Healthy, Wealthy, and Wise?

New Evidence from AHEAD Wave 3

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ABSTRACT: This paper utilizes the Asset and Health Dynamics of the Oldest Old (AHEAD) Panel to test for the absence of causal links from socio-economic status (SES) to innovations in health or mortality, and from health conditions to innovations in wealth. We conclude that there is no causal link from SES to mortality or to incidence of sudden onset health conditions (accidents and, probably, acute conditions), but there is an association of SES with incidence of gradual onset health conditions (mental conditions, and, probably, degenerative and chronic conditions), due either to causal links or to persistent unobserved behavioral or genetic factors that have a common influence on both SES and innovations in health. We conclude that there is no causal link from health status to innovations in wealth.

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1. Introduction

The links between health, wealth, and education have been the subject of study in a number of populations, with the general finding that higher socioeconomic status (SES) is associated with lower levels of morbidity and mortality. There has been considerable discussion of the causal mechanisms that may be responsible for this association. However, most papers in the literature show only that in various populations mortality or an index of health status is significantly correlated with some indicator of SES.[1] Many studies have indexed SES using occupation or education, but where comparisons are possible, the association of health and material well-being appears stronger than the association of health and occupation, at least in the U.S.. In this paper, we will use a vector of eight indicators for wealth, income, education, and neighborhood and dwelling characteristics to capture the features of SES.[2] We will distinguish innovations (events, news) in health or SES from historical state; e.g., a new heart attack is differentiated from cardiovascular history, and an event of dissaving is distinguished from asset history. Doing this, we are able to test for the *absence* of causal links by examining whether innovations are influenced by features of the historical state, once other observed factors are controlled; the test will generally pass *only* if no causal link is present *and* there are no persistent hidden factors that influence both initial state and innovations.

For health conditions and SES, Exhibit 1 depicts possible causal paths, with history built up by applying these transitions period by period over an individual's life. First, low SES may lead to reduced access to medical services, or less effective treatment. This was an important policy issue during the debate in the early 1990's on health insurance. Second, increased morbidity may result from exposure to risk factors that are linked to environmental or occupational hazards associated with low SES. Both these factors could provide a direct causal link from SES history to health events. Third, poor health may reduce the ability to work, lower productivity, and increase medical care expenditures, leading to reduced income and accumulation of assets. This would provide a direct causal link from health to SES events. There may also be observed and hidden *common factors* that lead to ecological

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[1] Backlund, Sorlie, and Johnson, 1999; Bosma et al,, 1997; Chandola, 1998,2000; Drever and Whitehead, 1997; Ecob and Smith (1999; Elo and Preston, 1996; Feinstein, 1992; Fitzpatrick et al, 1997; Fitzpatrick and Dollamore, 1999; Fox and Goldblatt, 1982; Goldblatt, 1990; Hayes, 1991; Hurd, 1987; Hurd and Wise, 1989; Karasek et al, 1988; Kitagawa and Hauser, 1973; Lewis et all, 1998; Marmot et al, 1997; Leigh and Dhir, 1997; Luft, 1978; Martin and Preston, 1994; Martin and Soldo, 1997; Rogers, 1991; Schnall, Landsbergis, and Baker, 1994; Shorrocks, 1975; Stern, 1983; Whitehead, 1988; Woodward et al, 1992.

[2] Specifically, our variables are indicators for the lowest and highest wealth quartiles and the lowest and highest income quartiles (with the intermediate quartiles omitted), indicators for the completion of high school and college, and indicators for self-reported poor or fair neighborhood and dwelling conditions (other conditions omitted). Wealth in our study is measured by accumulating over self-reported holdings in nine asset categories.

association of health and SES. For example, unobserved genetic frailty may influence resistance to disease, and through its impact on tastes and behavior influence ability to work and productivity, influencing SES. Causal links may be reinforced or confounded by behavioral response. Behavioral factors such as childhood nutrition, exercise, and smoking may influence both health and economic activity level, and genetic and socioeconomic factors may influence these behaviors. Tastes for work and for "clean living", whether genetic or learned, may influence both health and earnings. Finally, rational economic decision-making may induce robust consumers to accumulate in order to finance consumption over a long expected retirement, or unhealthy individuals to spend down assets. Preston and Taubman (1994) and Smith and Kington (1997) give detailed discussions of the possible causal mechanisms that may be at work, and the role of behavioral response from economic consumers.

Hurd, McFadden, and Merrill (1999) have studied the causal links between SES and health, using the first two waves of a panel study of older Americans. The third wave of this panel is now available, except for final tracking of panel members who have died, and this report updates our earlier analysis using these new data. Section 2 of this paper reviews the demographic composition of the survey panel and features of the data. Section 3 recaps our earlier investigation of the association of SES and prevalence of health conditions in the initial wave of the panel. Section 4 discusses the problem of econometric testing for causality. Section 5 analyzes incidence of new health conditions, using data from both waves 2 and 3 of the panel, and presents tests for non-causality of SES. Section 6 tests for the absence of a causal link from health conditions to wealth accumulation. Section 7 provides some working conclusions, and describes further speculations and planned research.

2. The AHEAD Panel

Our data come from the Asset and Health Dynamics among the Oldest-Old (AHEAD) study.[3] This is a panel of individuals born in 1923 or earlier, and their spouses. At baseline in 1993 the AHEAD panel contained 8222 individuals representative of the non-institutionalized population, except for over-samples of blacks, Hispanics and Floridians. Of these subjects, 7447 were over age 69; the remainder were younger spouses. There were 6052 households, including individuals living alone or with others, in the sample. The Wave 1 surveys took place between October 1993 and August 1994, with half the total completed interviews finished before December 1993. The Wave 2 surveys took place approximately 25.5 months later, between November 1995 and June 1996, with half the total completed interviews finished by the beginning of February 1996. The Wave 3 surveys took place approximately 25.5 months after that, between January 1998 and December 1998, with half the total completed interviews finished near the beginning of March 1998. In each wave, there was a long but thin tail of late interviews, heavily weighted with subjects who had moved, or required proxy interviews due to death or institutionalization. Subjects never interviewed, directly or by proxy, are excluded from the calculation of the distribution of interview months. AHEAD is a continuing panel, but it has now been absorbed into the larger Health and Retirement Survey (HRS), which is being interviewed on a three-year cycle.

[3] The AHEAD survey is conducted by the University of Michigan Survey Research Center for the National Institute on Aging; see Soldo, Hurd, Rodgers, and Wallace, 1997.

The AHEAD panel has substantial attrition, with death being the primary but not the only cause; see Exhibit 2. A significant effort has been made to track attritors, and identify those who have died through the National Death Register. For Wave 2, these data are released as a tracker file, and for the subjects where a proxy interview was possible, an "exit interview"giving information on cause of death. These files have not yet been released for Wave 3, so there is still some uncertainty regarding mortality and cause of death in this Wave. We formed a working sample of 7082 age-eligible respondents for our analysis by excluding 458 cases with inconsistent household identifier codes or critical missing information. In this working sample, there were additional attritors, and the working sample contains 6565 individuals with complete interview records through all three waves, or through all waves prior to death (or, in Wave 3, presumptive death). In a few cases, attritors in Wave 2 rejoined the sample in Wave 3, but we treat these as permanent attritors because the missing interview makes the observation unusable.

Exhibit 3 compares the actual mortality experience for males between Waves 1 and 2 in the AHEAD sample with the predictions of the 1997 Life Tables of the United States (U.S. Department of Health and Human Services, Center for Health Statistics, 1997).[4] Mortality hazard in AHEAD is somewhat lower than in the life tables, with a larger gap at more advanced ages. This is almost certainly due to selection, since AHEAD recruited only from the non-institutionalized population. Exhibit 4 shows the same curves between Waves 2 and 3. Here, the difference between the curves is much less, reflecting the diminished influence of the initial selection, except at advanced ages. In this range, the AHEAD data is sparse, so the curve is not precisely determined. However, it is possible that the 1997 life table, extrapolated to older ages, overstates the true mortality hazard. Exhibits 5 and 6 repeat this analysis for females, with similar results. The effects of selection are less apparent between Waves 1 and 2 for females under 80, presumably because the rate of institutionalization in this population segment is low, and the effects of selection between Waves 2 and 3 disappears at younger ages.

The AHEAD survey provides data on economics status, health, and family connections. A list of the variables we use, with definitions and summary statistics, is given in Appendix Table A.1. The collection and processing of some of the variables require comment. First, AHEAD individuals and couples were asked for a complete inventory of assets and debts, and about income sources. Subjects are asked first if they have any assets in a specified category, and if so, are asked for the amount. A non-response to the amount is followed by unfolding bracket questions to bound the quantity in question, and this may result in complete or incomplete bracket responses. Through the use of unfolding brackets, full non-response to asset values was reduced to levels usually less than 5 percent, much lower than would be found in a typical household survey. Generally, median responses among full respondents for an asset category are comparable to other economic surveys, such as the Survey of Consumer Finance. However, changes in reported assets between waves show long tails that suggest significant response errors between waves. To reduce the impact of response error, we work

[4]The following steps were used to construct the Exhibit: First, the life table past age 85 is extrapolated by fitting the annual mortality hazard rate as a quadratic function of age for the ages 70 to 85. Second, in the AHEAD data, there are a group of panel members who had (apparently) died at the time contact for a Wave 3 interview was made, so that no interview date was recorded. For these individuals, we impute an attempted contact date by drawing from the empirical distribution of contact dates for those who were interviewed in Wave 3. Finally, we smooth the observed mortality data from AHEAD by estimating, by maximum likelihood, a duration model in which the hazard rate is a quadratic in age.

mostly with indicators for quartile of wealth (aggregated over nine asset categories) and of income. There may also be an issue of bias in responses recovered by unfolding brackets. Hurd *et al* (1997) used experimental variation in the bracket sequences for two financial questions on Wave 2 of AHEAD, and found that anchoring to the bracket quantities was significant.

AHEAD has an extensive battery of questions about health conditions, including mental health. The study collects data on self-assessed health status, where the subject is asked to rate his or her health as excellent, very good, good, fair or poor. No reference is made to other groups such as "people of your age." Self-assessed health has been found to be highly predictive of mortality in the HRS (Hurd and McGarry, 1997). Expectations about longevity are elicited from AHEAD subjects by asking for the probability that they will live to a specified age, defined in the questionnaire to be 10 to 15 years beyond their current age. We use low responses to this question as another indicator of perceived health problems. The study also collects information on the number of ADL (activities of daily living) limitations and IADL (instrumental activities of daily living) limitations. A high ADL limitation count indicates that the individual has difficulty with personal self-care, while a high IADL limitation count indicates difficulty in household management. The study contains the CESD battery of questions measuring general mood; and from this we form an indicator for depression. The study measures cognition using in a battery of questions which aim to test a number of domains (Herzog and Wallace, 1997): learning and memory are assessed by immediate and delayed recall from a list of 10 words that were read to the subject; reasoning, orientation and attention are assessed from Serial 7's, counting backwards by 1 and the naming of public Exhibits, dates and objects.[5] This score reflects both long-term ability and impairments due to health events. To isolate the latter, we take education to be an indicator of long-term ability, and construct a scale that removes this component. In the AHEAD population, average education levels are higher in the younger cohorts due to changing education policy in the first decades of this century. As a first step, we regress years of education on age, by sex, and then for each subject predict an age-adjusted education level. The second step is to regress the cognitive score on age adjusted education, and use the residuals from this regression to produce a scale for variation in cognitive performance from long-term level. The final step is to construct an indicator of cognitive impairment that picks out the bottom 25 percent of the baseline sample in terms of this scale.

The AHEAD survey asked about a number of specific health conditions, usually in the form of "Has a doctor ever told you that you had….". We use data on the health conditions listed in Exhibit 7. We include death as a health condition because cause of death is unknown in Wave 3, and will be treated as a joint hazard. The second column classifies diseases into five categories defined roughly by intensity and duration of treatment. If there are causal links from SES to changes in health, they may divide along these lines. The final column indicates whether data was collected on new incidences of the same condition. We note that there are some major groups of health conditions that were not investigated in AHEAD: degenerative neurological diseases, kidney and liver diseases, immunological disorders, and accidents other than falls.

Missing data is an issue in the AHEAD panel, particularly for assets where total wealth accumulates over multiple asset categories. To avoid severe attrition due to item non-response, we use a nested hot-deck imputation procedure that conditions on asset information in the current and the last

[5] Serial 7's asks the subject to subtract 7 from 100, and then to continue subtracting from each successive difference for a total of five subtractions.

wave of data. First, missing ownership in a wave is imputed by choosing randomly from respondents, conditional on ownership in the other wave. For persons missing ownership in both waves, an ownership pair is drawn from complete respondents in both waves. Then, an amount or change in asset value is imputed to a complete or incomplete bracket response by drawing from continuous responses, conditioning on the bracket range in both waves. Total assets are then accumulated across categories following the imputation process.

Exhibit 8 shows the age gradients of wealth, income, and education in the AHEAD sample. These gradients reflect substantial cohort effects, as well as life-cycle and composition effects. Work, income, and asset accumulation patterns of the AHEAD population were impacted by World War II, and those over age 80 experienced the Great Depression during their prime working years. The U.S. was substantially rural when the AHEAD population was born, and education was truncated for work for many members of this population. In addition to cohort effects, the curve for assets reflects life-cycle disaccumulation of assets through the retirement years, and the curve for income reflects the rising proportion of widows in the survivors to older ages. There is an additional compositional effect from the association of SES and mortality: higher SES is selected preferentially among survivors.

