The effect of retirement on mortality

*Event history analyses and quasi-experimental evidence from Norway*

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Summary

On average, people who retire earlier tend to die sooner. This is partly because poor health is an important reason for early retirement. But does retirement affect mortality? A number of studies have investigated the relationship between retirement and mortality. While the majority of studies find earlier retirement to be associated with higher mortality, there is no consensus about the causal relationship between retirement and mortality.

In this thesis, I first investigate the association between retirement and mortality in two full Norwegian birth cohorts (1906 and 1907). Event history analyses show that those who have retired have a higher mortality risk than those who are the same age but have not retired. This difference increases with time since retirement, even when disability pensioners are excluded.

I then turn to a pension reform that came into effect in 1973. This reform lowered the eligibility age for old age pensions in the national insurance scheme from 70 to 67 years. I treat this reform as a natural experiment whereby some birth cohorts were allowed to retire up to three years earlier than other cohorts. This allows me to estimate the effect of being eligible for retirement and the effect of actually retiring earlier. For these analyses, I sample five full Norwegian birth cohorts born between 1902 and 1906. I then exploit the exogenous variation in retirement eligibility age that stems from this reform by employing difference-in-differences, individual fixed effects and instrumental variables methods. The main advantage of these approaches is that the estimates can be given a causal interpretation.

There are several other advantages to studying the 1973 reform. More or less the entire mortality history of the affected cohorts can be studied, and the reform affected most of the working population. Additionally, mortality was higher in the 1970s among people in their late sixties than among younger groups affected by later reforms.

The results show that retirement increased mortality in 1970s Norway. Retiring one year earlier increased the probability of dying before age 80 by 1.5 percentage points among men and 0.5 percentage points among women. Among men, the effect was larger among those with low education, while the opposite was true for women. These effects appear to be driven by a relatively short-term increase in mortality following retirement. In sum, these results are in line with the view of retirement as a stressful life event and with theories predicting retirement to have short-term negative health consequences. The results can thus be taken to support the notion that postponing retirement may entail some health benefits.
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None of the people mentioned here bear any responsibility for errors in this thesis. That responsibility is mine.

Adrian Farner Rogne
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1 Introduction

1.1 Earlier retirement and longer lives

Between the 1970s and early 2000s, there has been a trend towards earlier retirement in the OECD countries. This development has occurred despite general improvements in life expectancy, living conditions, health behavior and access to health care. Life expectancy has risen steadily in industrialized countries for decades, contributing to aging populations and prospects for increasing public pension expenditures (OECD, 2013; OECD/EU, 2014). Both rising life expectancies and earlier retirement mean that people spend an ever greater portion of their lives as retirees and that retirees constitute an increasing proportion of the population. Faced with aging populations and issues related to the fiscal sustainability of their pension systems, many countries have introduced pension reforms aimed at reducing the consequences of an increasing old age dependency ratio. These reforms have commonly included measures such as higher retirement ages, adjustment of benefits in relation to increasing life expectancies and the modification of indexation rules for pension benefits. Recent figures indicate that this has contributed to a slight increase in effective labor market exit ages over the last decade (OECD, 2013).

In light of these interrelated social, demographic and political trends, it should be no surprise that questions relating to retirees, retirement decisions, pension systems and the health and mortality of old people are becoming increasingly important research topics in several disciplines. Among the questions that have been raised in the scientific literature, are those concerning the relationship between retirement, health and mortality. Do people who retire earlier die sooner? Does poor health predict retirement and vice versa? Does retirement affect health and mortality? Answers to these questions should be of interest to both policymakers and individuals making retirement decisions.

In this thesis, I look at the relationship between retirement and all-cause mortality, and attempt to answer whether people who retire early die sooner and whether retirement affects mortality. I also investigate whether the effect varies between people of different educational levels and between the sexes. In doing this, I draw on research from several scientific disciplines, including geriatric medicine, psychology, economics and demography. However, I hope this thesis demonstrates that the subject can be analyzed and understood using sociological concepts and perspectives. After all, the questions relate to some of the core
1.2 Does retirement kill you? And if so, why does it matter?

A substantial body of research has investigated the relationship between retirement and health, and between retirement and mortality. Many of these studies have investigated whether and how retirement is associated with longer or shorter lives or relative changes in health. Others have studied whether there is a causal relationship between retirement on the one hand and either health or mortality on the other. Questions about association and questions about causality are both highly interesting and merit thorough investigation. But they have different implications for both policy and individual behavior.

