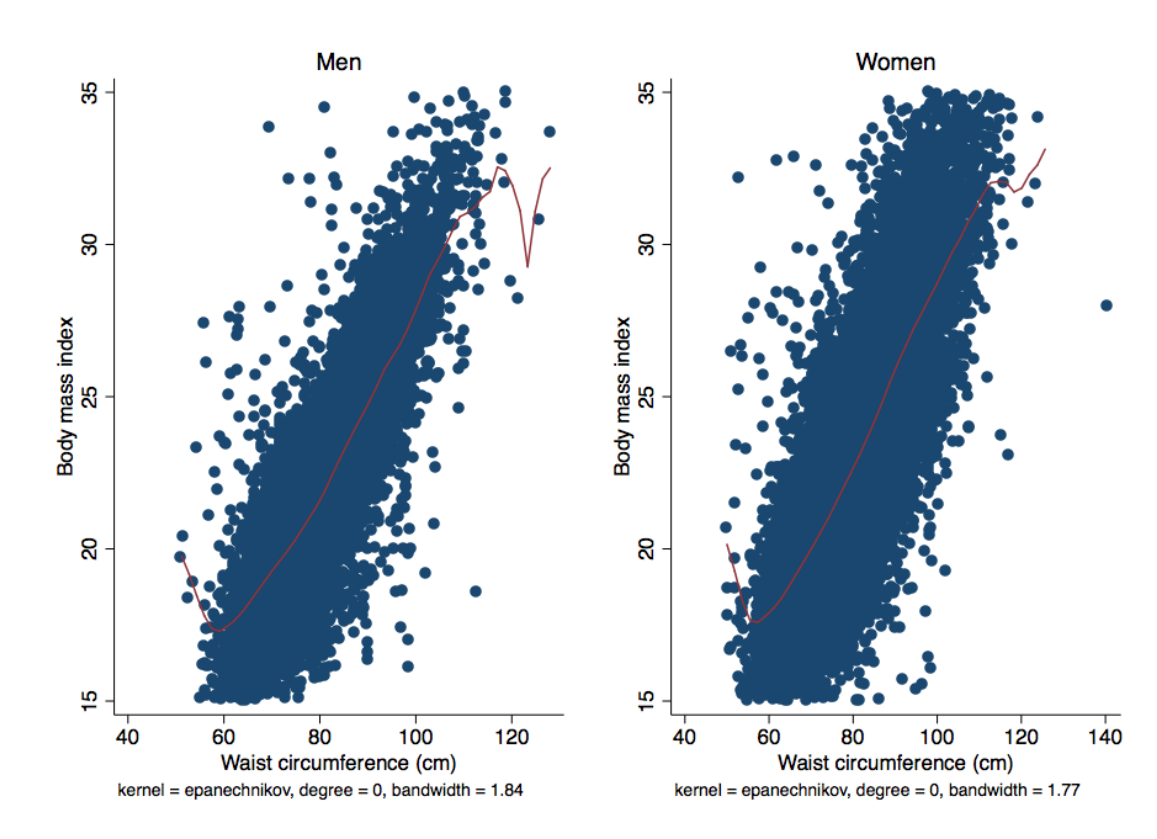
Standard errors are clustered by individual. 95% CI in parentheses.