3. SES and Prevalence of Health Conditions

We first give some descriptive statistics on the prevalence of health conditions in the AHEAD population. Exhibits 9-13 gives prevalence rates in the baseline wave 1 for five health condition groups, classified by age and sex. Generally, prevalence of health conditions does not show a strong age gradient, indicating broadly that morbidity rates among survivors do not increase much with age. The major exception is in the group of mental conditions, where cognitive impairment is increasingly common as age increases. The prevalence of acute and degenerative conditions among survivors fall after about age 80, reflecting the effect of selection due to deaths from these conditions. Males have higher prevalence of acute and degenerative diseases than do females, but females have higher prevalence of mental and chronic conditions, and accidents.

The wealth gradients for prevalence in the five health condition groups are shown in Exhibit 14. Note that these gradients reflect first-order association, uncorrected for initial health status and for composition effects. Each group shows a decline in prevalence with increasing wealth, with the sharpest declines for degenerative and mental conditions, and little relative decline for chronic conditions and accidents. To provide a clearer picture of the association of SES and health conditions, we estimate binomial logit models for the presence of a health condition in each group as a function of demographic variables (a linear spline in age, interacted with sex, marital status, race, ages of parent's deaths[6], Body Mass Index (BMI), an indicator for ever smoked, and SES variables (wealth and income quartiles, education, and indicators for condition of neighborhood and condition of residence). Appendix Table A.2 describes the specific variable transformations used in these models. We carry out likelihood ratio tests of the hypothesis that the eight SES variables can be excluded as factors associated with prevalence. To give a sense of the SES gradient, we calculate the relative odds of prevalence for a low SES individual (bottom quartile for wealth and income, less than a high school

[6]If a parent is still living, then we impute an age of death from life expectancy given sex and age, from standard life tables (extrapolated to older ages).

education, and a neighborhood and residence in poor/fair condition) versus a high SES individual (top quartile for wealth and income, a college education, and a neighborhood and residence in good condition), averaging over the sample empirical distribution of the demographic variables. The results are given in Exhibit 15. Every group of health conditions shows a strong association with SES, controlling for demographics. The relative odds are largest for degenerative and mental conditions, and weakest for acute and chronic conditions. We find that there is a statistically significant association of SES with each of the individual health conditions in Exhibit 7, with the exception of hip fractures. These results confirm that the observed association of SES and health cannot be explained away by demographic composition effects, but of course they provide no evidence on causal structure.

4. Association and Causality in Panel Data

The primary purpose of this study is to test for direct causal links between SES and health. The nature of causality and the interpretation of "causality tests" have been long debated among econometricians and philosophers of science. Insightful discussions of causality and measurement can be found in the papers of Granger (1969), Sims (1972), Zellner (1979), Schwert (1979), Engle, Hendry, and Richard (1983), Geweke (1984), Sobel (1997), Pearl (1997), and Hendry and Mizon (1999). We will not add to this discussion, but will try to be precise about our definitions and goals. Let X_t, Z_t, and Y_t denote vectors of random variables at date t, and interpret a realization of these vectors as an observation on a subject in one wave of a panel survey. Let $\boldsymbol{H}_t = (\boldsymbol{X}_t, \boldsymbol{Y}_t, \boldsymbol{Z}_t)$ be the information sets containing histories of these respective vectors through time t. Let $f(Y_t|\boldsymbol{H}_{t-1})$ denote a *model* of the conditional distribution of Y_t given $\boldsymbol{H}_{t-1}$, and term it a *valid* model for a given history if it correctly characterizes the true distribution of Y_t for this history. We say that f is a *structural* or *causal* model, or (*probabilistic*) *law,* for Y_t relative to a specified class of histories if it has the *invariance* property that it is valid for each history in this class. Operationally, this means that within specified domains, f has the *transferrability* property that it is valid in different populations where the marginal distribution of $\boldsymbol{H}_{t-1}$ changes, and the *predictability* or *invariance under treatments* property that f remains valid following policy interventions that alter $\boldsymbol{H}_t$. We say that X is *conditionally causal* for Y, given Z, if a valid model has $f(Y_t|\boldsymbol{X}_{t-1},\boldsymbol{Y}_{t-1},\boldsymbol{Z}_{t-1}) \neq f(Y_t|\boldsymbol{Y}_{t-1},\boldsymbol{Z}_{t-1})$; i.e., given $\boldsymbol{Y}_{t-1}$ and $\boldsymbol{Z}_{t-1}$, knowledge of $\boldsymbol{X}_{t-1}$ is *necessary* to achieve the invariance properties of a causal model, and knowledge of $\boldsymbol{X}_{t-1}$ *contributes to the predictability* of Y_t. Conversely, we say that X is conditionally *non-causal* for Y if $f(Y_t|\boldsymbol{Y}_{t-1},\boldsymbol{Z}_{t-1})$ is a valid model and Y_t is conditionally independent of $\boldsymbol{X}_{t-1}$. We say that Z is *exogenous* (relative to X,Y) if X and Y are conditionally non-causal for Z.

These definitions have two key properties. First, they are positivistic constructs in the spirit of the purely statistical treatment of "causality" by Granger (1969), and conditional non-causality is simply Granger's test for the absence of causality, augmented with an invariance condition. They meet the limited objective of providing a foundation for policy analysis, where it is the invariance property under policy interventions that is crucial to predicting policy consequences. Thus for example, suppose our analysis using this framework concludes that SES is not conditionally causal for new health events within the domain where the Medicare system finances and delivers health care. This finding would then support the conclusion that policy interventions in the Medicare system to increase access or reduce out-of-pocket medical expenses will not alter the conditional probabilities of new

health events, given the health histories of enrollees in this system. It is unnecessary for this policy purpose to answer the question of whether the analysis has uncovered causal structure in any deeper sense. However, our emphasis on invariance properties of the model, and on tests for Granger causality within invariant families, does go part way toward consistency with the view of philosophers of science that causality is embedded in "laws".

Several additional comments are needed on these definitions. First, the definition of a causal model $f(Y_t|\boldsymbol{H}_{t-1})$ does not include conditioning on contemporaneous variables. By definition this rules out what Granger calls "instantaneous causality". Our formulation is consistent with Wold's causal chain view that causal action takes time, and "instantaneous causality" is an artifact of time aggregation. In applications where time aggregation is an issue, one can treat observed variables as indicators for a latent causal chain structure. Second, our definition of a causal model is relative to a specified class of conditioning variables and domains for transferability and policy interventions. A model that is valid in one domain may fail in a broader domain. Short of controlled experiments with randomized treatments, it is impossible to definitively rule out ecological common factors as a source of what appears to be conditional causality. However, a finding of non-causality is unlikely if indeed strong direct causal effects are present. Third, by including temporal or spatial variables in Z, it is possible to weaken invariance requirements to fit almost any application. Done indiscriminately, this creates a substantial risk of producing an "over-fitted" model that is not genuinely causal. On the other hand, it may be necessary to model "regime shifts" to account for factors that are causal for some populations or time periods, and not for others. Fourth, in practical models, it is necessary to truncate or summarize history, so that the span of $\boldsymbol{H}_t$ is finite. Then, a conditional distribution $f(Y_t|\boldsymbol{H}_{t-1})$ can be written as a first-order Markov process $f(Y_t|X_{t-1},Y_{t-1},Z_{t-1})$ through the device of re-dating older observations. In some applications, postulated direct causal links are relatively short-acting, while common factors have longer-term effects. For example, if SES influences the choice of therapy for a disease, then its impact should be felt within the course of the days or months over which the disease plays itself out. On the other hand, genetic susceptibility or childhood nutrition may be common factors in SES and health that act over decades. The ability of a model $f(Y_t|X_{t-1},Y_{t-1},Z_{t-1})$ to be causal when common factors are operating will depend on the ability of components of Y_{t-1} to approximate the effect of these factors. To illustrate, if a common factor partitions a population into "movers" and "stayers" for whom the first-order transition probabilities from Y_{t-1} to Y_t differ, including the number of past moves in Y_{t-1} may be enough to give f the required invariance properties for a causal model. Fifth, the definitions above can apply to any economic time series, but panel data provide the "natural experiments" needed to sort out functional forms and identify conditional distributions that are plausibly valid causal models.

Now consider in more detail the issues that arise in implementing causality tests for innovations in SES and in health. One problem is timing of observations. There are roughly two years between each of the first three waves in the AHEAD panel, and the intervals between interviews can vary considerably across subjects. There may be multiple health events between waves, including multiple episodes of the same condition, but the data do not provide an event history that gives the timing and sequencing of events. As a consequence, there is some time aggregation that could confound causal links; e.g., if wealth is causal to health condition A, which in turn is causal to health condition B, and health condition B is causal to changes in wealth, then in a time aggregate wealth will appear causal to condition B and condition A and wealth innovations may appear to be mutually "instantaneously

causal". The inability to precisely date events such as death within the interval between waves also complicates the modeling of transitions.

The joint and competing structure of health risks, and the coding of health events, may also confound causality tests. Consider, for example, heart disease. AHEAD codes whether the subject has ever had heart disease, and whether there has been a new incident since the last interview. The questions do not define precisely what constitutes a new incidence for a subject with a previous history of heart disease. A new heart attack is presumably reported as a new incidence, but it is unclear whether subjects interpret procedures such as angioplasty or bypass surgery as new incidents or as treatments for previous conditions. If a subject dies, cause of death is asked in the "exit interview" of a proxy, but the coverage is incomplete, and in Wave 3 is not yet available. Then, death is a "competing risk" for heart disease, as well as for other acute diseases such as cancer and stroke. Some risks are joint. For example, health conditions such as cancer or arthritis are likely to induce depression, and diabetes leads to heart disease. If there is an SES gradient for some conditions, then competing risks may create a spurious SES gradient for other conditions. For example, suppose a low SES population has a 20 percent hazard rate and a high SES population has a 10 percent hazard rate for death from heart disease, while both groups have a 10 percent hazard rate for cancer. Then, 8 percent of the low SES group and 9 percent of the high SES group survive and are diagnosed with cancer. In addition, for some diseases such as cancer, detection is a factor which may have an SES gradient, so that detected incidence may rise with SES even if true incidence has no gradient. To reduce competing risk effects, we concentrate on the five broad health categories listed in Figure 7: acute (mortality, heart disease, cancer, stroke, diabetes), degenerative (lung disease, diabetes), chronic (high blood pressure, arthritis, incontinence), mental (depression, psychiatric, cognitive impairment), and accidents (falls, hip fractures).and chronic conditions (high blood pressure, psychiatric disease, arthritis, falls requiring treatment, depression, hip fractures, incontinence, cognitive impairment).[7]

Definition and measurement of some variables is also a concern. For example, current smoking, Body Mass Index (BMI), ADL, and IADL status are predictive for future health problems, but may also be altered as a result of health conditions already present. The AHEAD data does not reliably report cause of death in Wave 2, and this information is not yet available for Wave 3. In addition, there may be response error in some health measures, due to faulty recall or time telescoping, and to confusion between conditions and treatments. Detection problems may cause actual incidence to be under-reported. This may be a particular issue for cancers, where quality of medical care may affect the timeliness of detection, and for strokes, where some minor strokes may go untreated. Measurement error could induce spurious association of health and wealth if healthy individuals were more optimistic, and this extended to overstatement of assets or understatement of health problems, or if unhealthy individuals were more likely to forget about assets.

If there is an SES gradient for some conditions, then competing risks may create a spurious SES gradient for other conditions. For example, suppose a low SES population has a 20 percent hazard rate and a high SES population has a 10 percent hazard rate for death from heart disease, while both groups have a 10 percent hazard rate for cancer. Then, 8 percent of the low SES group and 9 percent of the high SES group survive and are diagnosed with cancer. In addition, if there is an SES gradient for detection of health conditions caused by the variation in availability or utilization of medical services,

[7] Our categories of acute and degenerative diseases include five of the six leading causes of death for elderly Americans in 1996 (National Center for Health Statistics).

this may cause diseases such as cancer to have an apparent SES gradient even if true incidence has no gradient, and these apparent gradients may show *increasing* incidence with SES.

5. Incidence of Health Conditions and Tests for Causality in the AHEAD Panel

We will use the incidence of new health problems (or recurrence of acute problems), conditioned on initial demographic, health, and SES status in the baseline Wave 1, to test for the absence of causality. We define incidence for a group of health conditions to be the occurrence of a condition from the group that was not previously reported, or a recorded reoccurrence in the case of an acute condition (cancer, heart disease, stroke). Due to the difficulty of identifying cause of death, and the consequence that death may appear in the panel as a "competing risk" for incidences of acute diseases, we include mortality as an acute condition. Then, an incidence of an acute condition in the following analysis would be a non-fatal occurrence or reoccurrence of cancer, heart disease, or stroke, or death from any cause. Exhibits 16-21 are graphs of annual rates of incidence by age for males and females, for mortality and for each of our five health condition groups. These graphs are not corrected for initial health status or other composition effects, and consequently reflect some combination of true age dependence for the hazard, selection due to competing risks and to prior occurrence of continuing conditions, and demographic factors. Noting that the rates are imprecise at advanced ages due to relatively small numbers of observations, only mortality, incidence of acute conditions which are dominated by mortality, and incidence of mental conditions show systematically increasing hazard rates with age. Except for mortality, there are not strong systematic differences in incidence rates between males and females. In part, this is due to the selection effect of higher male mortality. Exhibit 22 shows the crude wealth gradients for the health condition groups, again not adjusted for age or composition effects. In terms of relative odds of incidence, mortality and incidence of acute conditions show the strongest wealth gradient.