One imaginable scenario could be that earlier retirement is associated with relative health deterioration and/or increased mortality risk, but that this relationship is due to selection processes rather than causation. In this case, the individual should not be too concerned about potential health and mortality consequences of retirement decisions. For policymakers, however, this information could be very useful as it identifies early retirees as a potential risk group and it would point towards health improvements among older workers as a potentially important means to prolong people’s working lives. If the relationship is chiefly a causal one, however, individuals should perhaps be advised to take health and mortality concerns into account when making retirement decisions. Also, policymakers may be interested to know if the fiscal gains from raising the minimum age of retirement in order to reduce pension expenditures may be offset by greater health expenditures. The opposite case, that retirement benefits health and longevity, would perhaps be an argument in favor of preserving the opportunities to retire early for health reasons and the flexibility of the Norwegian pension system. But the combination of earlier retirement and resulting longer life expectancies may in itself pose a challenge for pension systems. Finally, it is plausible that both causal processes and selection processes are at work. In such a case, it would be interesting to know about the relative strengths of such processes. In sum, the policy implications of research on this topic are intricate but interesting.

This thesis addresses both the question of association and the question of causality, and it adds to the research literature in several ways. First, it uses longitudinal data on two full Norwegian birth cohorts (1906 and 1907) to explore the relationship between retirement and
all-cause mortality in event history analyses. Second, it samples the cohorts born between 1902 and 1906 and exploits a pension reform that lowered the eligible retirement age by three years. These analyses provide estimates of the causal effect of retirement on subsequent mortality, using both difference-in-differences (DiD), individual fixed effects (FE) and instrumental variable (IV) methods. The use of this reform allows for an analysis with a long observation window where I can observe (virtually) the entire mortality history of the birth cohorts under study. The reform took place in 1973, and affected people in an age group and a period with higher mortality than those affected by later reforms. These “favorable” conditions mean that the reform can be seen as constituting a critical case (Flyvbjerg, 2006); it is a scenario where one should expect to be able to identify an effect if there is one. Third, the thesis provides a test of a common interpretation of Robert C. Atchley’s (1976) theory about different phases of retirement, namely that retirement causes short-term fluctuations in age-specific mortality (Ekerdt, 1987; Minkler, 1981; Solem, 1987).

The identification strategy used in this thesis is a strength with regard to the internal validity of the causal claims I make. However, the use of the 1973 reform clearly make the results somewhat dated, and this should inform the generalization of the results. Although the reform affected more or less the entire elderly working population, the Norwegian society has changed a great deal since 1973. Therefore, the effects estimated here will be local (meaning that they relate to a specific time period, group and context), and may not be generalizable to other time periods or contexts. On the other hand, the causal mechanisms producing an effect in the 1970s may very well still be at work today. In the end, the generalization of the findings is a theoretical exercise, and making an informed assessment requires knowledge about the context and how Norwegian society has changed over time.

1.3 The Norwegian setting
Norway is a Nordic welfare state with a relatively generous public pension system and affordable public healthcare (Kjølsrød, 2011). Since the end of World War II, Norway has experienced a formidable economic growth. This growth has been helped by the discovery of offshore oil reserves on the continental shelf at the end of the 1960s (Brathaug and Skoglund, 2012; Eika, 2008), and Norway is currently ranked fourth in the world when it comes to per capita GDP (The World Bank, n.a.). The petroleum sector has, in combination with high and progressive taxes, allowed for the development of an expansive public sector with low-priced
or freely provided public services, while at the same time maintaining relatively moderate income inequality. Between 1970 and 2008, the number of people employed in the welfare services sector has more than tripled (Kjølsrød, 2011).

In 1967, the national insurance scheme (“Folketrygden”) was introduced. Over the next few years, a number of different social security benefits (such as sickness benefits, unemployment benefits and pensions) were organized into one large system. With a few exceptions, the national insurance scheme covers everyone who resides in Norway. The pension system in the national insurance scheme has been subject to many small reforms and revisions over the course of its history. Of special importance for this thesis is the fact that the minimum retirement age was lowered from 70 to 67 years in 1973. More recently, the entire pension system was subject to a major reform in 2011 (Mæland and Hatland, 2016).

