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# A cardiovascular life history

## A life course analysis of the original Framingham Heart Study cohort

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**Aims** The objective of this paper is to measure the potential burden of cardiovascular disease within the original Framingham Heart Study cohort by transforming its well-described epidemiological measures into time-based health policy measures, such as life years lost to or lived with the disease.

**Methods and Results** We constructed multi-state life tables of the Framingham Heart Study cohort to calculate dwelling times with a history of cardiovascular disease. Age-specific probabilities determined transitions from healthy through disease to death. For this synthetic cohort, from age 50 men (women) live on average 26 (32) years; 20 (26) free of cardiovascular disease. Allowing occupancy of more than one disease state, 50-year-old males (females) live 2·9 (1·2) years with a history of myocardial infarction, 0·93 (1·2) with a history of stroke, and 0·67 (0·93) with congestive heart failure. Having ever suffered acute myocardial infarction, stroke or congestive heart failure, life expectancy is reduced by 9 (13), 12 (15) or 16 (16) years, respectively in 60-year-old men (women).

**Conclusions** Transforming occurrence probabilities into time-based health measures, the prevalence of cardiovascular disease is remarkable: from age 50, 20% of remaining life expectancy is lived with the disease. Such measures are integral to appropriate health planning and assessment of the potential population health value of various treatment and prevention strategies.
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**Key Words:** Cardiovascular diseases, morbidity, mortality, population, prevention.

## Introduction

Over the past 50 years the epidemiology of cardiovascular disease has been extensively investigated and described. Large prospective studies have identified and quantified the major modifiable risk factors[1–4] and numerous studies have demonstrated that altering these risk factors causes a reduction of event rates[5–9]. The same studies have also demonstrated the importance of irreversible risk factors. From a cardiovascular disease perspective, the presence of a Y-chromosome is a powerful genetic risk factor. Even more powerful is progressing age, with coronary heart disease risk doubling every decade at middle age[10,11]. However, while cardiovascular disease has been identified as a leading cause of disability and premature mortality[12], the population health burden of it and its various subtypes has not been fully analysed.

The significance of disease lies in the consequences to the life history of the individual, altering their healthy life-span. However, the complex interaction of competing forces of mortality and morbidity at older ages makes intuitive estimation of the impact of cardiovascular disease on this healthy life-span impossible. It is therefore of interest to transform the well-described death and incidence probabilities into life years lived, and life years lived free of disease and disability. The health effects of population trends and interventions decreasing incidence and/or mortality can then be translated into saved life years and saved life years free of disease or disability. These population measures, derived from a health-based life history, are necessary both for accurate assessment of changing health care needs and for intervention choices.

The backbone of such a life history is the age schedule at death, and disease. Parsimonious models estimating

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life histories from the age schedules of various events (such as marriage, or entry in the labour force) are a time honoured tradition in demography, laid down in the multi-state life table. We present the cardiovascular life history of the original Framingham Heart Study population, aged 28–62 at study onset, and followed-up between 1948 and 1991.

## Methods

### *Data source*

The Framingham Heart Study is a well-known ongoing longitudinal study focusing on cardiovascular disease. The original study cohort consisted of 5209 respondents from a random sample of adults aged 28 through 62 years residing in Framingham, Massachusetts, U.S.A. between 1948 and 1951. Follow-up within the study was performed by biennial physical examinations with life-style interviews and surveillance of hospital admissions, death registries and other available medical sources, ensuring highly accurate follow-up of death and clinically presenting cardiovascular disease. For the current study we used the data on age at cardiovascular disease or death collected during 40 years of follow-up (exam rounds 1 to 21) for the 5070 participants without cardiovascular disease at study entry.

The diseases examined were[13]: cardiovascular disease: incorporating all the types of cardiovascular disease listed below; coronary heart disease: defined as acute myocardial infarction, angina pectoris, and coronary insufficiency; cerebrovascular accident: defined as stroke (ABI, embolism, haemorrhage and other cerebrovascular accident), and transient ischaemic attack; congestive heart failure; and intermittent claudication. Within this report we refer to those free of cardiovascular disease as healthy and we analyse the states 'history of any cardiovascular disease', 'history of any coronary heart disease', 'history of acute myocardial infarction', 'history of stroke', and 'history of congestive heart failure'.