We have carried out two econometric exercises to control for demographic and health status, and perform tests for causality. The first simply pools the intervals between Waves 1 and 3, and examines the incidence of health conditions within the (median) 51 month interval between these waves. We estimate a binomial logit model for incidence in each group of health conditions as a function of baseline SES, demographic, and health status, and conduct likelihood ratio tests for the influence of the SES variables on incidence. We do not take account of variation across subjects in "time at risk" due to differences in the inter-wave interval. For most subjects, the interval between Waves 1 and 3 was within a month or two of the 51 month sample median. For a small number of subjects, the interval was substantially longer due to difficulty in completing Wave 3 interviews. These events tended to be related to events such as death or institutionalization, so that inter-wave intervals are endogenous, and are related to incidence of health conditions.

Exhibit 23 summarizes the results of likelihood ratio tests for significance of the eight SES variables, given baseline demographic and health status. The null hypothesis is that there is no causal link from these variables to innovations in health conditions. Relative odds ratios between low SES and high SES subjects are given for each group of health conditions as an aid to judging their economic importance. These tests accept the hypothesis of no causal link to SES for degenerative conditions, accidents, and chronic conditions. We consider the tests for mortality, and acute and mental conditions to be inconclusive, given the sample size. The relative odds between low and high

SES are substantial for mortality and for degenerative diseases, but not for the remainder. The exhibit also summarizes the variables that are individually significant (at the five percent level), as indicated by their T-Statistics. Examining the individual health conditions within the groups, the null hypothesis is rejected for lung disease and for all the mental conditions (psychiatric disease, depression, and cognitive impairment), and accepted for the remaining conditions.

A drawback of the econometric analysis just described is that it does not permit tests of the invariance properties of the models. We have argued in Section 4 that this is an essential part of a convincing test for the absence of causality. As a partial remedy, we have conducted a second econometric analysis in which the data on incidence from Wave 1 to Wave 2, given state in Wave 1 and the data on incidence from Wave 2 to Wave 3, given state in Wave 2, are stacked. One test for the invariance property that the model should have if it is to provide a satisfactory test for non-causality is that it should be the same for the Wave 1 to 2 transitions and for the Wave 2 to 3 transitions, except for adjustments for "time at risk" between the waves. We conduct Chow tests for the null hypothesis that the models are invariant across waves. Conditional on acceptance of the Chow test, we test the null hypothesis of no causality. We also report the significance of joint tests of invariance and non-causality. Of course, our single test for invariance is considerably short of the battery of tests that one would like a model to pass to be fully credentialed as a valid causal model.

The results are given in Exhibit 24. All the groups of health conditions accept the hypothesis of invariance except acute conditions. Conditioned on acceptance of invariance, the hypothesis of non-causality is accepted for mortality and accidents. The conditional tests for degenerative and chronic conditions are inconclusive. The joint test of invariance and non-causality is accepted for chronic conditions, but remains inconclusive for degenerative conditions. The relative odds for low versus high SES are substantial for degenerative and mental conditions, and modest for the remainder.

We conclude from this analysis that if there is a causal link from SES to mortality or to accidents, then it is sufficiently weak to escape our detection. Within the framework of causality testing that we have set out, we have failed to establish an invariant model for acute conditions that is prerequisite to a non-causality test. Had the invariance property been accepted, the conditional test would have accepted the no-causality hypothesis. However, we cannot rule out the possibility that this is an artifact of non-invariance. Examining the individual diseases within the acute group, we find that the invariance failure is attributable to heart disease. The most likely source of such a failure is an insufficiently detailed description of cardiovascular state, so that chronic and life-threatening conditions are intermingled, with a mix that is changing between waves, or a failure to account for interactions between cardiovascular disease and other health conditions. However, we cannot rule out the possibility that the definition of a heart condition or of recurrence has changed between waves, at least in terms of the subject's understanding, or that there is substantial selection in the sample caused by death due to heart attacks or competing hazards.

For degenerative diseases, the relative odds of 2.29 for low versus high SES suggests that SES is important, even though the statistical significance test is inconclusive, rejecting non-causality at the five percent, but not at the one percent, significance level. When the analysis is done separately for lung disease, low income and poor/fair dwelling condition are found to be statistically significant contributors to incidence, suggesting some combination of a causal link from living conditions to lung disease and a common influence on SES and lung disease of unobserved common behavioral and environmental factors (e.g., exposure to airborne hazards in low wage occupations).

For mental conditions, there appears to be an association with SES attributable either to a causal link from SES to mental conditions, perhaps because access to mental health services is the least uniformly delivered service provided to the elderly under medicare, or to common genetic or behavioral factors that influence both SES and mental health. We find that each of the individual conditions in the mental group, psychiatric disease, depression, and cognitive impairment, passes the invariance test and fails the no-causality test, and that the relative odds for low versus high SES are large for psychiatric disease (3.36) and for depression (2.64). Individual logit models for both psychiatric disease and depression show significant effects from low wealth and low income. It is plausible that these factors, and the strain they place on living satisfactorily, are directly causal. It is also possible that undetected psychiatric problems that lower productivity during working life surface later as the result of other health problems.

6. Tests for Causality from Health Status to Asset Accumulation

Health may influence asset accumulation of elderly households because of the cost of medical treatment and related services, such as home health care and drug purchases. Also, health conditions may limit the consumption of other goods, and because health status is an indicator of longevity, an individual planning consumption and precautionary reserves over remaining life may adjust target wealth based on altered perceptions of longevity; see Alessie, Lusardi, and Kapteyn (2000), Attanasio and Hoynes (1995), Hurd (1987), Hurd and Wise (1989), Hurd, McFadden, and Gan (1998). These effects could induce a positive association of SES and health status even if there were no causal links from SES to health. We analyze couples and singles separately, and exclude observations if either the subject or the spouse if present, die between the waves. Then, our tests will be for invariance and for non-causality of health state, conditioned on survival of all household members between the waves. While we are not testing whether mortality causes wealth to change, and measurement problems associated with a change in financially responsible respondent make it difficult to determine precisely the impact of death, it is obvious that terminal medical expenses, burial expenses, estate taxes, and other estate settlement costs, and insurance payments, will have a substantial impact on the size of the decedent's estate or surviving spouse's assets. We measure wealth changes for households between waves as changes in the *percentile* the person occupies in the wealth distribution. We do OLS regressions of the change in the percentile position on initial wealth percentile, income quartile, demographic variables, and health conditions in the starting wave, with the data stacked for the Wave 1 to 2 and the Wave 2 to 3 transitions. We then test for invariance of the regression model between the waves, and test for the significance of initial health conditions. The results are summarized in Exhibit 25. For both couples and singles, the invariance test and the non-causality test conditioned on invariance pass, indicating that there is no detectable causal link from health conditions to wealth innovations in this population. This finding is consistent with the high coverage rate for medical expenses by Medicare and Medicaid in the post-65 population. However, the explanatory power of these regressions for wealth changes is low, so that the tests probably do not have sufficient power to detect causal links that may be present but are small in terms of dollar impacts of health costs on savings.

7. Summary and Speculations for Further Research

This paper has used innovations in health conditions and in wealth in the AHEAD panel to carry out tests for causality from SES to health, and from health conditions to wealth. By advancing beyond the detection of association to a framework in which there is some possibility of detecting the absence of causal links, this paper is an advance on much of the literature on this subject. We conclude that for mortality and for accidents, the hypothesis of *no* causal link from SES is accepted, and for incidence of mental problems the hypothesis is rejected. The results for chronic and degenerative diseases are not definitive, but using the preferred test procedure summarized in Exhibit 24, the hypothesis is marginally rejected in both cases. The hypothesis appears to be accepted for acute conditions, but the necessary invariance property fails, so we cannot rule out the possibility that this is an artifact. The pattern of results suggests that incidence of acute, sudden onset health conditions does not exhibit a significant SES gradient, while incidence of chronic, mental, and degenerative conditions appears to have an association to SES due to some combination of direct causal links and common unobserved behavioral or genetic factors. Specifically, there may be an SES gradient in seeking treatment for the second class of conditions, which may influence detection, or for maintaining preventative regimens that may maintain some conditions below the reporting thresholds. Our findings are not inconsistent with the possibility that for mental and chronic illnesses where the acute care procedures covered by Medicare are often inapplicable, ability to pay may be a causal factor in seeking and receiving treatment.

Future waves of the AHEAD (HRS) panel will allow the hypotheses of invariance and non-causality to be tested with greater power. This will particularly be the case when full tracking of decedents, and determination of cause of death from medical records, become part of the data. However, suitably defined natural or designed experiments, the nature of which is not yet in sight for this population, will probably be necessary in the end to establish definitively the nature of the causal links between SES and health.

The modeling structure used in this paper is parametric, and the high dimensionality of the vector of possible explanatory variables and the relatively limited information contained in binomial outcomes in the AHEAD panel make it difficult to move to a more robust non-parametric analysis. However, we have been flexible in specifying the variable transformations that appear in our models, and we interpret our analysis as conforming in spirit, if hardly in fact, to a method of sieves approach to non-parametric analysis. One of the major limitations of our models, which would be likely to lead them to fail invariance tests in situations where a sharp test is possible, is that they do not account adequately for the multiple risk structure of health conditions and its implications for the duration patterns that can emerge. Some outcomes, such as mortality and non-fatal heart conditions, are competing risks, while others, like diabetes and heart conditions, are complementary risks. For future research, we are investigating models in which a latent vector of propensities for all health and SES conditions follows a first-order Markov process, conditioned on demographic state, and all possible causal links across the components of this latent vector appear in the model. Given thresholds that trigger observed states, this model provides a consistent but computationally demanding data generation process for the vector of observed Markov states. Within this model, it is possible to carry out joint tests for the absence of classes of causal links. However, we have been unable to implement this model fully, and partial implementations we have done have not produced results amenable to

interpretation. The next wave of this research, incorporating Wave 4 of AHEAD, will include full development of flexible multiple-risk duration models.

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Ex. 1. Causal Paths for SES and Health