At the same time as the national insurance scheme was introduced, Norway started recording detailed data on its population in national population-wide registers. Today, these registers contain demographic information such as migrations, marriages and childbirths, and information on tax records, education histories, the use of public benefits such as pensions and much more. These rich, high-quality individual-level data make it possible to prospectively investigate the relationships between retirement and mortality in a population-wide sample with an observation window of up to nearly fifty years. In the present thesis I exploit both the long observation window and the aforementioned 1973 reduction in retirement eligibility age to investigate the relationship between retirement and mortality.

The body of theory I discuss below suggests that retirement may affect health and mortality both positively and negatively. This effect may work through several channels, such as through reduced income or more available time, through relief from work related strain, stress and health risks, through role or identity loss, and through changes in health behavior. While such mechanisms may perhaps be more or less universal, the size of the effect they produce may be highly dependent on national context, and there may also be substantial effect heterogeneity (i.e. difference in effects for different subgroups). In Norway, the pension system is relatively generous, the labor market is relatively regulated, and the healthcare system is relatively affordable and expansive. This could mean that any effect of retirement on health and mortality (be it positive or negative) could be attenuated in here compared to other countries. The large differences in labor market participation and the differences in
gender roles between men and women in the cohorts studied here could also mean that an effect may be larger for men than for women.

1.4 Defining retirement, health and health behavior
Retirement can be understood and conceptualized in different ways. Theoretically, I understand retiring as exiting (or greatly reducing) work due to old age and/or disability. In most cases, retirement is regulated and associated with public benefits, but people may also retire on their own savings. Being disabled or unemployed and receiving disability or unemployment benefits is not the same as being retired. But the distinction is not always sharp and the timing of retirement is sometimes hard to pinpoint. People may retire for a time, then go back to work (“bridge employment”), and people may move directly from unemployment to retirement. Some people may even consider themselves retired while formally defined as unemployed, sick, disabled or not working for some other reason. In one sense, then, being retired is a subjective state. Following the 2011 pension reform, the opportunities for flexible retirement have also increased greatly (The Ministry of Labor and Social Inclusion/Arbeids- og inkluderingsdepartementet, 2006; Fredriksen and Stølen, 2011). But retirement is not only a subjective status, role or identity. It is also strongly related to a thoroughly regulated public welfare scheme. By providing pension benefits according to a set of rules and regulations, the government is both enabling and to some degree incentivizing people to retire, and the transition into retirement is then strongly related to the public benefits provided. From the perspective of the welfare state, retirees can be understood as those receiving retirement benefits.

In theoretical discussions I switch between the above definitions, but in the empirical section I use only benefit uptake as a proxy for retirement. When operationalizing retirement it would ideally be nice to have information on both work hour reductions, subjective assessments and public benefit recipiency, but only the last is readily available. It could be an option to operationalize retirement as combining pension uptake with pensionable income reductions. But pensionable income is an imperfect measure of work hours, the variable has a lot of missing values, there is a lot of time variation in pensionable income unrelated to retirement, and reductions in pensionable income are very strongly associated with retirement benefit uptake. Therefore, I simply use the uptake of retirement benefits as a proxy for retirement.
The World Health Organization (WHO, 1946) defines health as “a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity.” In a sense, with such a definition, hardly anyone is really healthy. Others have suggested that health should be understood as being able to cope with everyday demands (Braut, 2015). For the purposes of this thesis and interpretation of the results, only those aspects of health that affect mortality are really relevant. As such, except from presentations and discussions of others’ research findings, health is here understood as those aspects of physical and mental health that may impact on longevity. This is clearly an overly narrow understanding of health. But in light of the topic at hand, it makes sense to narrow the scope.

In its widest definition, health behavior can be said to encompass all human action. In practice, such a definition is of little use. Rather, it is common to define health behavior as those actions and habits that most affect disease risk (Nylenna, 2009). Of particular importance are factors such as smoking, diet, physical activity, alcohol consumption etc. In this thesis, the precise definition of what is included in this term is not crucial, since I do not have data on health behavior. In general, when I use the term, I refer to those aspects of behavior that have direct health consequences and where these consequences are typically intended, known or taken into account to some extent. For instance, people can begin to work out to improve their health, but they typically do not get married for health reasons. As such, it is sometimes useful to distinguish between health behavior and health-affecting behavior, where the latter term is given a broader meaning.