\*\*\* p<0.001, \*\* p<0.01, \* p<0.05, + p<0.1

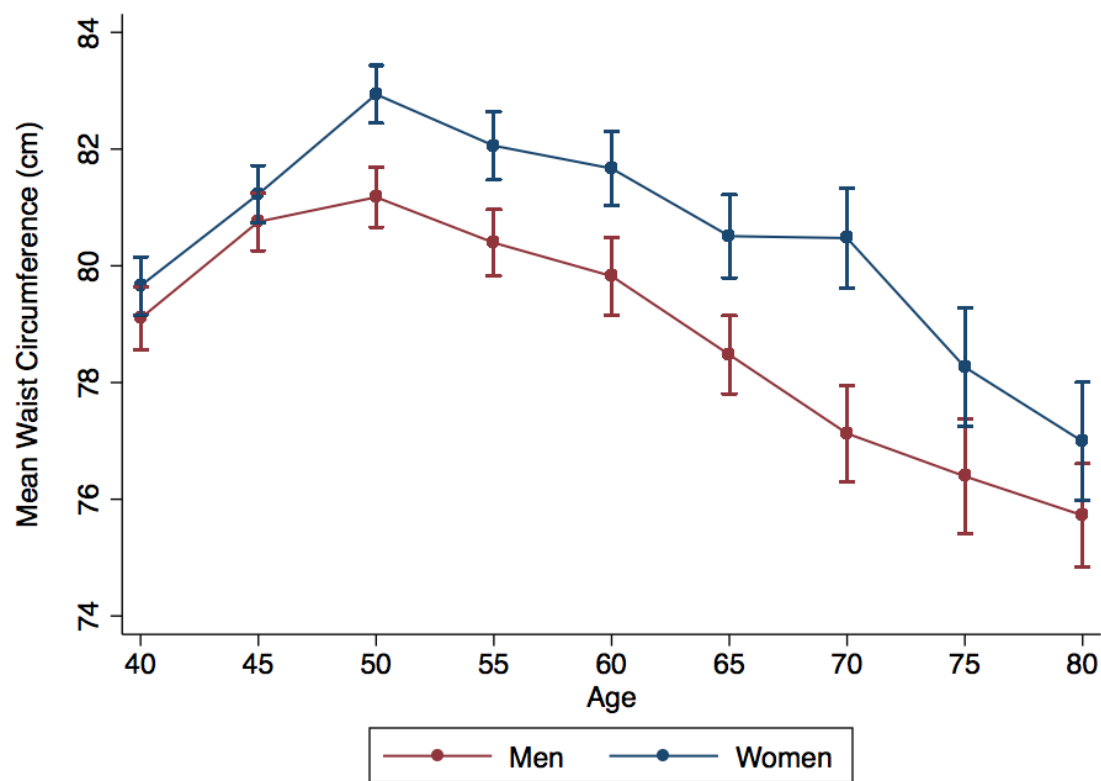
Notes: Models include the following covariates: age (quadratic), period of observation, urban, schooling, religion, marital status, primary job, per capita expenditure (quadratic), and days of moderate of physical exercise per week (flexible).

*Appendix C: Results for waist circumference*

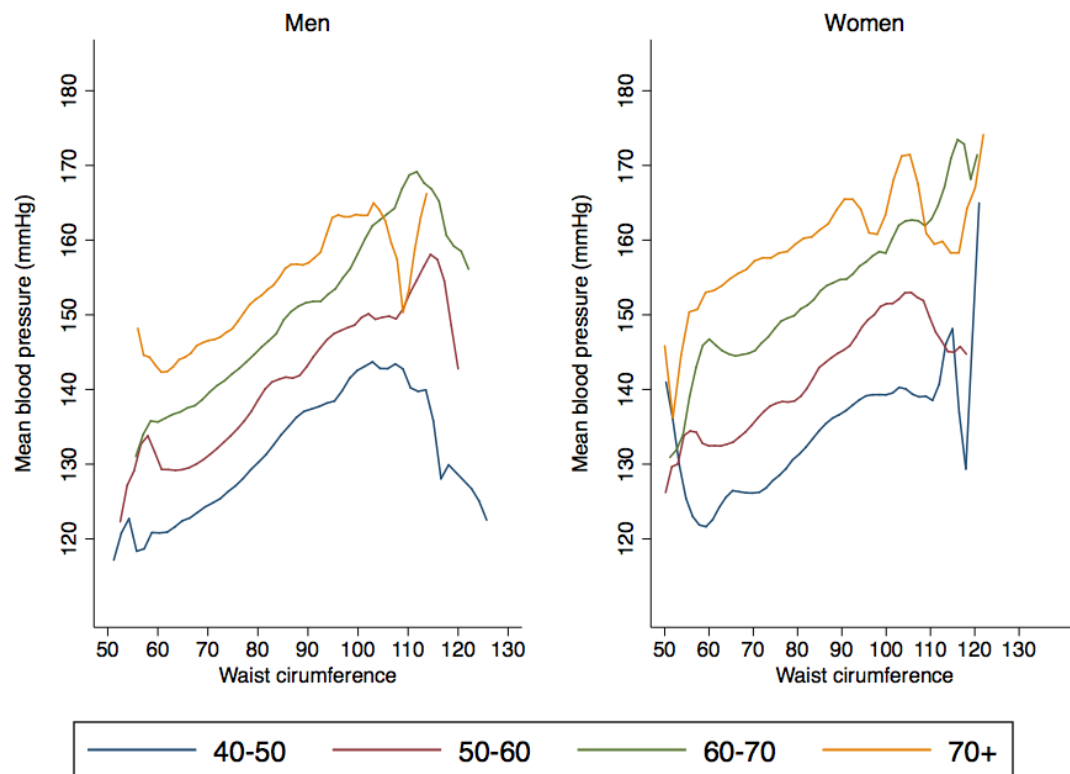
Appendix Figure 2.5: Estimated relationship between BMI and waist circumference, 2000-2014. Data were pooled over the three survey years. N = 21,914