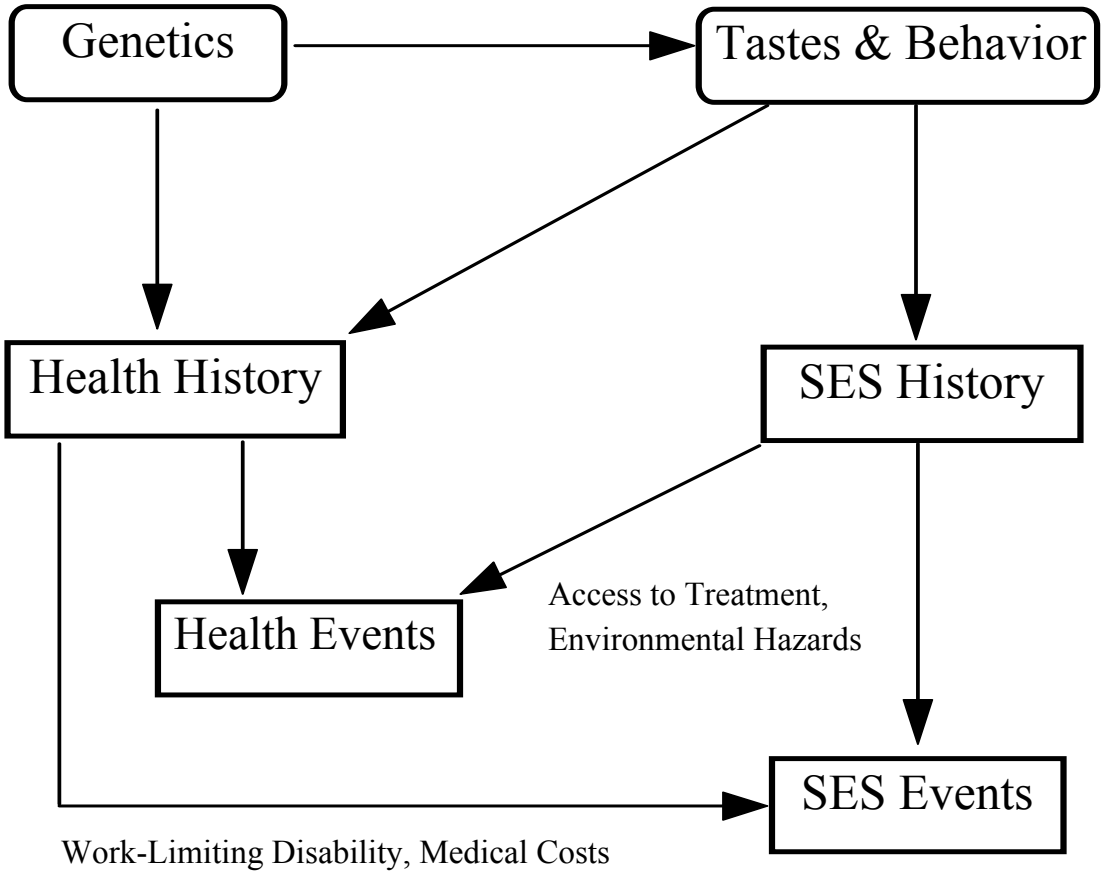
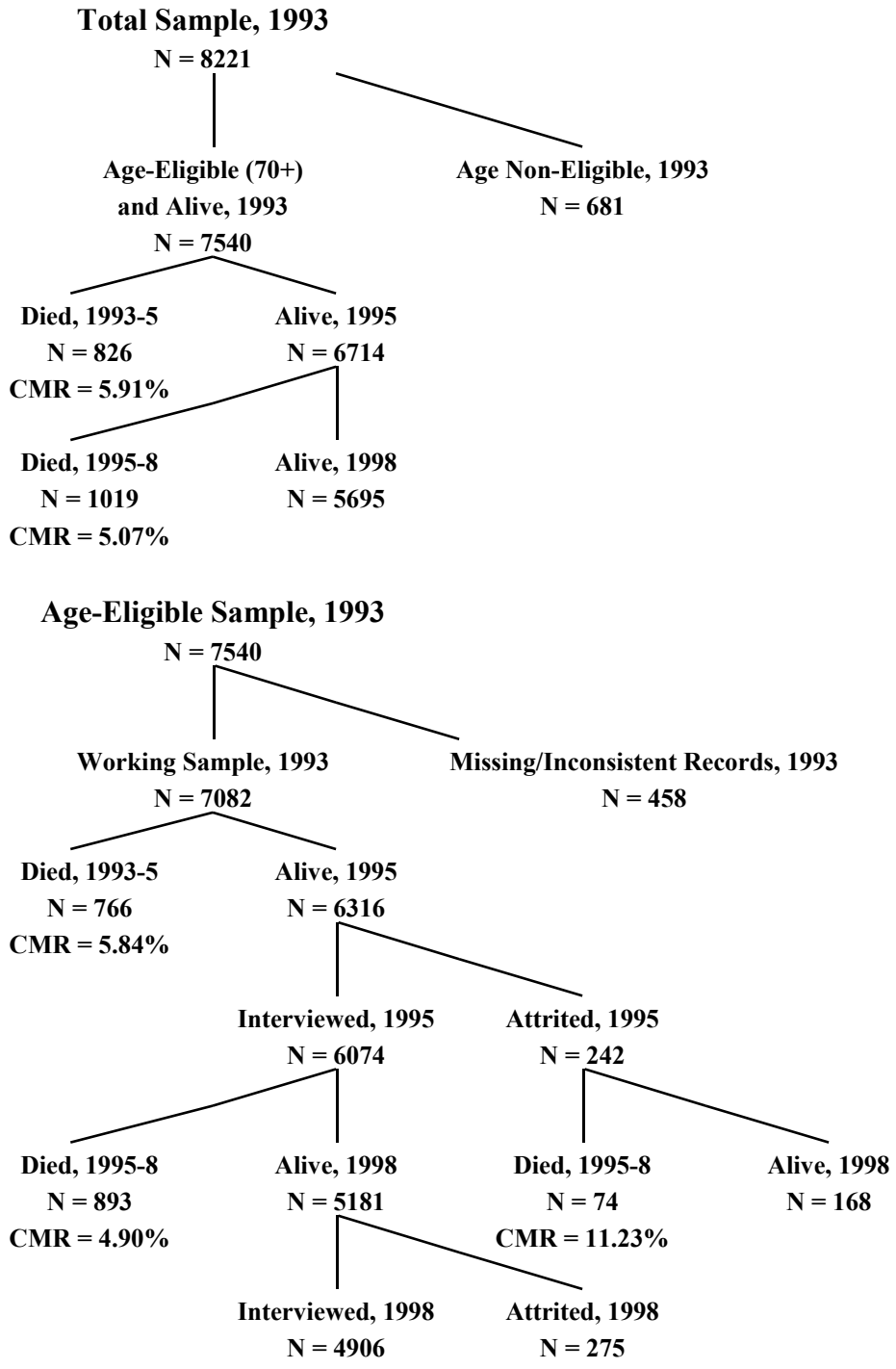
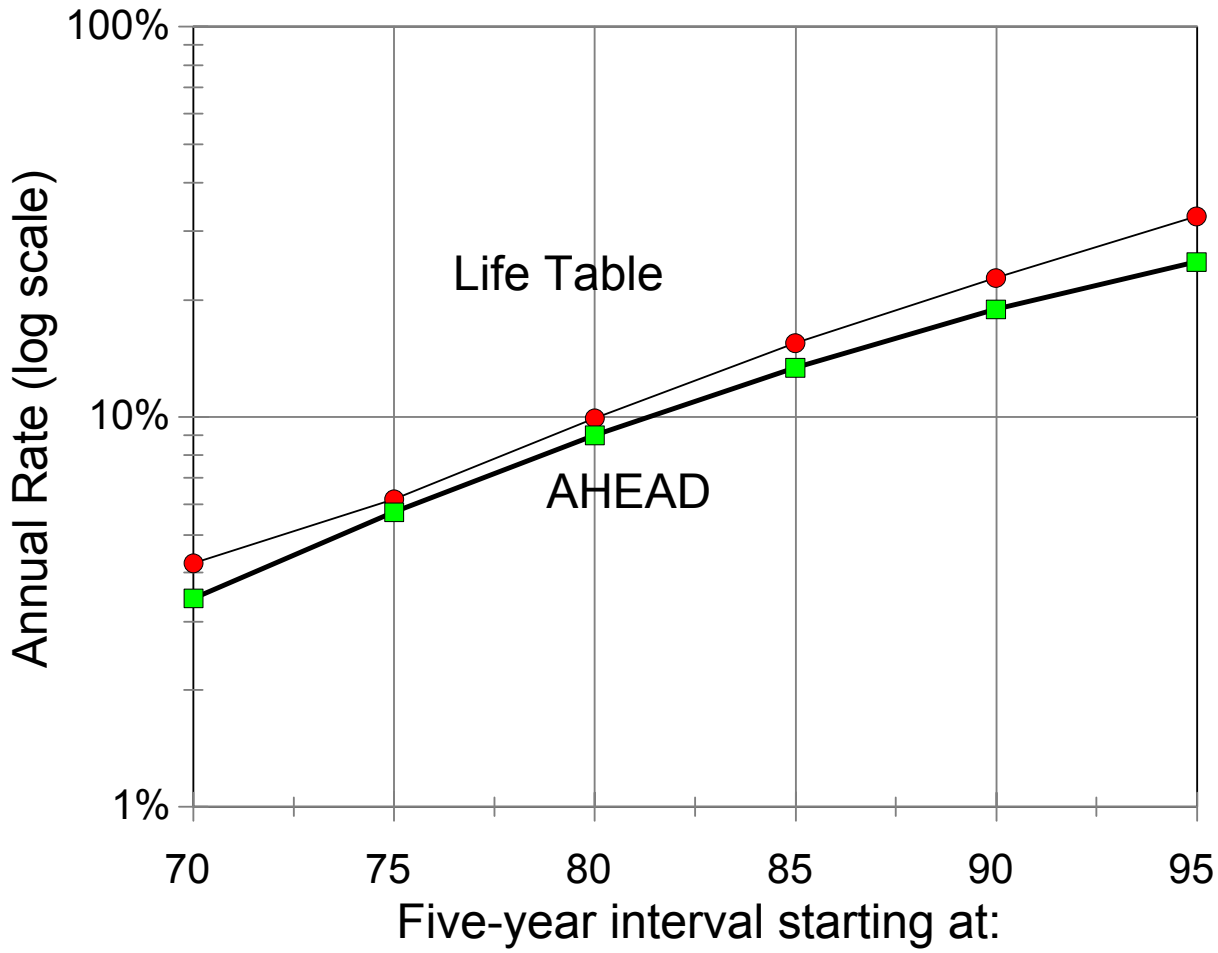


Exhibit 2. AHEAD Sample Characteristics



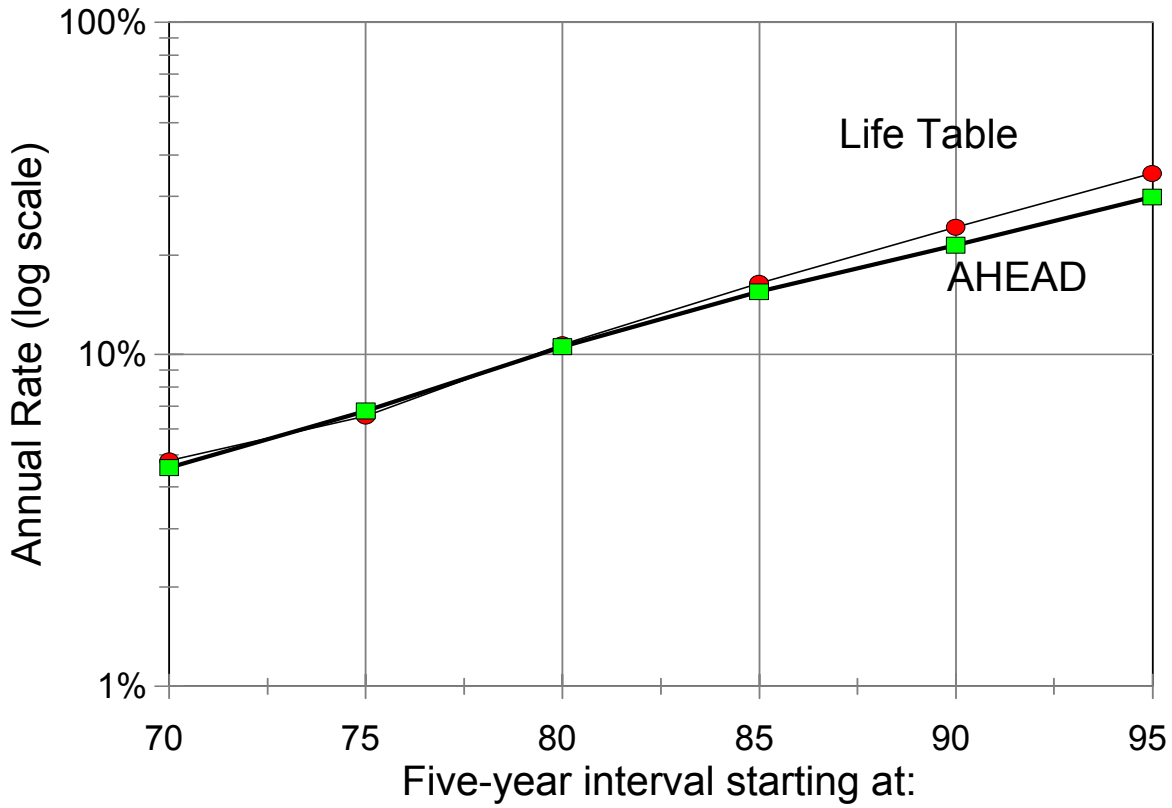
Notes: N = sample count, CMR = crude mortality rate, annualized. The 1995-8 CMR for the sample alive in 1995 is 5.11%.