1.5 The structure of this thesis
The remainder of this thesis is organized as follows. In chapter 2, I introduce a number of theories that may inform the discussion on the relationship between retirement and mortality. I attempt to compare these theories with regard to their predictions about the direction and temporal profile of the effect. In chapter 3, I review the research literature on the relationships between retirement and health and retirement and mortality. I then return to the theoretical predictions in chapter 4, and attempt to deduce a set of testable hypotheses about the relationship between retirement and mortality. In chapter 5, I discuss possible empirical strategies that may be applied to shed some light on the hypotheses I have deduced. Chapter 6 details the data I employ, and chapter 7 explains the methods I use on these data. Chapter 8 provides descriptive statistics of the samples I have used. In chapter 9, I present the results.
from my analyses, and I discuss these results more thoroughly in chapter 10. Finally, in chapter 11, I point out what conclusions may be drawn from these analyses.
2 Theory

In this chapter, I first briefly discuss health behavior in relation to action theory. I then present different theoretical perspectives on the relationship between retirement, health and mortality, including theory about health behavior and work related risks, Michael Grossman’s health investment model, several gerontological theories about aging and retirement, and the process theory put forth by Robert C. Athcley. These theories originate from different research traditions and schools of thought, but they have all been influential in their respective fields and are frequently cited in the research literature. Also, more importantly, they differ in their predictions about the effect of retirement on mortality.

2.1 Action theory and health behavior

Before embarking on a long discussion on theories that may inform a discussion on the relationship between retirement and mortality, I should make a short note on action theory and health behavior. From an action theoretical perspective, and especially when seen through the lens of rational action theory, health behavior and mortality constitute an interesting case. Many types of behavior that directly affect health are not primarily carried out because of their health effects. For instance, one may eat a lot of sweets because of the taste or go hiking to enjoy the fresh air. In other words, the health consequences of many health-affecting behaviors could be said to be unintended (although sometimes known). In such cases, health-affecting behaviors constitute clear deviations from a strict understanding of rationality, since rationality requires intention (Elster, 2015: 235-237). In other words, one would be hard pressed to provide a plausible and purely rational and intentional explanation for much of the health related behavior observed (though, as will become evident below, Michael Grossman has attempted to do just that). A similar argument can be made with regard to mortality. Except for the case of suicide, dying is usually not intentional, but longevity may be seen to some extent to be affected by intended and unintended consequences of behavior.

Further, it would often be problematic to assume that actors have all the relevant information about the health effects of their actions. Information gathering may be difficult because information is not available (smokers in the first half of the 20th century are a relevant example) or because it is conflicting or difficult to understand. Health attitudes and beliefs

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That is, unless the actors actually weigh the marginal utility of each piece of cake against the (discounted) disutility of a slight weight gain. Some people probably do so, but hardly everyone.
have also been shown to follow a socioeconomic gradient (Wardle and Steptoe, 2003). Thus, knowledge about unintended health consequences of actions may be unequally distributed in the population.

On the other hand, a lot of health behavior intuitively appears both intentional and rational. For instance, a lot of people apparently exercise with the intention to improve their health (although for some, aesthetics may be more important). In this thesis, I do not make any strong assumptions about the motivations of individuals’ health behavior. Some people and some behaviors may be motivated by a wish to maximize utility, while norms, emotions or habits may be more important to others. Motivations may also vary between situations. I do, however, attempt to discuss some of the theoretical contributions presented below with regard to their action-theoretical underpinnings.

2.2 Health behavior and work related risks as determinants for health and mortality

Mortality is often used as a proxy for the general health of a population, while disability adjusted life years (DALYs) is a broader indicator of general health (Stoltenberg, 2014). According to the WHO (2009) report “Global health risks”, risk factors such as tobacco use, high blood pressure, overweight, physical inactivity, high blood glucose levels and alcohol use are among the top five risk factors for either mortality or reduced DALYs in high income countries. These risk factors are at least partly attributable to past or present health behavior.

The importance of health behavior as a determinant of health and mortality – especially for deaths due to non-communicable diseases – is widely recognized (WHO (2015)). However, there is also a need for some caution. The relationship is not a deterministic one, and a lot of variation in health and longevity appears simply to be due to bad luck or factors out of our control, such as genetics or childhood environment (Fredriksen, 2005).