Appendix Figure 2.6: Age patterns of waist circumference by sex, Indonesian Family Life Survey, 2000-2014. Data were pooled over the three survey years. N = 21,914



Appendix Figure 2.7: Estimated mean systolic blood pressure by waist circumference .  
Results are smoothed using a locally weighted mean by 2 unit bins. N = 21,914



Appendix Table 2.3: Estimated relationships between waist circumference and systolic blood pressure, Indonesian Family Life Survey, 2000-2014

	Men		Men		Women		Women	
	Pooled OLS	Pooled OLS + CEM	Fixed Effects	Pooled OLS	Pooled OLS + CEM	Fixed Effects	Pooled OLS	Fixed Effects
Waist circumference (cm)	0.586*** (0.0284)	0.586*** (0.0240)	0.243*** (0.0312)	0.461*** (0.0257)	0.464*** (0.0196)	0.394*** (0.0420)		
R-squared	0.170	0.076	0.338	0.180	0.056	0.310		
Overall Mean	138.59	138.59	138.59	141.87	141.87	141.87		

Standard errors are clustered by individual. 95% CI in parentheses.

\*\*\* p<0.001, \*\* p<0.01, \* p<0.05, + p<0.1

Notes: The pooled OLS and fixed effects models include the following covariates: age (quadratic), period of observation, urban, schooling, religion, marital status, primary job, and per capita expenditure (quadratic). The OLS + CEM Model includes dummies for strata defined by 5 year age groups, urban residence, schooling, and period of observation in addition to covariates for religion, marital status, primary job, and per capita expenditure (quadratic).



Appendix Table 2.4: Estimated relationships between waist circumference and systolic blood pressure stratified by mean waist circumference, Indonesian Family Life Survey, 2000-2014

Men	Mean waistcir 40-60	Mean waistcir 60-80	Mean waistcir 80-100	Mean waistcir 100+
Waist circumference (cm)	0.238+ (-0.0301 - 0.505)	0.357*** (0.232 - 0.482)	0.327*** (0.187 - 0.467)	1.084*** (0.746 - 1.422)
Observations	1,386	4,244	2,577	1,084
R-squared	0.312	0.281	0.356	0.369
Women				
Waist circumference (cm)	0.272* (0.0449 - 0.500)	0.222*** (0.110 - 0.334)	0.169** (0.0639 - 0.274)	0.399** (0.121 - 0.677)
Observations	1,492	4,052	3,897	1,670
R-squared	0.324	0.317	0.382	0.339

Standard errors are clustered by individual. 95% CI in parentheses.

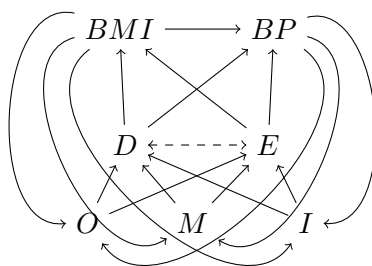
\*\*\* p<0.001, \*\* p<0.01, \* p<0.05, + p<0.1

Notes: Models include the following covariates: age (quadratic), period of observation, urban, schooling, religion, marital status, primary job, and per capita expenditure (quadratic).

# *Appendix D: Results without potentially endogenous variables*

One important consideration is the potential for bias introduced by conditioning on variables that are caused by both BMI and hypertension. For example, if high BMI and high blood pressure both cause lower earnings, or make an individual less likely to be married, conditioning on income and marital status in the multivariate models may inflate the size of the estimated relationship (this is known as collider or endogenous selection bias – shown in the DAG below). In this appendix, I re-estimate the multivariate models without adjusting for potentially endogenous variables (income, job, and marital status) and find very little change to the estimated effects.