Ex. 3. Mortality Hazard
Males, Wave 1 to Wave 2



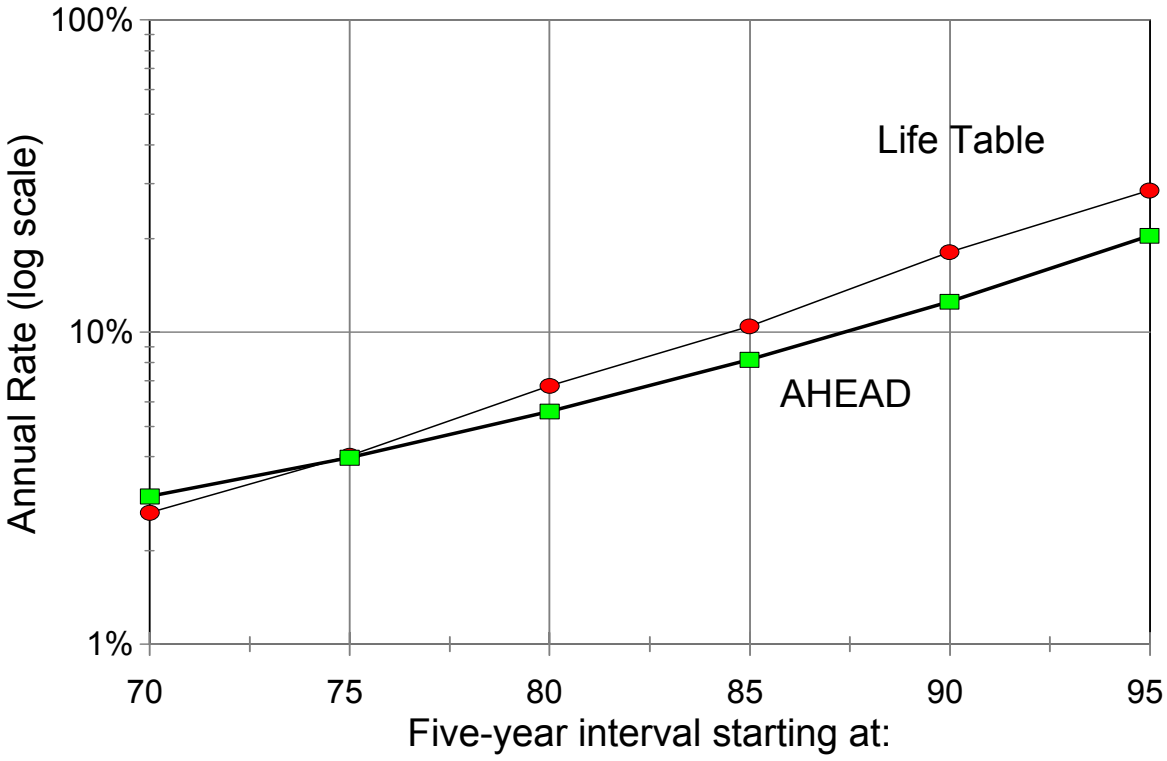
Ex. 4. Mortality Hazard

Males, Wave 2 to Wave 3



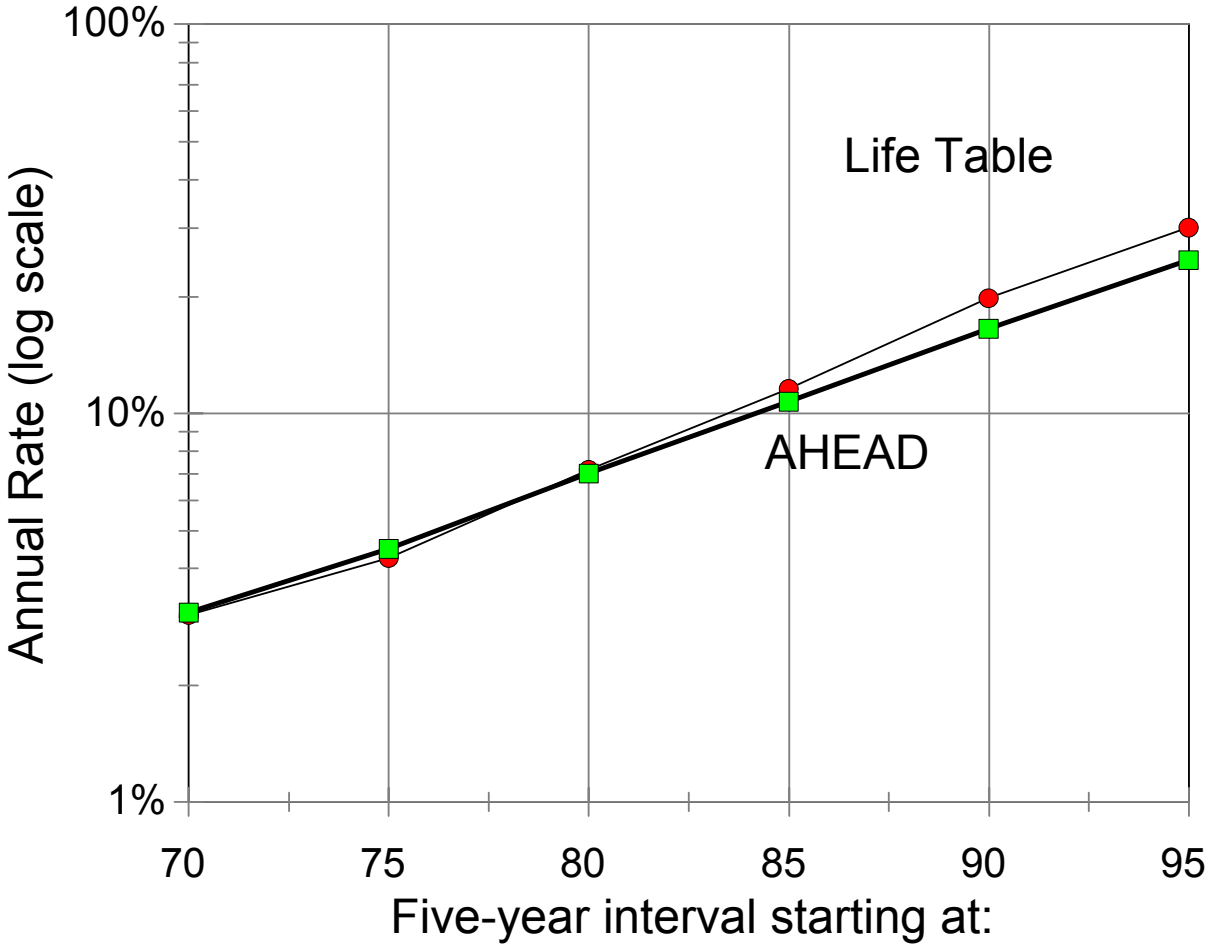
Ex. 5. Mortality Hazard

Females, Wave 1 to Wave 2



Ex. 6. Mortality Hazard

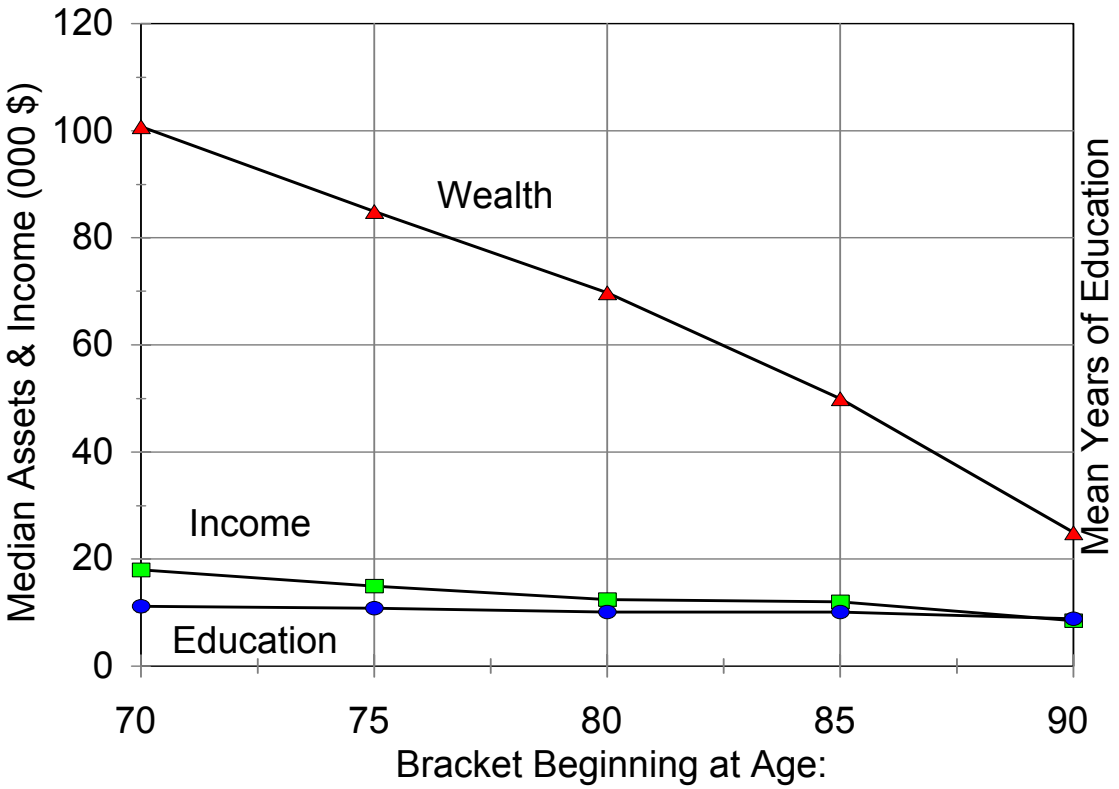
Females, Wave 2 to Wave 3



Ex. 7. Specific Health Conditions Recorded in the AHEAD Panel

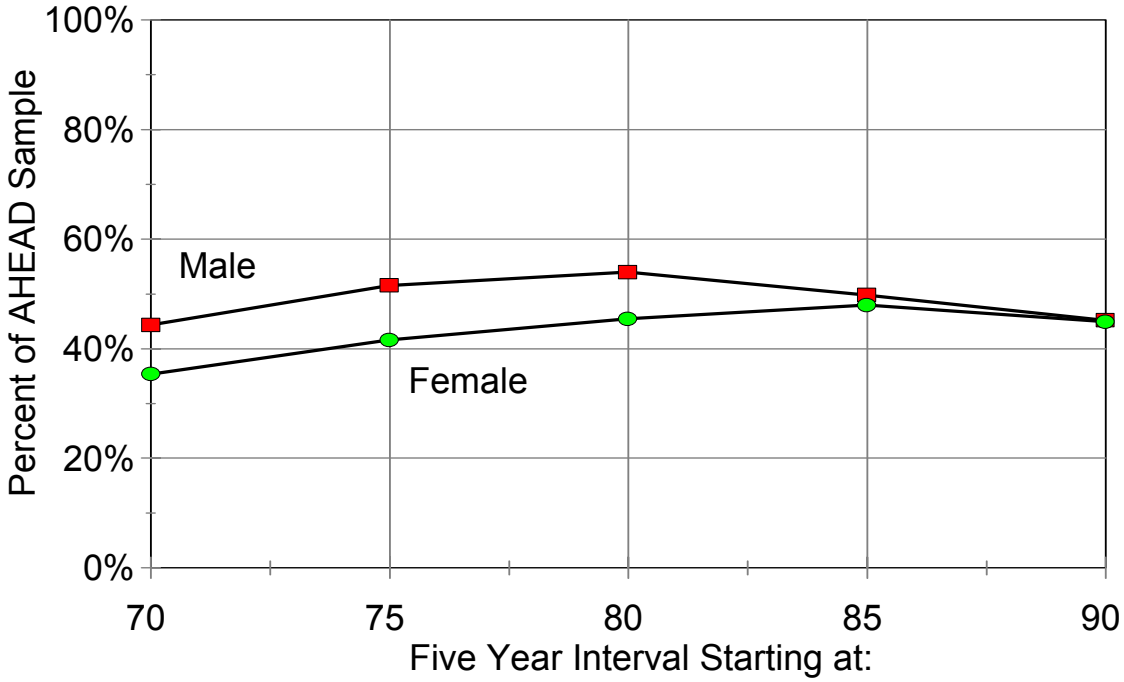
Health Condition	Group	Recurrence Recorded	Prevalence Rates by Age				
			70-74	75-79	80-84	85-89	90+
Mortality	Acute						
Cancer	Acute	Yes	12.09%	14.82%	15.65%	13.83%	11.93%
Heart Disease	Acute	Yes	28.04%	31.62%	35.43%	34.93%	11.93%
Stroke	Acute	Yes	6.57%	9.44%	10.70%	11.35%	13.33%
Lung Disease	Degenerative	No	11.97%	12.08%	11.99%	8.01%	4.56%
Diabetes	Degenerative	No	15.43%	13.81%	12.53%	9.90%	5.61%
High Blood Pressure	Chronic	No	49.32%	52.08%	50.95%	48.03%	41.05%
Arthritis	Chronic	No	22.64%	29.09%	27.91%	29.11%	28.07%
Incontinence	Chronic	No	15.69%	19.49%	21.54%	25.91%	30.53%
Depression	Mental	No	8.33%	10.20%	11.72%	11.06%	11.58%
Psychiatric Disease	Mental	No	12.35%	12.03%	9.42%	7.42%	4.91%
Cognitive Impairment	Mental	No	14.46%	19.97%	30.89%	43.09%	53.38%
Fall Requiring Treatment	Accident	No	6.04%	7.61%	8.67%	10.92%	14.39%
Hip Fracture	Accident	No	3.04%	4.16%	5.89%	10.77%	12.63%