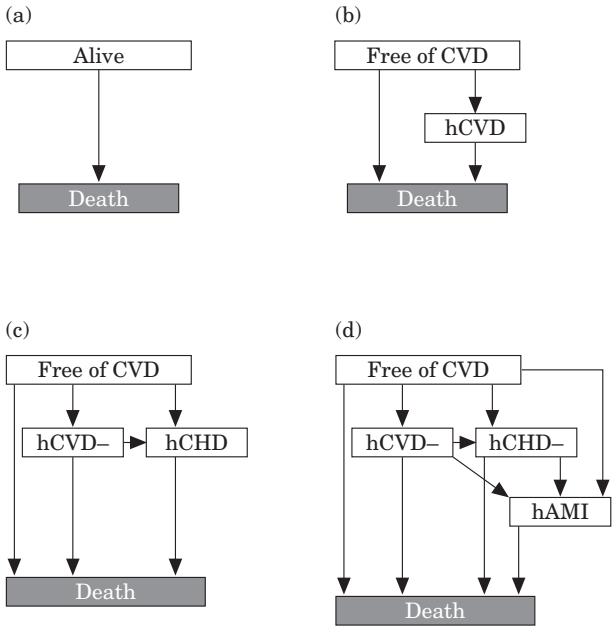


***Figure 1*** **Illustration of the life table models (a) represents transitions from alive to dead. The population within the 'alive' state is the total population, deriving total life expectancy. (b) Represents transitions from healthy to history of cardiovascular disease (hCVD) to death. Individuals can pass from healthy to death, spending no time in the cardiovascular disease state, or they can first transit through this state. This model estimates life expectancy (LE) free of cardiovascular disease and LE with cardiovascular disease. (c) Represents transitions from healthy to hCVD- (excluding coronary heart disease) to history of coronary heart disease (hCHD) to death. This model adds LE free of coronary heart disease and LE with coronary heart disease. Two other models with the same design were constructed — one with congestive heart failure and the second with history of stroke in the place of coronary heart disease. These models added LE free of congestive heart failure, LE free of stroke, LE with congestive heart failure, and LE with stroke. (d) Represents transitions from healthy to hCVD- to hCHD- (excluding acute myocardial infarction) to a history of acute myocardial infarction (hAMI) to death. This model adds LE free of acute myocardial infarction and LE with a history of acute myocardial infarction.**

### *Construction of life tables*

We constructed a number of different life tables for this report, based on an age-cohort observational plan[14]. The age-specific transition probabilities are calculated from the Framingham Heart Study. The life table is a synthetic cohort which experiences age-specific transitions as observed within the Framingham cohort. In the standard life table, an individual moves from life to death. In our hierarchical multi-state disease model an individual may exit through death but may also move from health to disease and death. The different models divide disease into specific disease states, but all states are hierarchically related and all transitions represent the first entry into a state, with no back-flow permitted (see Fig. 1). The set of models includes a general cardiovascular disease model and disease-specific models focusing on: coronary heart disease; acute myocardial infarction; stroke; or congestive heart failure. As each state represents ever having experienced a specified cardiovascular event, time spent at a state represents time spent at increased risk for further events and at increased probability of morbidity. However, while each disease label confers an implicit value weight of morbidity severity, this has not been quantified in this study.

Life table analysis is a duration analysis[15]. For instance, in the classic life table the average duration, or waiting time, until death is derived — known as the life expectancy. For chronic disease modelling, individual

***Table 1 Construction of the life table (data summarised from unabridged life table)***

| Age group | Entry | Death | Exit* | Pi† | Risk set‡ | Age | q(x)§ | l(x)‖ | L(x)¶ | T(x)# | e(x)** |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 40–49 | 1716 | 78 | 0 | 1859 | 2717 | 40 | 0·03 | 1·00 | 9·89 | 38·8 | 38·8 |
| 50–59 | 1364 | 326 | 0 | 3497 | 4179 | 50 | 0·08 | 0·97 | 9·39 | 28·9 | 29·7 |
| 60–69 | 119 | 674 | 19 | 4535 | 4585 | 60 | 0·15 | 0·90 | 8·38 | 19·5 | 21·7 |
| 70–79 | 0 | 1039 | 1217 | 3961 | 3352·5 | 70 | 0·32 | 0·77 | 6·55 | 11·1 | 14·5 |
| 80–89 | 0 | 762 | 649 | 1705 | 1380·5 | 80 | 0·63 | 0·52 | 3·60 | 4·56 | 8·77 |
| 90–99 | 0 | 153 | 139 | 294 | 224·5 | 90 | 0·90 | 0·19 | 0·93 | 0·96 | 4·92 |