Appendix Figure 2.8: Causal relationship between body mass index and blood pressure with collider bias



Appendix Table 2.5: Estimated relationships between BMI and systolic blood pressure without potential colliders, Indonesian Family Life Survey, 1997-2014

	Men		Men		Men		Women		Women	
	Pooled OLS		Pooled OLS + CEM		Fixed Effects		Pooled OLS		Pooled OLS + CEM	
Body mass index	1.744*** (1.599 - 1.888)		1.765*** (1.655 - 1.876)		1.674*** (1.457 - 1.892)		1.209*** (1.084 - 1.335)		1.207*** (1.121 - 1.292)	
										1.308*** (1.137 - 1.478)
Observations	16,121		16,121		16,121		19,998		19,998	
R-squared	0.206		0.070		0.281		0.242		0.042	
Overall Mean	135.43		135.43		135.43		137.82		137.82	

Standard errors are clustered by individual. 95% CI in parentheses.

\*\*\* p<0.001, \*\* p<0.01, \* p<0.05, + p<0.1

Notes: The pooled OLS and fixed effects models include the following covariates: age (quadratic), period of observation, urban, schooling, and religion. The OLS + CEM Model includes dummies for strata defined by 5 year age groups, urban residence, schooling, and period of observation in addition to covariates for religion.

Appendix Table 2.6: Estimated relationships between BMI and systolic blood pressure without potential colliders stratified by mean BMI, Indonesian Family Life Survey, 1997-2014

	Mean BMI 15-20	Mean BMI 20-25	Mean BMI 25-30	Mean BMI 30-35
Men				
Body mass index	2.125*** (1.653 - 2.598)	1.301*** (1.000 - 1.603)	1.233*** (0.802 - 1.663)	1.515 (-0.899 - 3.929)
Observations	5,529	8,140	2,273	179
R-squared	0.236	0.284	0.324	0.271
Women				
Body mass index	1.930*** (1.424 - 2.436)	1.046*** (0.800 - 1.292)	1.033*** (0.737 - 1.330)	1.011** (0.376 - 1.647)
Observations	4,975	9,314	4,785	924
R-squared	0.274	0.338	0.347	0.386

Standard errors are clustered by individual. 95% CI in parentheses.

\*\*\* p<0.001, \*\* p<0.01, \* p<0.05, + p<0.1

Notes: Models include the following covariates: age (quadratic), period of observation, urban, schooling, and religion.

*Appendix E: Results for diastolic blood pressure*

Appendix Table 2.7: Estimated relationships between BMI and diastolic blood pressure, Indonesian Family Life Survey, 1997-2014

	Men		Men		Men		Women		Women	
	Pooled OLS		Pooled OLS + CEM		Fixed Effects		Pooled OLS		Pooled OLS + CEM	
Body mass index	1.113*** (1.030 - 1.196)		1.106*** (1.047 - 1.166)		1.040*** (0.909 - 1.171)		0.876*** (0.812 - 0.940)		0.865*** (0.819 - 0.910)	
										0.933*** (0.842 - 1.024)
Observations	16,121		16,121		16,121		19,998		19,998	
R-squared	0.112		0.088		0.085		0.094		0.072	
Overall Mean	82.13		82.13		82.13		82.60		82.60	
	Standard errors are clustered by individual. 95% CI in parentheses.									

\*\*\* p<0.001, \*\* p<0.01, \* p<0.05, + p<0.1

Notes: The pooled OLS and fixed effects models include the following covariates: age (quadratic), period of observation, urban, schooling, religion, marital status, primary job, and per capita expenditure (quadratic). The OLS + CEM Model includes dummies for strata defined by 5 year age groups, urban residence, schooling, and period of observation in addition to covariates for religion, marital status, primary job, and per capita expenditure (quadratic).

## *Appendix F: Estimating the coarsened match*

The goal behind any matching algorithm is to improve inference by selecting valid counterfactuals for each “treated” individual. Ideally, treated and control individuals should be exactly matched on all the characteristics that may confound the treatment-outcome relationship; however, as the number of covariates increases, the number of strata rapidly increases, increasing the difficulty of the match substantially. For example, even with 30 single-year ages, 4 education groups, and 2 sexes, there are already 240 strata. Coarsened exact matching seeks to solve the issue of dimensionality by matching individuals within regions of the covariates, rather than exact values. For example, rather than matching on single-year ages, individuals could be matched on 5-year age groups. By specifying ranges for each covariate, or for a subset of the covariates, the size of the covariate space decreases substantially.