In this thesis, I generally assume that the main mechanism through which retirement may affect mortality is by inducing changes in health behavior. By this I mean that retirement may affect factors such as what and how much people eat, how much they exercise, and how much alcohol and tobacco they consume, et cetera. In addition to affecting such lifestyle-

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2 A lengthy discussion of the exact physiological mechanisms through which retirement may affect mortality is interesting in its own right, but not strictly necessary for this thesis, as I do not have data on health behavior or cause of death, and therefore cannot test such mechanisms directly.
related risk factors for physical and mental health, retiring from work may have direct positive or negative health consequences, depending on the nature of the job one retires from. On the one hand, retirement can provide relief from stress and work-related health risks (Westerlund et al., 2009), but to stop working may also entail a reduction in physical and cognitive activity and social involvement.

In sum, then, the health effects of retirement can be assumed to depend on two factors and their relative strength; the characteristics of the job one retires from (including the status accompanied with the role as employee) and the life one leads as a retiree. All else equal, retiring from a stressful, physically straining or otherwise demanding or unsatisfactory job may yield health benefits, while retiring from a rewarding job, a job that provides structure, physical activity, cognitive stimuli and/or a social network of colleagues may have adverse health consequences (see for instance Bonsang et al., 2012). Further, the potential effect of retirement could depend on the behavior or lifestyle of the retiree. These could include physical activity, diet, alcohol and cigarette use etc. (Syse et al., 2015), but also housing and other relevant factors. Such factors could further be constrained or otherwise influenced by factors such as the pensions received, financial assets, marital status and family networks, baseline health, healthcare availability and so on (see for instance Skirbekk et al. 2010 for a brief discussion).

To simplify, one can imagine that there are two types of jobs; jobs that are harmful and jobs that are beneficial to health, and that there are two types of retirement lifestyles; healthy and unhealthy. In such a perspective, moving from a harmful job to a healthy lifestyle as a retiree would be beneficial for health, and the reverse would be true with regard to retirement from a healthy job into an unhealthy lifestyle. In a sense then, retirement would function as a catalyst for the difference in “healthiness” between one’s job and spare time. This understanding resembles the time constraints in the Grossman model discussed below. As illustrated in Table 2.1, however, the effect of the two remaining possibilities is not given, as it would depend on the relative health effect of the job and the lifestyle. If one also allows for the fact that one’s lifestyle in retirement may to a large extent be a continuation of the lifestyle led during one’s working life (in accordance with the continuity theory or the activity theory discussed below), the increased spare time following retirement may lead to diverging health behavior between those with healthy and unhealthy lifestyles, respectively. In other words, there could be substantial effect heterogeneity. Given socioeconomic differences in working
conditions and health behavior, such a scenario should thus lead us to expect this heterogeneity to follow a socioeconomic gradient.

I should note that there is little reason to assume that the mortality effects of retirement are particularly pronounced compared to other factors that affect mortality. While retirement may impact on health behavior in old age, most people do eventually retire, and minor variations in retirement age are not likely to have a big impact compared to major factors such as diet, exercise, smoking et cetera. Nevertheless, by providing more time to do more of what one already does, be it jogging or smoking, retirement may act as a catalyst for established habits.

### Table 2.1: Hypothetical health effects of retiring from jobs that are harmful or beneficial to health into lifestyles that are healthy or unhealthy.

<table>
<thead>
<tr>
<th>Healthy lifestyle</th>
<th>Beneficial job</th>
<th>Harmful job</th>
</tr>
</thead>
<tbody>
<tr>
<td>Healthy lifestyle</td>
<td>?</td>
<td>+</td>
</tr>
<tr>
<td>Unhealthy lifestyle</td>
<td>-</td>
<td>?</td>
</tr>
</tbody>
</table>

#### 2.2.1 Socioeconomic differentials in health behavior and work related risks

An extensive research literature has documented large socioeconomic differences in health and mortality and clear socioeconomic differentials in health behavior such as tobacco and alcohol consumption, diet, physical activity and so on (see for instance Eikemo et al., 2014; Næss et al., 2007; Pampel et al., 2010; SCB, 2016; Williams and Collins, 1995). Although some of the socioeconomic gradient in health outcomes is explained by observed differences in health behavior, most of it is not (Elstad, 2010; Lantz et al., 1998; Pampel et al., 2010), and although health behavior is unlikely to be perfectly measured, some of the remaining association between health or mortality and socioeconomic status (SES) may be attributable to other mediating mechanisms such as working conditions, but also possibly confounded by genetics.