*Age at censoring.
†Number alive at the beginning of the age interval: Pi(x)=pi(x − 1)+entry[x − 1,x) − death[x − 1,x) − exit[x − 1,x).
‡Number at risk of event at the beginning of the age interval: Risk set(x)=Pi(x) − 0·5*exit[x,x+1)+0·5*entry[x,x+1).
§Estimated probability of dying during the interval; taken from the unabridged life table.
‖Estimated survival function.
¶Person years lived within an age interval; summed from the unabridged life table.
#Total number of person years lived beyond age x given survival until age x; summed from the unabridged life table.
**Expected number of years lived by an individual beyond age x (equivalent to the mean age at death).

age is the time scale of interest. The age of the individual is measured at study entry, at the event(s) of interest, and at censoring. The attrition date is either the age at death or the age at censoring, and represents the final follow-up date. In this study the age at censoring is the age at examination 1 plus 40 years, representing the theoretical age at examination 21. Events occurring later were excluded. An example of construction of the life tables is given through a simple single decrement life table (life to death) in Table 1. Life tables represented the ages 40 to 101+, assuming a cardiovascular disease-free population at age 40. There were no further deaths from some disease states within the Framingham Heart Study cohort beyond age 95, so the probabilities of death between 96–100 inclusive were exponentially extrapolated from the values between ages 50 and 95. Standard errors around the residual life expectancies in the single state models (Fig. 1(a)) were calculated using the method described by Chiang[16].

***Table 2 Lifetime risk of developing cardiovascular disease for individuals free of cardiovascular disease at age 40 or 60***

| | Probability of developing disease | | | |
|---|---|---|---|---|
| | Within lifetime | | Before age 85 | |
| After age | 40 | 60 | 40 | 60 |
| Males | | | | |
| Cardiovascular disease | 67% | 62% | 63% | 57% |
| Acute myocardial infarction | 32% | 28% | 31% | 25% |
| Stroke | 16% | 17% | 15% | 16% |
| Congestive heart failure | 18% | 17% | 16% | 15% |
| Females | | | | |
| Cardiovascular disease | 59% | 57% | 49% | 45% |
| Acute myocardial infarction | 17% | 16% | 15% | 13% |
| Stroke | 21% | 21% | 15% | 14% |
| Congestive heart failure | 19% | 19% | 14% | 13% |

## Results

### *Event occurrence*

At study onset the 5070 cardiovascular disease-free Framingham Heart Study members ranged between the ages of 28 and 62 and were 45% male. Over 40 years of follow-up, 50% of this cohort developed cardiovascular disease and 60% died. Out of those who developed cardiovascular disease, 90% lived for at least one day after its identification. Of the original cohort, 34% developed coronary heart disease, 20% acute myocardial infarction, 14% stroke and 14% congestive heart failure. It is well known that cardiovascular disease mortality and incidence probabilities increase with increasing age, as does mortality from other causes. However, quantification of the population burden of the different diseases or determination of how this is distributed across age groups, is not intuitive. Creation of life tables from the transition probabilities enables analysis of lifetime probabilities of disease and dwelling times in the various health states.