Exhibit 8. SES by Age in AHEAD



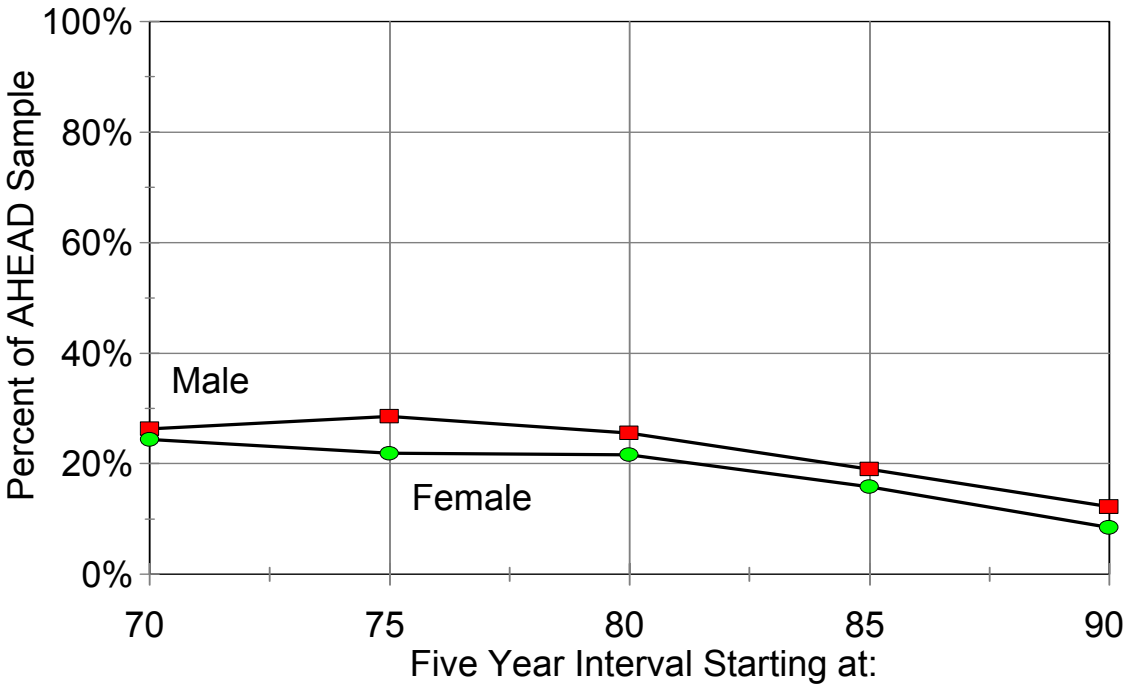
Ex. 9. Acute Health Conditions

Prevalence



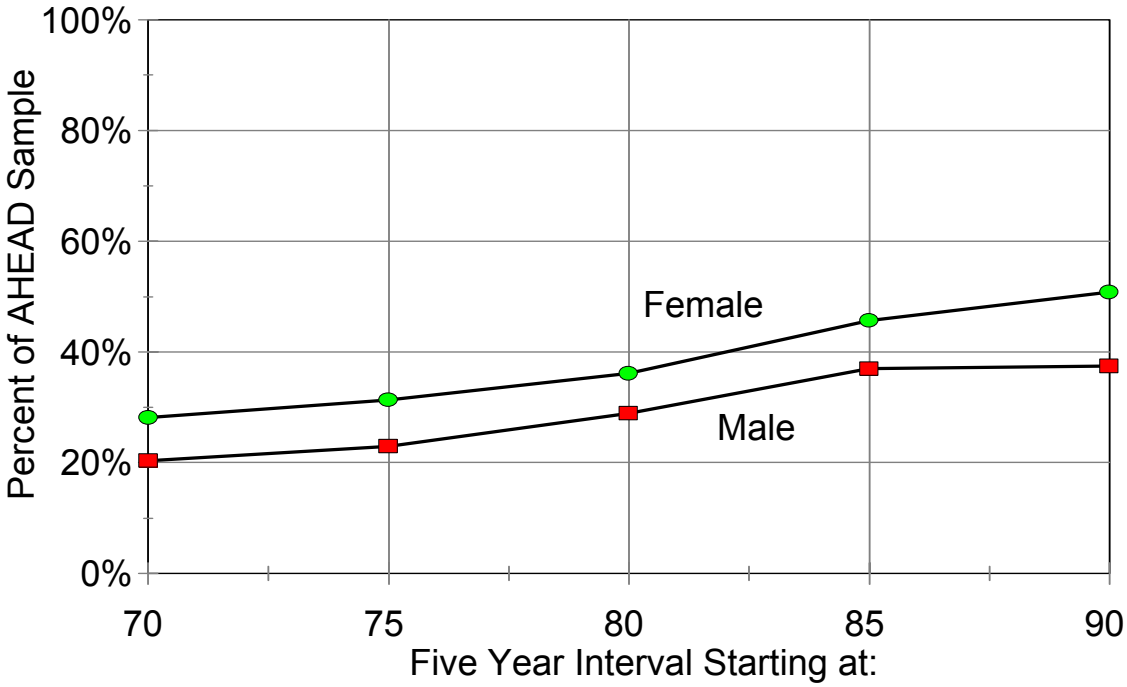
Ex. 10. Degenerative Health Conditions

Prevalence



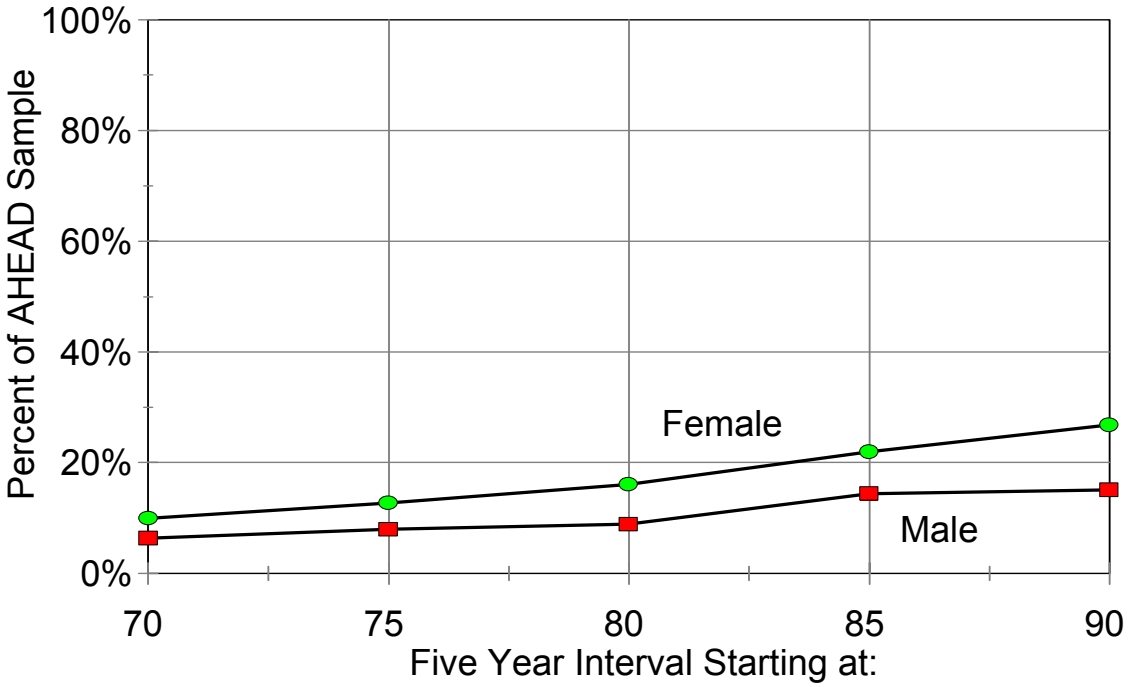
Ex. 11. Mental Conditions

Prevalence



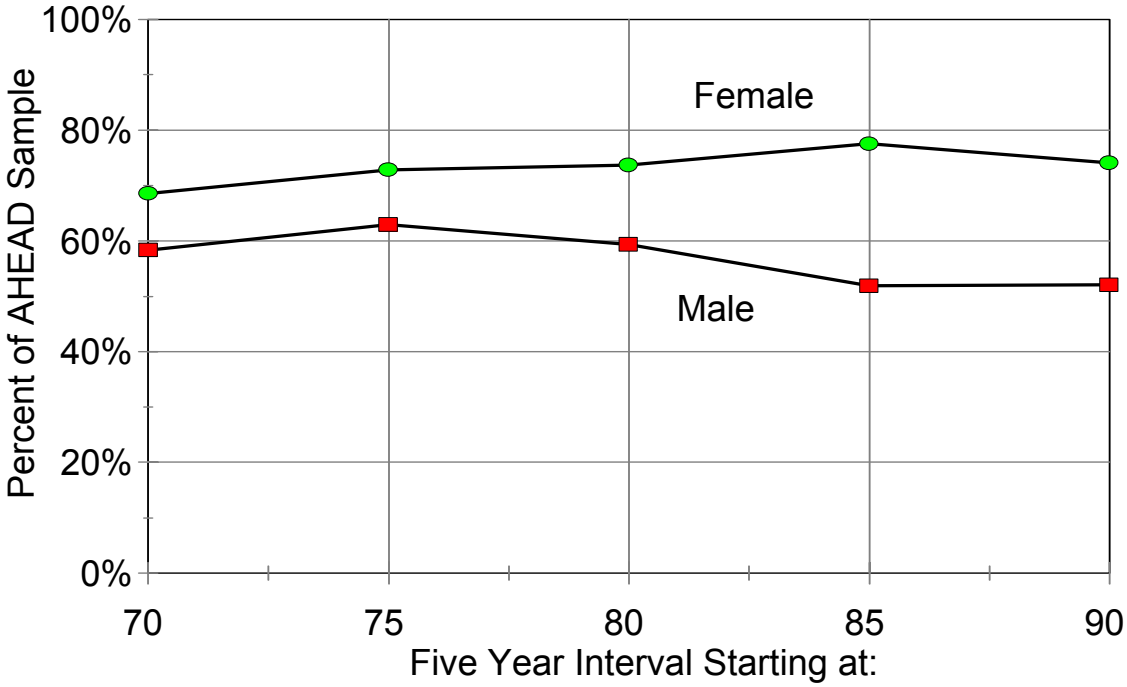
Ex. 12. Accidents

Prevalence



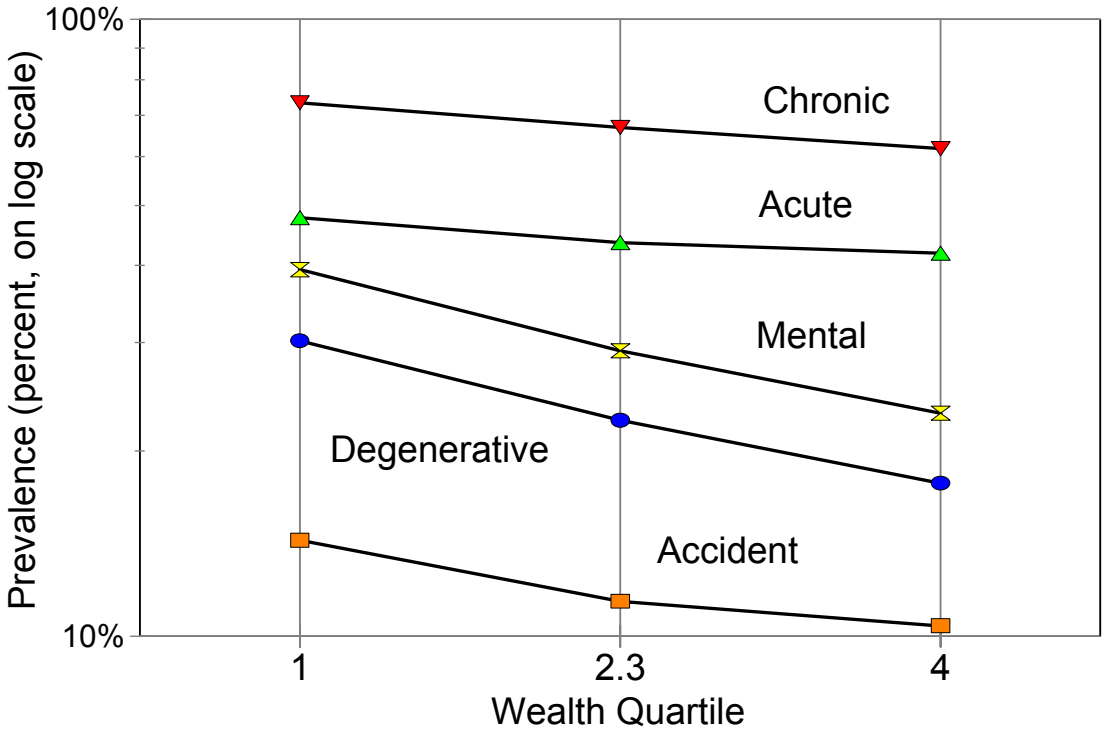
Ex. 13. Chronic Conditions

Prevalence



Ex. 14. Prevalence/Wealth Gradient

AHEAD Baseline

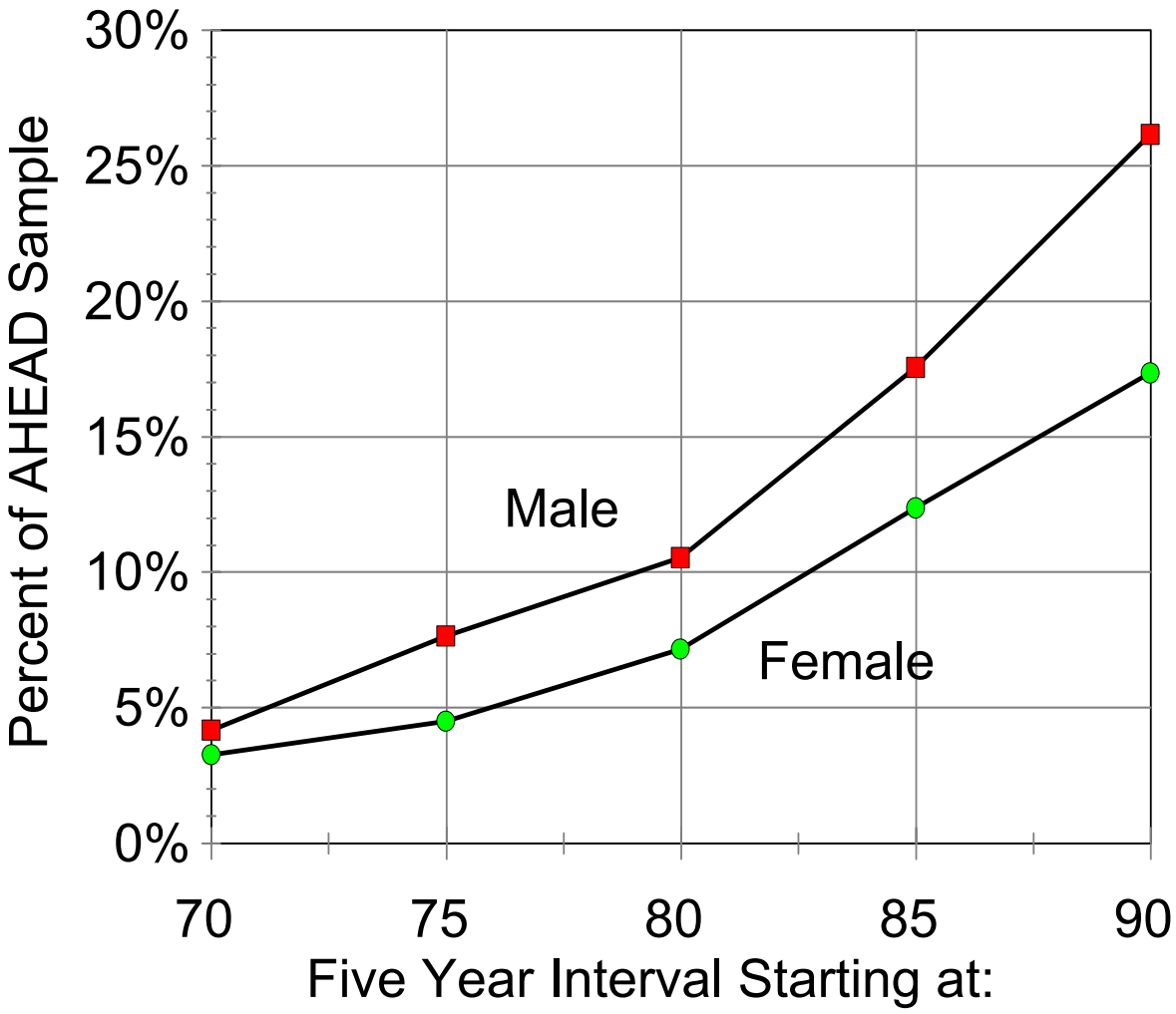


Ex. 15. Tests of Association of SES and Health Condition Prevalence

Group	Significance Level	Relative Odds Low vs High SES	Significant Covariates
Acute	0.000	1.29	Age, Sex, Marital Status, Race, Father's age at death, Smoking history, Wealth
Degenerative	0.000	1.90	Age, Marital Status, BMI, Smoking history, Wealth, Education, Condition of dwelling
Mental	0.000	1.53	Age, Sex, Marital Status, Race, BMI, Smoking history, Wealth, Income, Education, Condition of neighborhood and dwelling
Accident	0.005	1.32	Marital Status, Race, BMI, Wealth, Condition of neighborhood
Chronic	0.000	1.22	Age, Sex, Race, Mother's age at death, BMI, Wealth, Condition of neighborhood, Condition of dwelling
Self-Reported Health Status	0.000	2.30	Age, Sex, Father's age at death, BMI, Smoking history, Wealth, Income, Education, Condition of neighborhood and dwelling
ADL/IADL Impairment	0.000	2.53	Age, Father's age at death, BMI, Wealth, Income, Education, Condition of dwelling
Self-Reported Mortality Risk	0.000	1.28	Age, Marital Status, Mother's age at death, BMI, Education, Condition of dwelling