In a very interesting and interdisciplinary discussion on possible mechanisms that may produce the observed SES-differentials in health behaviors, Pampel et al. (2010) draw on numerous theories in sociology, economics and public health. The nine clusters of theories considered span from Gary Becker’s rational addiction model and differences in access to information, to childhood socialization, Bourdieusian class distinction, peer influence and neighborhood effects. Genetic inheritance is not discussed in the paper. The authors conclude
that researchers in the field should improve their research designs to better be able to distinguish and contrast the different mechanisms possibly at work in producing SES-gradients in health behavior.

Retirement may affect health through several mechanisms, and the effects may depend on socioeconomic factors. Such socioeconomic conditioning of the effects of retirement may work through for instance socioeconomic differences in health behavior, family situations, economic resources or working conditions. Thus, among other things, the health and mortality effects of retirement should be expected to depend on the job one retires from and the lifestyle one retires to. And the health relevant aspects of jobs and lifestyles should be expected to follow socioeconomic gradients or divides.

2.3 Grossman’s microeconomic health capital framework

An important strand of theory regarding health behavior and mortality revolves around Michael Grossman’s (1972) concept of health capital and his health investment model. As a health economist working in the microeconomic tradition established by Gary Becker, Grossman developed the concept as part of his work with building an economic model of health behavior and investments in personal health.

2.3.1 Health capital and the health investment model

The main tenets of the health capital theory are, first, that health can be viewed as a stock that depreciates over time, until it reaches zero and death occurs. The rate of depreciation increases with age, at least after some threshold age. Second, people produce health, and may invest in their own health through inputs such as exercise, diet and other aspects of their lifestyle, use of their own time, and medical care, housing, and other market goods. Further, health is more efficiently produced by people with more education. Through this production of health, which increases the stock, people to some extent choose their own life span through rational investment behavior. This second tenet was seen by some as a great advancement in the field of health economics. It shifted the understanding of health economic theory from seeing health as something that was exogenously determined (given) to seeing health as an endogenous variable (resulting from individual actions and behaviors) (Fuchs, 1972). Third, consumers’ demand for health is driven by two factors. On the one hand, health is considered a consumption commodity, meaning that people, all else equal, prefer to be healthy – good health increases utility. On the other hand, health is an investment commodity which returns
healthy days available for market or nonmarket activity, which again yields monetary or other gains. Lastly, Grossman’s theory implies that the demand for medical care or health care services is a derived demand, as people primarily demand good health rather than medical care. Consequently, medical care is seen as an input in the production of health, not a consumption good in itself.

The health capital concept forms part of a fairly comprehensive mathematical model which attempts to describe rational decision-making in relation to one’s health behavior. However, given the proliferation of different capital-concepts within sociology (Lyngstad, 2009), I will mainly refer to investments in “health” rather than in health capital from here on. This distinction is merely semantic. Further, I should specify that in the Grossman model, investments in health are assumed to encompass intentional changes in health behavior, the use of medical services, and other aspects of one’s life affecting health and where health considerations impact on decisions. Health aspects relating to work and the work environment are not explicitly part of the investments in Grossman’s theory.

2.3.2 Strengths and weaknesses of the Grossman model

In its original conception, Grossman’s model clearly has several weaknesses, some of which he points out himself. Many relate to the simplifying assumptions of the model. One example is the assumption that actors have full information, and that they thus know with certainty at which age they will die. Such assumptions are obviously erroneous, and Brown and Vickerstaff (2011) point out that people tend to be overly pessimistic with regard to their own health and remaining life span – perhaps a case of biased beliefs (Rydgren, 2009: 73).