To conduct the match, I first selected age, schooling, urban/rural residence, and period of observation as matching variables. I then “coarsened” the match for age and schooling by specifying the algorithm to match within 5-year age groups and three groups of schooling (no schooling, primary, secondary or beyond) using the “cem” Stata command. This results in  $11 \times 4 \times 3 \times 2 \times 2 = 528$  strata; in practice however, only 498 strata contained both an treatment and control individual.

At this point, the estimate could be calculated by looping through each of the strata and averaging across strata, taking into account the number of observations per strata. An alternative way, that also allows for additional controls, is to use a regression with fixed effects for strata. I followed this approach by estimating an OLS regression of blood pressure on dummy variables for each of the strata along with covariates for religion, marital status, primary job, and per capita household expenditure:

$$bp_i = \sum_{j=2}^{498} \delta_j + \sum X\beta + \epsilon_i$$

Appendix Table 2.8: Estimated relationships between BMI and diastolic blood pressure stratified by mean BMI, Indonesian Family Life Survey, 1997-2014

Men	Mean BMI 15-20	Mean BMI 20-25	Mean BMI 25-30	Mean BMI 30-35
Body mass index	1.350*** (1.086 - 1.614)	0.998*** (0.809 - 1.187)	1.191*** (0.929 - 1.453)	1.221+ (-0.102 - 2.545)
Observations	5,529	8,140	2,273	179
R-squared	0.054	0.087	0.145	0.273
Women				
Body mass index	1.279*** (1.011 - 1.548)	0.952*** (0.819 - 1.085)	0.933*** (0.772 - 1.095)	0.543** (0.190 - 0.895)
Observations	4,975	9,314	4,785	924
R-squared	0.069	0.109	0.130	0.145

Standard errors are clustered by individual. 95% CI in parentheses.

\*\*\* p<0.001, \*\* p<0.01, \* p<0.05, + p<0.1

Notes: Models include the following covariates: age (quadratic), period of observation, urban, schooling, religion, marital status, primary job, and per capita expenditure (quadratic).



## Chapter 3 Appendices

Appendix Table 3.1: Likelihood ratio tests of alternative twin models, Add Health, MIDUS, and MTR twins

	# Parameters	-2logLL	Degrees of freedom	P-value
<i>Add Health</i>				
ACE	38	6467.586	2948	Reference
AE	28	6474.119	2958	0.769
CE	28	6540.775	2958	0.000
E	18	6880.66	2968	0.000
<i>MIDUS</i>				
ACE	36	13200.03	5990	Reference
AE	26	13233.94	6000	0.000
CE	26	13279.41	6000	0.000
E	16	14027.45	6010	0.000
<i>MTR</i>				
ACE	24	13592.4	6896	Reference
AE	18	13607.61	6902	0.019
CE	18	13703.8	6902	0.000
E	12	14422.4	6908	0.000

Appendix Table 3.2: Proportions, means, and standard deviations by zygosity

	Add Health		MIDUS		MTR	
	MZ	DZ	MZ	DZ	MZ	DZ
Categorical						
<i>Smoker</i>						
Never	63.1%	57.5%	66.3%	62.2%	62.3%	54.6%
Ever	31.3%	37.1%	20.1%	23.1%	25.4%	32.2%
Heavy	5.6%	5.4%	13.6%	14.7%	12.3%	13.1%
<i>Drinker</i>						
Never	23.5%	21.6%	23.7%	24.0%	29.8%	28.9%
Ever	57.5%	57.8%	57.2%	50.6%	65.6%	63.5%
Heavy	18.9%	20.7%	19.1%	25.4%	4.6%	7.6%
Continuous						
<i>Vigorous activity per week</i>						
Mean	2.4	2.5				
SD	2.5	2.6				
<i>Vigorous activity per month</i>						
Mean			6.4	6.3		
SD			5.3	5.4		
BMI						
Mean	28.0	28.2	26.4	26.9	25.8	25.9
SD	7.3	7.3	4.9	5.3	4.6	4.7

Appendix Table 3.3: Estimated OLS, within-MZ twin, and within-MZ twin IV regressions of smoking, drinking, and BMI on schooling, MTR Twins, N = 1,294