### *Life history*

*Cumulative probabilities of disease*

The life table provides a simple method for the calculation of lifetime risks, automatically accounting for competing causes of morbidity and mortality, and censoring. For the synthetic cohort, the lifetime probability, at age 40, of getting any cardiovascular disease, including sudden cardiovascular death is 67% for males and 59% for females (Table 2). For 40-year-old males and females without cardiovascular disease, the lifetime probability of succumbing to acute myocardial infarction is 32% and 17%, respectively. One in five healthy

***Table 3 State-specific life expectancy (standard error around the further life years lived) for subpopulations with or without a history of disease at each given age***

| Age | Healthy* | Cardiovascular disease | Coronary heart disease | Acute myocardial infarction | Stroke | Congestive heart failure |
|---|---|---|---|---|---|---|
| Males | | | | | | |
| 50 | 26·7 (0·27) | 15·9 (0·75) | 16·4 (0·88) | 13·9 (1·1) | N/A† | N/A† |
| 60 | 20·0 (0·24) | 12·3 (0·39) | 12·6 (0·44) | 10·8 (0·53) | 7·98 (0·98) | 4·00 (0·62) |
| 70 | 13·5 (0·22) | 8·78 (0·26) | 8·85 (0·31) | 7·48 (0·33) | 5·50 (0·44) | 4·43 (0·41) |
| 80 | 8·29 (0·25) | 5·26 (0·28) | 5·15 (0·34) | 4·30 (0·35) | 3·75 (0·36) | 2·17 (0·29) |
| Females | | | | | | |
| 50 | 32·3 (0·26) | 20·3 (1·3) | 22·8 (1·9) | 14·9 (3·3) | N/A† | N/A† |
| 60 | 24·5 (0·23) | 16·1 (0·46) | 17·6 (0·57) | 11·6 (0·86) | 9·81 (0·86) | 8·26 (0·84) |
| 70 | 17·2 (0·22) | 11·0 (0·31) | 11·7 (0·42) | 7·18 (0·51) | 7·11 (0·48) | 5·45 (0·50) |
| 80 | 10·8 (0·22) | 7·02 (0·27) | 7·66 (0·38) | 5·34 (0·48) | 4·96 (0·39) | 3·30 (0·37) |

*Without a history of cardiovascular disease.
†Based on a risk set with less than 10 people.

40-year-old women and one in six healthy 40-year-old men will suffer a stroke. The lifetime probability of suffering congestive heart failure is similar for healthy 40-year-old men and women: 18% and 19%, respectively. The higher lifetime probabilities of stroke and congestive heart failure in females are largely caused by the greater female life expectancy. The probabilities at age 40 of acute myocardial infarction, stroke or congestive heart failure before the age of 85 are all greater or equal in males than females (31% vs 15%, 15% vs 15%, or 16% vs 14%, respectively).

*State-specific life expectancies*

While cumulative risks are an important indicator of the potential burden of a disease, they give no indication of the impact of that disease on life expectancy. Cardiovascular disease is a significant cause of premature mortality and here we present the estimated years of life lost by specified disease groups within our life table cohort due to its high mortality risks. Table 3 shows the state-specific differences in life expectancies between the population without cardiovascular disease and the subpopulations with a history of any cardiovascular disease, any coronary heart disease, acute myocardial infarction, stroke or congestive heart failure at a given age. The population without cardiovascular disease at a given age may develop it in the future. The state-specific life expectancies refer to individuals who are in a specified disease state for more than a single day before death. At age 50, a man free of cardiovascular disease may expect to live 27 years and a similar woman 32 years. At age 70, such a man and woman will live, respectively, 14 and 17 years. Any cardiovascular disease will make a 50-year-old person lose 11–12 years of life, and a 70-year-old person lose 5 (as a man) or 6 (as a woman) years of life, compared to those without cardiovascular disease. The severe impact of a history of acute myocardial infarction, stroke or congestive heart failure on residual life expectancy is illustrated in Table 3.

*Life expectancy*

The extra value of the multi-state life table lies in its ability to synthesize the consequences of age-specific incidence and mortality probabilities and calculate life expectancies, or dwelling times, in specific disease states. This adds the measure of years lived with a history of disease to that of reduced life expectancy, indicating more accurately the potential public health burden of the disease. Within this synthetic cohort, cardiovascular disease-free at age 40, total life expectancy from the age of 50 was 26 years for males and 32 years for females, consistent with a relatively healthy population[18] (Table 4). At age 50 a striking 20% (6·3 years for males and 5·7 years for females) of a population's residual life expectancy is spent with cardiovascular disease (Table 4; Fig. 2). Much of this, 4·7 (males) and 3·7 (females) years, is spent with coronary heart disease, with lesser amounts spent with stroke (0·93 and 1·2 years) or congestive heart failure (0·67 and 0·93 years). Individuals can be in more than one disease state at any point in time, but we have not specifically modelled this co-morbidity. As mentioned, while the age-specific event probabilities for all the examined cardiovascular events are consistently higher in males, the greater longevity of females means that the burden of disease can be higher in females. Fifty-year old females will live on average 0·29 more years with stroke and 0·26 more with congestive heart failure when compared to males. This compares to 50-year-old males spending 0·61 more life years with any cardiovascular disease, 1·0 more with any coronary heart disease and 1·6 more with a history of myocardial infarction.