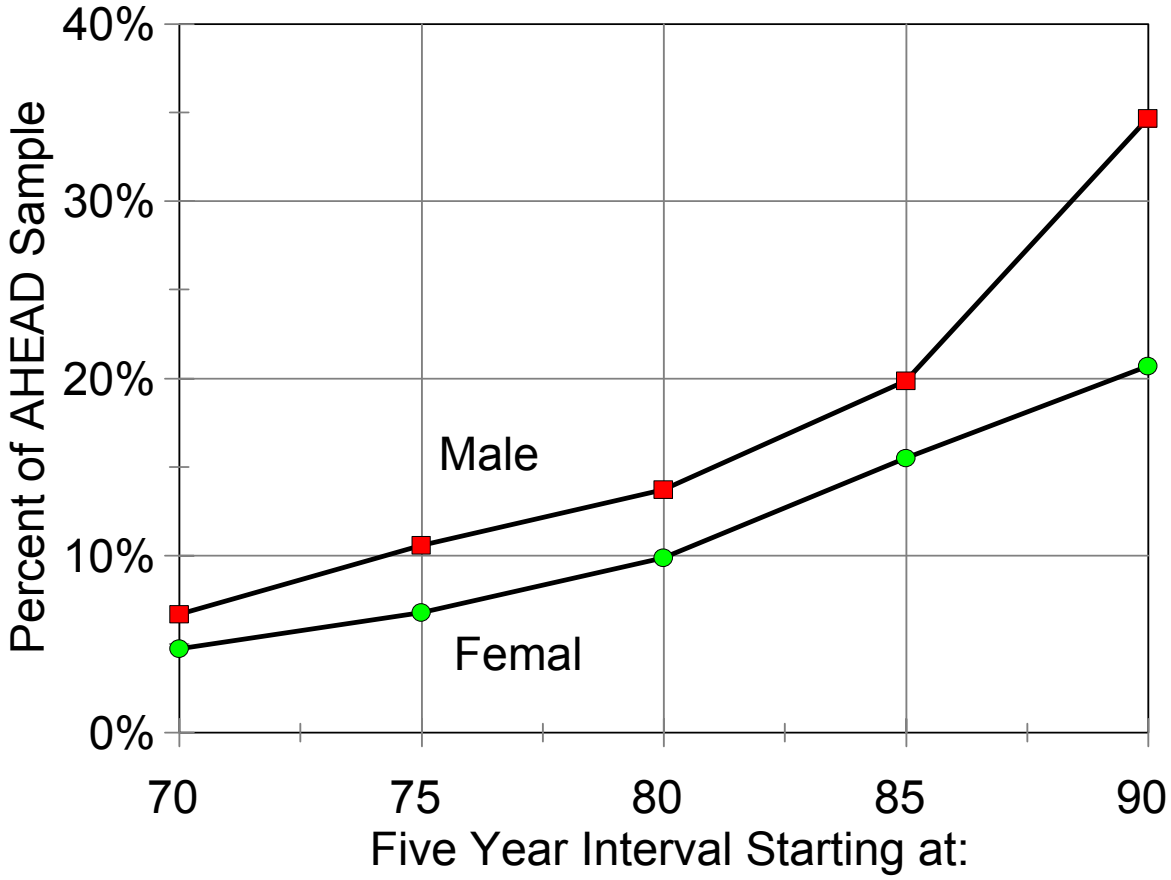
Ex. 16. Mortality

Annual Incidence Rates



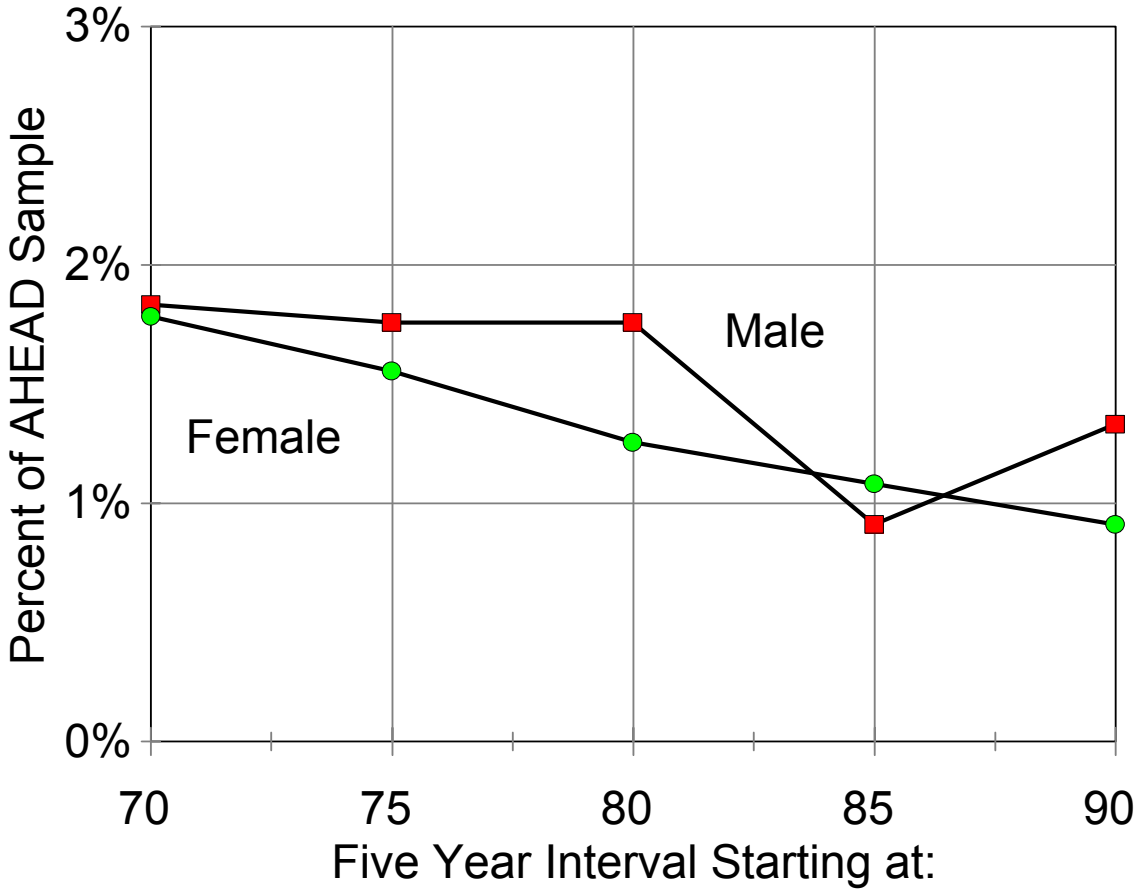
Ex. 17. Acute Health Conditions

Annual Incidence Rates



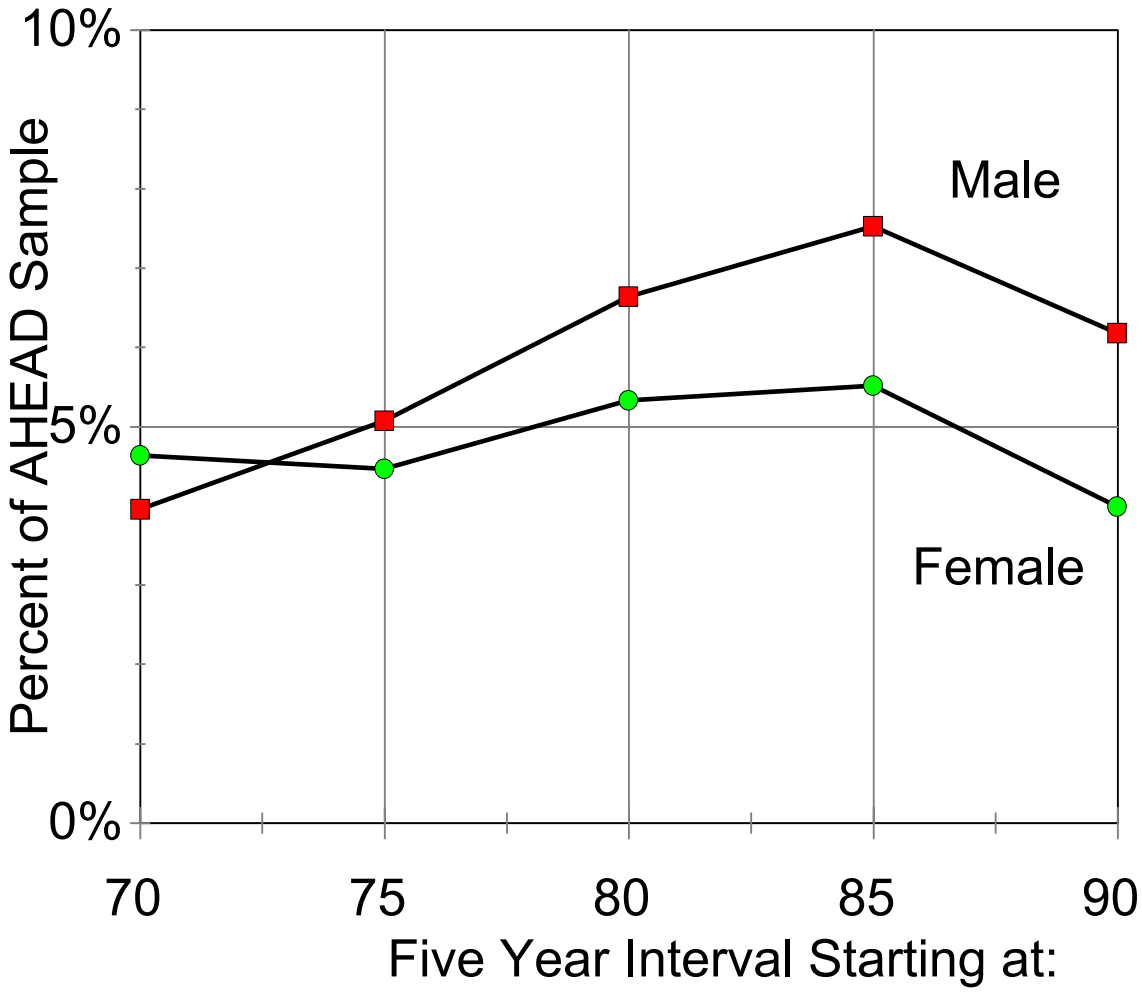
Ex. 18. Degenerative Conditions

Annual Incidence Rates



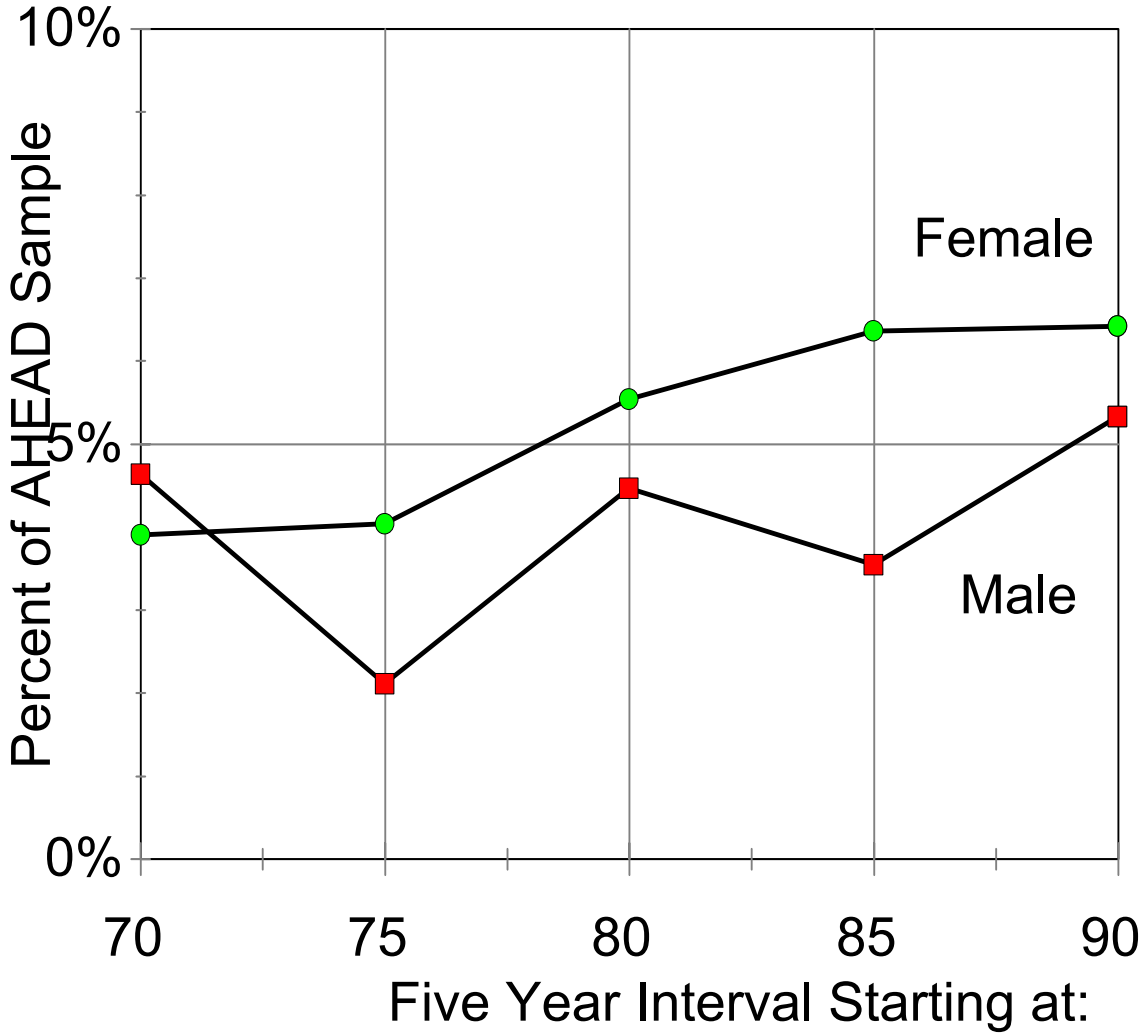
Ex. 19. Mental Conditions

Annual Incidence Rates



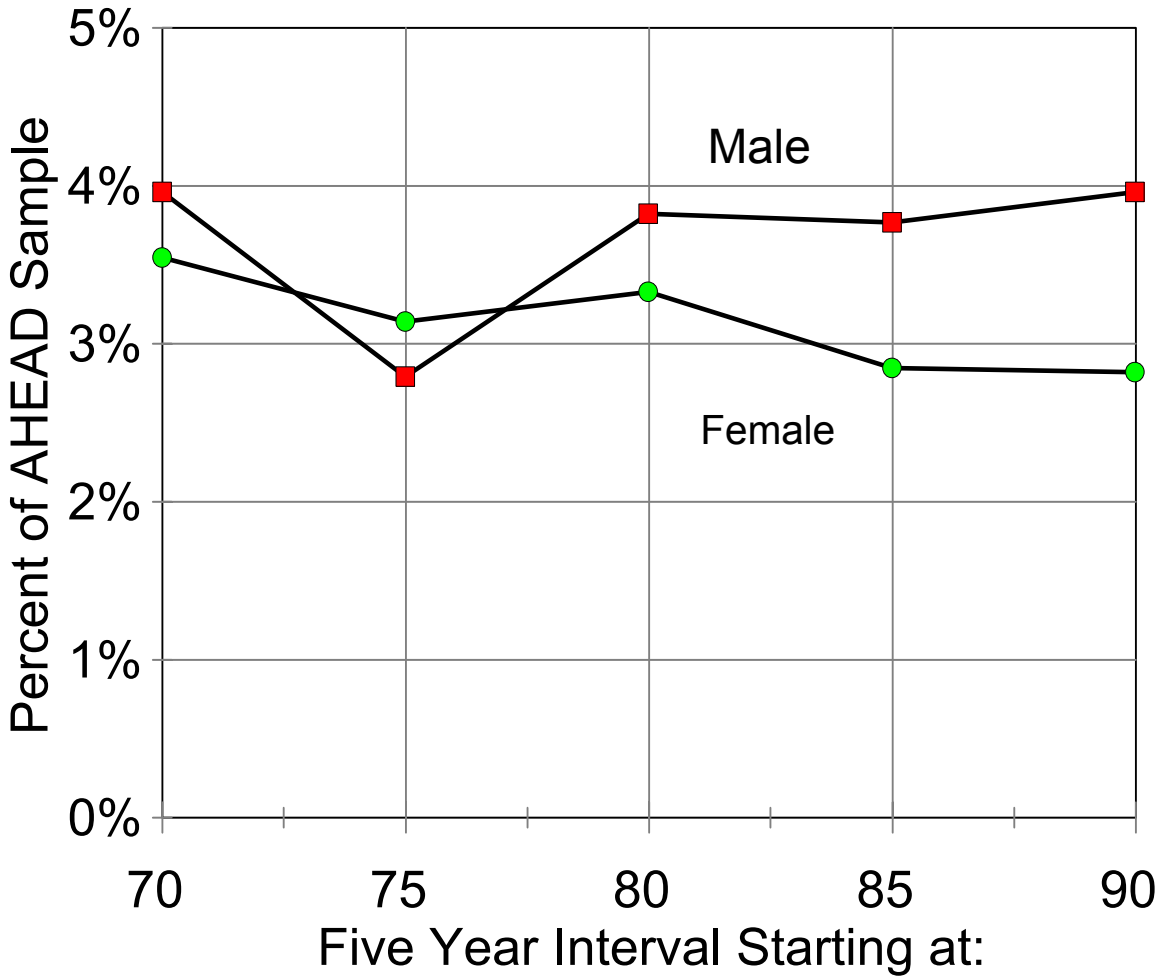
Ex. 20. Accidents

Annual Incidence Rates



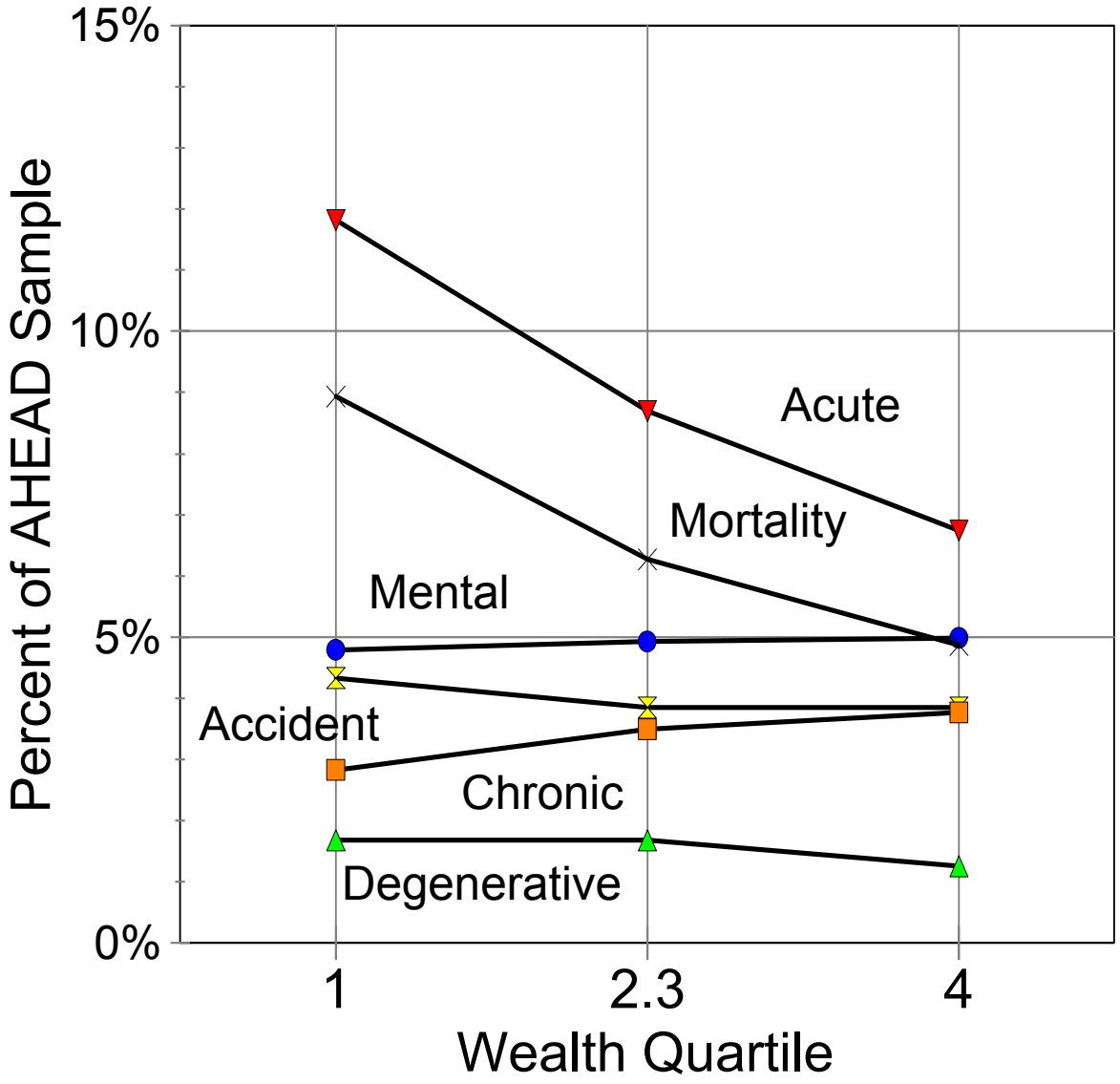
Ex. 21. Chronic Conditions

Annual Incidence Rates



Ex. 22. Incidence/Wealth Gradient

AHEAD Waves 1 to 3, Annual Rates



Ex. 23. Is SES Causal to Incidence of Health Conditions?

Group	Significance Level	Relative Odds Low vs High SES	Significant Covariates
Mortality	0.019	1.46	Age, Marital Status, Race, Parent's Ages at Death, Smoking history, Health (cancer, heart, stroke, lung, diabetes, depression, cognitive impairment, high blood pressure), Wealth