VARIABLES	OLS	Ever Smoker FE	FE IV	OLS	Heavy Smoker FE	FE IV	OLS	Ever Drinker FE	FE IV	OLS	Heavy Drinker FE	FE IV	OLS	BMI FE	FE IV
Years of schooling	-0.040*** (0.005)	-0.016 (0.010)	-0.012 (0.015)	-0.017*** (0.003)	-0.001 (0.007)	-0.001 (0.012)	-0.001 (0.005)	-0.003 (0.007)	0.002 (0.014)	0.004+ (0.002)	0.007 (0.005)	0.005 (0.008)	-0.160** (0.054)	0.041 (0.061)	0.096 (0.105)
Age	-0.008** (0.003)			-0.004* (0.002)			0.000 (0.003)			-0.003* (0.001)			-0.096*** (0.029)		
Male = 1	0.159*** (0.033)			0.034 (0.021)			0.197*** (0.029)			0.043** (0.015)			1.336*** (0.308)		
R-squared	0.073	0.005		0.025	0.000		0.042	0.000		0.021	0.003		0.038	0.001	

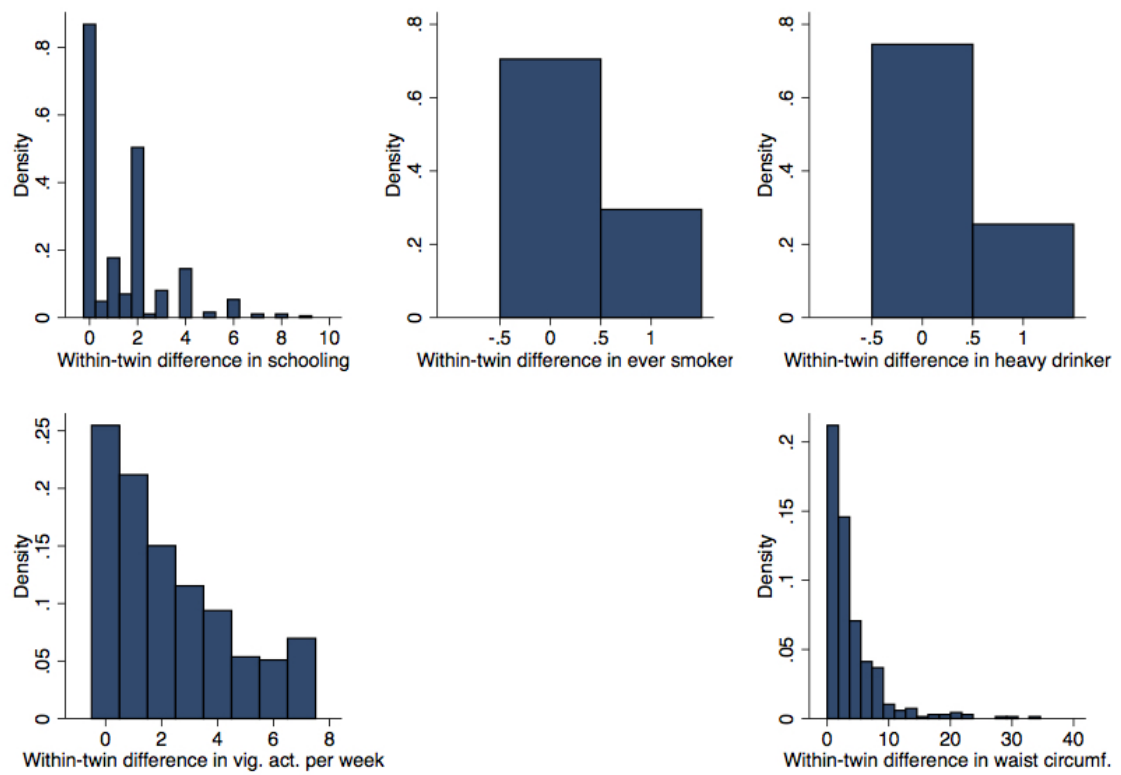
Standard errors are clustered by twinship. Linear probability models were estimated for dichotomous outcomes.  
 \*\*\* p<0.001, \*\* p<0.01, \* p<0.05, + p<0.1

Appendix Table 3.4: Means and percentages for main variables for included and missing samples