Partial life expectancies illustrate the changing burden of cardiovascular disease with age, representing the average number of years lived within a 5-year period per individual (independent of health status) present at the beginning of the period. While disease incidence increases with age, so does disease-specific and all-cause mortality. The combination of these results in different

***Table 4 Life expectancy (LE) and residual LE free of disease from specified ages, based on a population free of cardiovascular disease at age 40***

| Age | LE | LE (proportion in %) free of a history of: | | | | |
|---|---|---|---|---|---|---|
| | | Cardiovascular disease | Coronary heart disease | Acute myocardial infarction | Stroke | Congestive heart failure |
| Males | | | | | | |
| 50 | 26·2 | 19·9 (76) | 21·5 (82) | 23·3 (89) | 25·2 (96) | 25·5 (97) |
| 70 | 12·0 | 7·38 (61) | 8·69 (72) | 9·94 (83) | 11·2 (93) | 11·4 (95) |
| Females | | | | | | |
| 50 | 32·1 | 26·4 (82) | 28·4 (89) | 30·9 (96) | 30·9 (96) | 31·2 (97) |
| 70 | 16·0 | 11·3 (71) | 13·0 (82) | 15·0 (93) | 14·9 (93) | 15·2 (95) |

distributions of the burden of the different diseases, shown in Fig. 3 up to the age of 90. The years lived within each 5-year period with a history of myocardial infarction or congestive heart failure increases to 0·78 years and 0·24 years, respectively, for males between the ages of 75–79. The burden of stroke peaks between the ages 80–84 with 0·39 years lived with a history of the disease. After these ages the disease-specific partial life expectancies decrease. In contrast to this rise and decline over time, women have a continually greater amount of each 5-year period lived with these diseases. After the age of 90 the number of years lived with a history of myocardial infarction appears to decrease for women, but small numbers within the data-set limit accurate estimation. Greater female longevity results in a decreasing difference between the male and female partial life expectancies for each of the diseases with increasing age.

## Discussion

Here we present an analysis of life expectancy with and without cardiovascular disease, estimated from a white American population: the Framingham Heart Study original cohort. The enormous impact of cardiovascular disease on the human life course is translated into life years lost to disease and life years lived with a history of disease within a synthetic cohort. From the age of 40, more than 60% of the population develop cardiovascular disease within their lifetime, reducing residual life expectancy by 11 and 12 years, for 50-year-old males and females, respectively. Lloyd-Jones *et al.*[19] reported that one in two men and one in three women would develop coronary heart disease from the age of 40. In addition, one out of six men and one out of five women will suffer a stroke, and consequently from age 60 lose on average 12 and 15 years of life, respectively. One in three men and one in six women will suffer an acute myocardial infarction, and consequently from age 60 lose on average 9 and 13 years of life, respectively. One in five men and women will experience congestive heart failure and consequently from age 60 lose on average 16 years of life. We have shown that the greater longevity of women is the primary cause of both their greater lifetime probabilities of congestive heart failure and stroke and the greater number of years of life lost for an equivalent disease, when compared to men.

Cardiovascular disease not only reduces life expectancy, it is also a major cause of morbidity. Its potential contribution to population morbidity is highlighted, with approximately 20% of this life table cohort's residual life expectancy from the age of 50 spent with the disease. Taking into account disease-associated increases in mortality, 50-year-old men spend 11% of their residual life expectancy with a history of myocardial infarction, 4% with a history of stroke and 3% with congestive heart failure. Fifty-year-old women spend 3–4% of residual life expectancy with each of these diseases. For men, the highest disease burden is experienced between the ages of 75 and 85, where around 0·8, 0·4 and 0·2 years out of five are spent with a history of myocardial infarction, stroke and congestive heart failure. For women, the patterns of stroke, myocardial infarction and congestive heart failure morbidity do not peak until at least ten years later. The health care implications of these results are that for every 1000 75-year-old men approximately 780, 310 and 240 person years of care will need to be provided for the consequences of myocardial infarction, stroke and congestive heart failure, respectively, within the following 5 years. One thousand 85-year-old women need approximately 380, 390 and 290 person years of care provided for the consequences of myocardial infarction, stroke and congestive heart failure, respectively.