The action-theoretical basis of the model may also be subject to considerable (and predictable) theoretical criticism from different perspectives, as it stands firmly in the theoretical framework of the much-debated homo economicus. In the model, actors are assumed to behave in the ways predicted by intricate calculations of costs and benefits, even with regard to their lifestyle and health behavior. This view of rationality is akin to what Elster (2015: 255) defines as hyperrationality – “[...] the propensity to search for the abstractly optimal decision, that is, the decision that would be optimal if we were to ignore the costs of the decision-making process itself”. In this case, these costs could be said to approximately amount to the direct costs and opportunity costs of attaining a degree in economics so that one could learn to do the necessary calculations, or at least hire someone to do them at every decision point. The point made above should also be repeated here; if the
health consequences of much health affecting behavior are unintended, they can hardly be said to be rational, simply because rationality implies intentionality.

Further, the amount of evidence showing that people tend to not behave purely rationally, even when faced with relatively simple choices, is substantial (see Elster 2015: 256-261 for examples – some of which are of an anecdotal kind). Such observations cast serious doubts on the truthfulness, if not the utility, of a strict or narrow rationality paradigm. Importantly, many aspects of health behavior are hard to conceive of as motivated purely by instrumentally rational considerations; for instance, the sudden rush to the gyms in January appears to be driven more by norms or feelings of guilt than by utility calculations. This is not to say that rational explanation should be abandoned in general – the baby should not be thrown out with the bathwater – rather, It might be beneficial to loosen some of the more restrictive assumptions to allow for the many and obvious deviations from a strict understanding of rationality (Boudon, 2003). In risk of making a programmatic statement; the more pragmatic desires, beliefs and opportunities (DBO) model (Hedström, 2005: 37-66) could serve as an alternative and more intuitively plausible model of action. That is, if one is willing to trade formalism and predictive precision for realism.

However, keeping the shortcomings of Grossman’s model in mind, the fact still remains that although “all models are wrong, some are useful” (Box and Draper, 1987: 424), and in my view, Grossman’s general approach to health behavior does indeed give us some very useful conceptual and analytical tools, as it can be used to specify some of the mechanisms through which retirement may affect health and mortality. It also serves as a prominent and influential example of a strand of theorizing whereby retirement is seen as an event or transition.

### 2.3.3 Predictions from the Grossman model

The model developed by Grossman (1972) postulates that the net investment in the stock of health in the $i^{th}$ period (i.e. the change in health stock in the period spanning from time $= i$ to time $= i+1$) is equal to the gross investment in health, minus the depreciation that occurs in this time interval:

$$H_{i+1} - H_i = I_i - \delta_i H_i$$

where $H$ denotes the stock of health, $I$ is gross investment and $\delta$ is the depreciation rate, which varies with age. $i$ is an index for age. The depreciation rate is assumed to be exogenous. In the model, people are assumed to derive utility form their stock of health and from consuming
other commodities \((Z_i)\). As shown below, both health and the aggregate commodity \(Z_i\) are produced by the individual using time, human capital and goods and services purchased in the market. Informally put, people may use their time and money on either health investments or everything else. Both health and other things yield some utility. But health is also an investment commodity, as fewer sick days mean that the actor can spend more time working and earning money.

Applying Grossman’s framework to the study of the impact of retirement on health and mortality, several important mechanisms through which retirement may affect health become apparent. Full retirement (in the sense of quitting work completely and start receiving a moderate monthly pension) would affect several of the components of the model that impact on incentives or constraints that influence health decisions. Specifically, the model reveals several contradictory mechanisms through which retirement may affect health. I will not present or make use of the entire model here, since such an elaboration is unnecessary for the purposes of this thesis. Instead, I will attempt to present each of these mechanisms in a brief and semi-formal way.

The available time-mechanism

In the model, the total available time \((\Omega_i)\) is divided between hours of work \((TW_i)\), time lost due to illness \((TL_i)\), time spent investing in health \((TH_i)\) and time spent on other stuff \((T_i)\):

\[
\Omega_i = TW_i + TL_i + TH_i + T_i
\]

Time spent on health investments \((TH_i)\) is an important input in the production of health investments. Gross investments in health are produced according to a household production function:

\[
I_i = I_i(M_i, TH_i; E_i)
\]

where \(M\) is medical care (or other health inputs purchased in the market), \(TH\) is the time input in health investments, while \(E\) is a stock of human capital (measured by education). It follows that retirement increases the time available for time-demanding
(i.e. lifestyle-related) investments in health. All else equal, and if preferences for time use are not altered, this should lead to increased health investments.