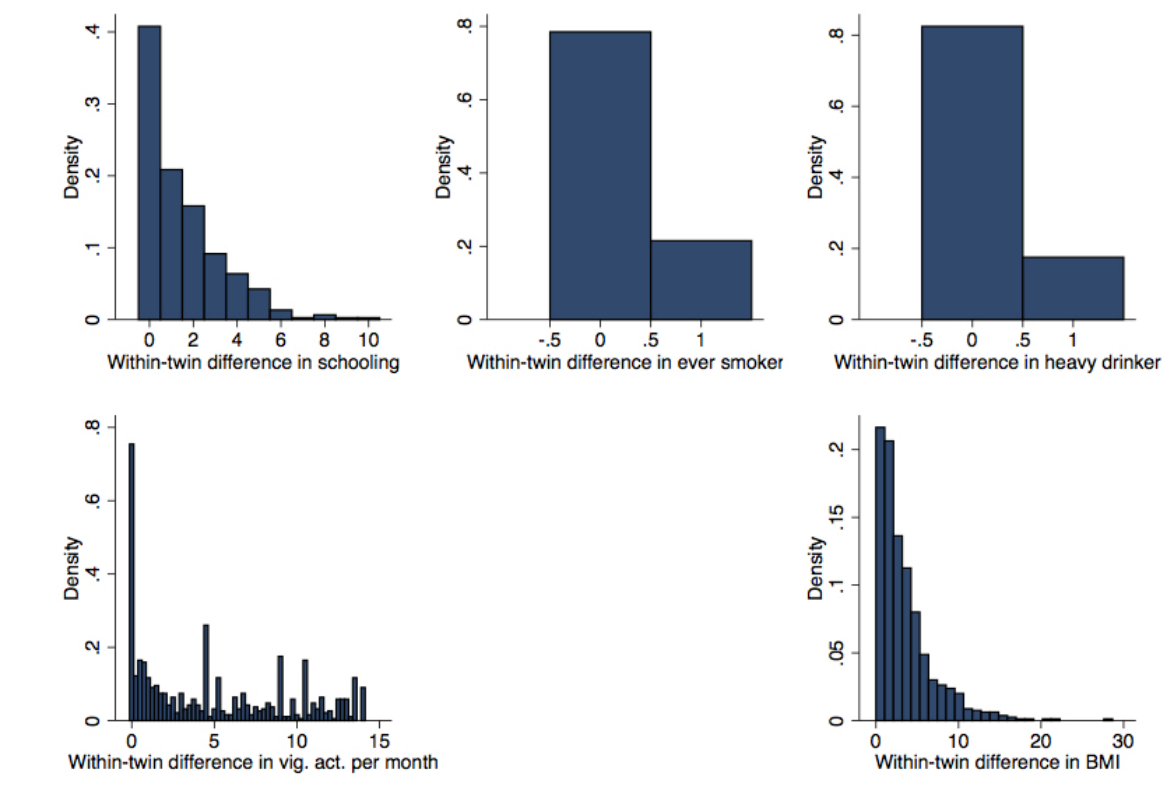
	Midus Wave 1			Midus Wave 2			MTR		
	In sample	Dropped	P-val	In sample	Dropped	P-val	In sample	Dropped	P-val
Age	45.0	43.5	0.038	52.9	54.7	0.009	47.1	47.3	0.360
Male	42.9%	44.1%	0.691	39.7%	45.4%	0.060	34.9%	40.5%	0.001
Years of schooling	13.7	13.6	0.726	14.3	14.2	0.704	13.7	13.4	0.003
Ever smoker	47.8%	46.1%	0.558	39.9%	45.5%	0.061	41.1%	45.0%	0.022
Heavy smoker	16.2%	23.8%	0.001	9.5%	12.9%	0.077	12.7%	14.3%	0.171
Ever drinker	84.9%	80.8%	0.065	57.6%	53.1%	0.137	70.6%	69.4%	0.432
Heavy drinker	29.2%	28.6%	0.853	6.6%	6.6%	0.970	5.9%	6.9%	0.245
BMI	26.2	40.6	0.000	27.4	35.2	0.000	25.8	26.0	0.274

Notes: The Add Health data were not included due to the small amount of missingness (5.6%)

Appendix Figure 3.1: Within-MZ twin difference in health-related behaviors, Add Health Twins, N = 373 twin pairs



Appendix Figure 3.2: Within-MZ twin difference in health-related behaviors, MIDUS Twins, N = 753 twin pairs



Appendix Figure 3.3: Within-MZ twin difference in health-related behaviors, MTR Twins, N = 1,153 twin pairs