These results demonstrate the utility of transformation of epidemiological data into time-based health policy measures. While epidemiological data enables prediction of the number of coronary heart disease events and deaths, the life table also enables estimation of the overall potential burden of specific diseases in terms of years of life lost to and lived with disease. This combined effect of differences in disease incidence and mortality probabilities cannot be intuitively estimated but is important for health care planning. Previous analyses have demonstrated the burden of cardiovascular disease to be a loss of approximately 15 300 years of life and approximately 3000 years lived with disability in countries such as the U.S.A. and Western Europe[12].

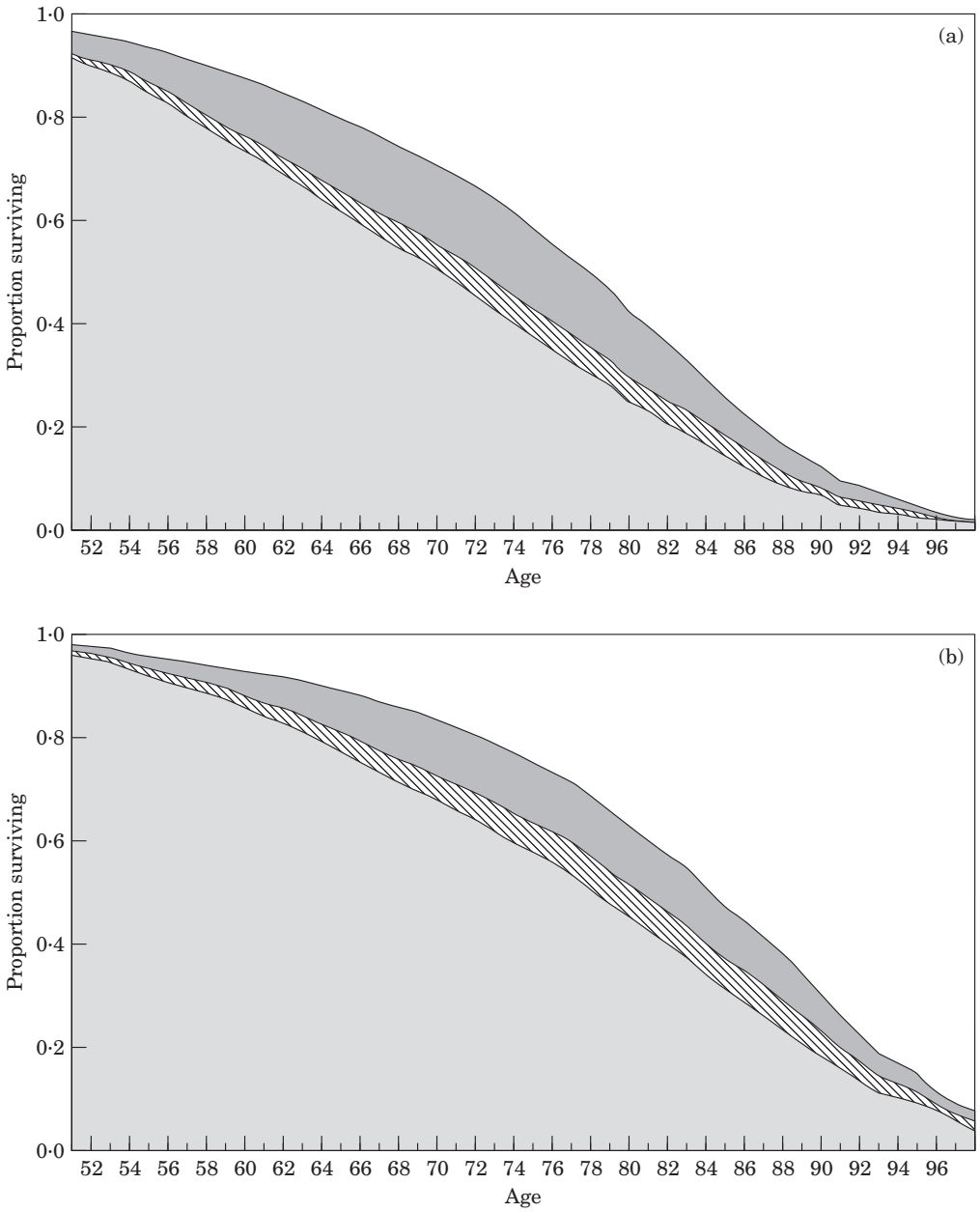


***Figure 2*** **Survival of a cohort of cardiovascular disease-free 40-year-old males (a) or females (b) demonstrating the proportion of life spent: free of cardiovascular disease (light grey portion); with a history of coronary heart disease (dark grey portion); or with a history of other (non-coronary heart disease associated) cardiovascular disease (patterned portion).**

However, more detailed analyses have not been previously performed. The advantages of the present analysis are the range of cardiovascular disease subtypes and age groups examined, the accuracy of disease definitions and the internal consistency of the various transition probabilities within the Framingham Heart Study.

One of the model's strengths is that it represents the relationships within a single, homogeneous historical cohort. However, one of its major limitations is also derived from this property. Because of the long follow-up and the broad age range at onset of the cohort, the forces of mortality and disease incidence by age are a mixture of cohort and period effects. At younger ages, the cohorts are exposed to the higher mortality of the more ancient periods; at older ages, the cohorts are exposed to the lower mortality of more recent periods. Consequently, transition probabilities for the intermediate ages are derived from a number of different periods. In addition, coronary heart disease case-fatality and incidence rates changed significantly during this period in the U.S.A.[20–22]. As people are