Acute	0.013	1.33	Age, Sex, Marital Status, Panents' ages at death, Smoking history, Health (cancer, heart, stroke, lung, diabetes, depression), Wealth
Degenerative	0.155	1.63	Sex, Smoking history, BMI, Health (heart), Wealth
Mental	0.026	1.13	Age, Sex, Mother's age at death, Health (hip fracture), Wealth, Education
Accident	0.571	1.14	Age, Sex, Race, Health (lung, diabetes, psychiatric, cognitive impairment,), Wealth
Chronic	0.140	0.42	Race, Mother's age at death, Health (heart, diabetes)

Ex. 24. Is SES Causal to Incidence of Health Conditions?

Group	Sig. Level Invariance	Sig. Level Causality	Sig. Level Joint	Relative Odds Lo v Hi SES
Mortality	0.641	0.658	0.718	1.24
Acute	0.000	0.147	0.000	1.22
Degenerative	0.105	0.036	0.028	2.29
Mental	0.389	0.000	0.000	1.48
Accident	0.259	0.536	0.317	1.22
Chronic	0.987	0.004	0.481	1.16

Ex.25. Are Health Conditions Causal to Innovations in Wealth?

Group	R-Squared	Significance Level for Invariance	Significance Level for Causality
Couples	0.1621	0.999	0.999
Singles	0.1597	0.999	0.674