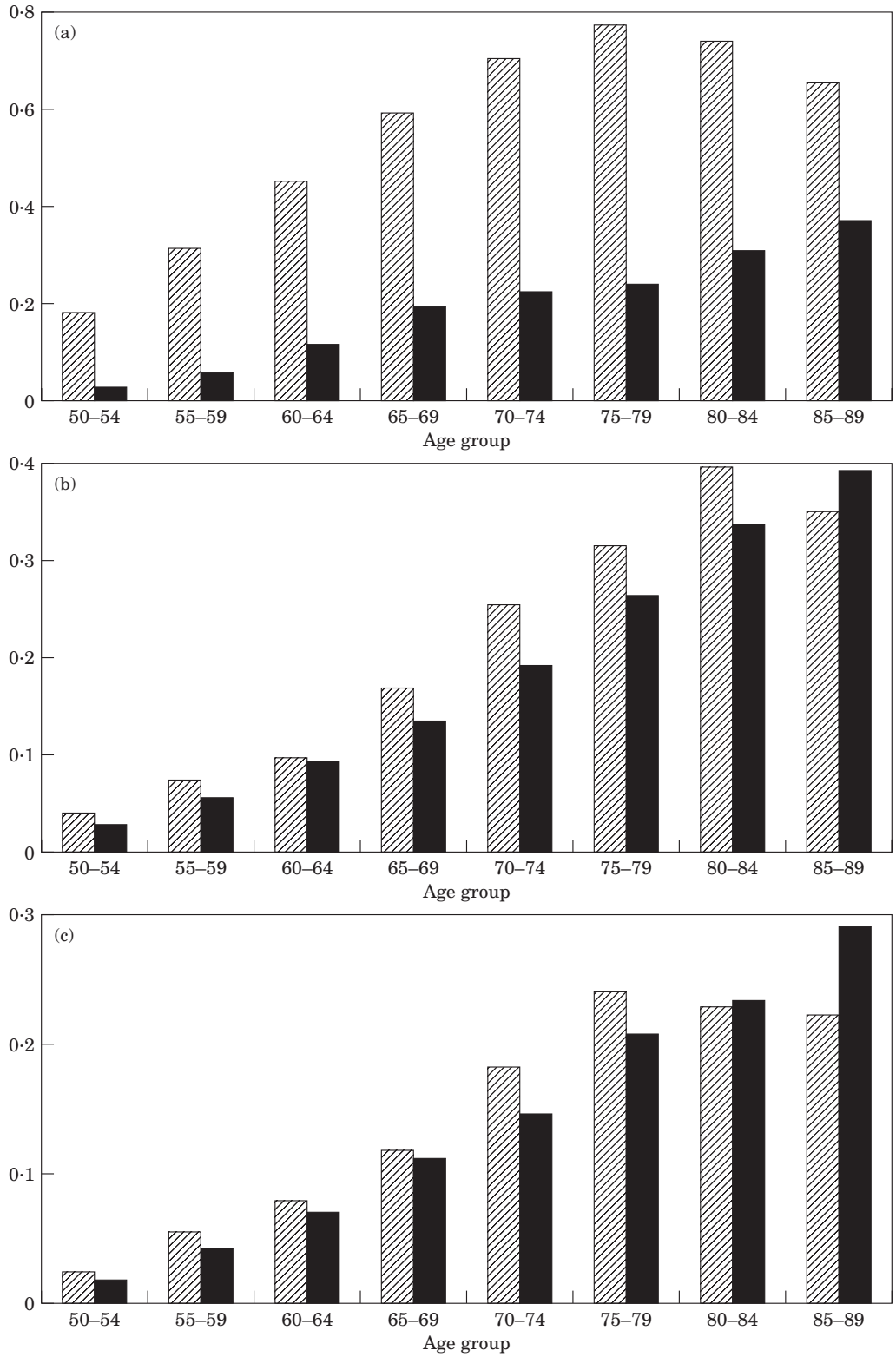


***Figure 3*** **Five-year partial life expectancies with disease for males (▨) and females (■). The partial life expectancy represents the average number of years lived with disease throughout a 5-year period per individual alive (independent of disease status) at the beginning of that period. The disease states represented are (a) a history of acute myocardial infarction, (b) a history of stroke, and (c) a history of congestive heart failure.**

surviving longer with cardiovascular disease, we expect the life expectancies with cardiovascular disease presented here to be less than those for current low mortality populations. The extent to which this is offset by decreasing cardiovascular disease rates is still to be determined. An analysis of life tables constructed solely using the period between exams 11 and 21 (approximately 1970–1990) indicated that total life expectancy and life expectancy with cardiovascular disease from the age of 50 were approximately 1 and 0·4 years higher than those presented here (data not shown). The low number of events in the restricted period prohibited the analysis from being solely carried out using this data. These results suggest that the total and disease-specific life expectancies presented here are an approximation of those experienced by a similar population today.

The second limitation of these analyses lies in the interpretation of the potential burden of morbidity. All our analyses refer to membership of a disease state, which can only ever be an approximation of the disease burden without taking into account the severity of the health state. This is particularly an issue for the burden of coronary heart disease, which is derived from a very heterogeneous group. The morbidity due to angina pectoris can vary greatly between individuals and can change in response to readily available treatments such as revascularization. Due to these uncertainties of disease history and date of onset, angina pectoris has not been modelled explicitly within this analysis. The next step will be to add in one further layer of complexity: estimations of the severity of disease, or disability, associated with the specific health states.

One further limitation is that the current model structure is primarily of use for descriptive rather than interventional analyses. Here a unidirectional flow is used as the simplest way to capture all time spent with a history of disease without the creation of further mixed disease states. In addition, inclusion of back-flow in the model requires age- and gender-specific transitions from all health states, requiring more power than was available from the Framingham original cohort. While this structure is appropriate for the descriptive analyses presented here, a more biological pathway would be preferred (for example allowing coronary heart disease to congestive heart failure transitions or remission from angina pectoris) for any interventional analyses.

A dilemma currently facing low mortality societies is how to best maximize the health of increasingly long-lived people. One of the major targets for improving population health is cardiovascular disease. These results quantify the striking population burden of cardiovascular disease. Decisions regarding which treatment and prevention strategies to follow and how best to respond to changing secular trends are made difficult by the complexity of the interactions between mortality and morbidity at older ages. Here we present a simple and transparent method to enable meaningful conclusions about the potential impact on population morbidity and mortality of specific interventions and societal trends.

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