**The consumption commodity mechanism**

In the model, the opportunity cost of spending one unit of time on health investments is equal to the wage rate \( W_i \) times one unit of time spent on work. Since retirement means that wages (pensions) are no longer related to hours worked, this relationship is replaced by a fixed wage that does not depend on working hours. Retirement thus removes income loss as an opportunity cost of spending time on investments in own health, and reduces the opportunity cost to alternative uses of one’s time. This reduced opportunity cost of health investment should in isolation lead to a greater demand for health as a consumption commodity, and thus increase investments in health.

**The investment commodity mechanism**

Retirement removes the incentive to invest in health as an investment commodity. Since the time available for market work is constrained according to the equation above, it follows that the monetary output from working is given by

\[
W_i TW_i = W_i \Omega_i - W_i TL_i - W_i TH_i - W_i T_i
\]

In other words, there is an incentive to maintain good health in order to keep the time lost to illness \( TL_i \) as low as possible, so that one can work more and earn more money. Now, retirement removes the connection between hours worked and money received (the actor receives a pension without working), reducing the opportunity cost of sick days to the alternative uses of one’s time. This removes the incentive to invest in health as an investment commodity for the purpose of harvesting the monetary returns of being healthy enough to work. This should lead to a reduced investment in

---

3 An important side note is that more available time would also increase time for consumption \( T_i \) of other things \( Z_i \), as a second household production function determines the production of all other commodities \( Z_i \) that enter into the utility function:

\[
Z_i = Z_i(X_i, T_i; E_i)
\]

where \( X \) denotes the input of goods in the production of the aggregate commodity \( Z \), and \( T \) is the time input in the production of these commodities. \( Z_i \) may in one sense include TV shows, ice cream, cigarettes, booze and other consumption goods of which consumption in real life may have unintended health consequences. On the other hand, unintended health consequences of different consumption patterns are not part of the Grossman model in its original conception, so it is not clear where such goods should be included.
health. Due to the relatively generous sickness benefits in Norway, this mechanism is perhaps unlikely to be very strong, though.

**The income mechanism**

As mentioned, in the Grossman model, each individual has perfect foresight and is assumed to know the present value of all future earnings, and should be able to save for retirement so that retiring does not affect the money available for market purchases. Disregarding this somewhat dubious assumption, it can be assumed (at least equally improbably) that retirement is some exogenous event that the individual does not prepare for (or at least imperfectly prepares for). The loss of income related to retirement may then affect health through the ability to purchase medical services and other health related goods \((M_i)\) in the market. The purchase of such “medical services” and goods used in the production of the aggregate commodity \(Z_i (X_i)\) are constrained by a goods budget constraint where the present value of these goods equal the present value of income over the life cycle and initial assets:

\[
\sum P_i M_i + V_i X_i \frac{(1 + r)^i}{(1 + r)^i} = \sum W_i T W_i + A_0
\]

where \(P_i\) and \(V_i\) are the prices of \(M_i\) and \(X_i\), respectively, \(r\) is the interest rate, \(W_i\) is the wage rate, \(T W_i\) is the time spent working and \(A_0\) denotes initial assets.

If we assume that pensions \((PE_i)\) are paid as a fixed amount, that \(PE_i < W_i T W_i\), and that retirement does not shift the relative spending in favor of “medical services” and health investments, retirement should lead to a reduction in health investments through reduced income. The fact that medical services are relatively low-cost (or in some cases free) in Norway may attenuate the effect of this mechanism.

In sum, Grossman’s model does not tell us if retirement should increase or reduce mortality risk. In fact, the original model does not cover retirement explicitly, but as I have shown, the effects of retirement can be deduced from the general framework. In such an application of the model, retiring is understood as an event or transition, whereby the individual moves from one state with one set of opportunities and constraints (wages, available time etc.) to another state with another set of opportunities and constraints. As I have shown, the model then points toward some important mechanisms that may affect the mortality risk in different directions, and it tells us that the net effect depends on the relative strength of these mechanisms. In other
words, according to the Grossman model, the net effect of retirement on mortality can be either positive, negative or zero (although, in the Norwegian context, it may lean more towards the positive side than in some other contexts, because of the generous sickness benefits and affordable healthcare). Also, the model can be said to allow for effect heterogeneity in the sense that different subgroups may respond differently to changing opportunities and incentives following retirement. A zero net effect may in such a case mask substantial but diverging effects between subgroups. Finally, it should be noted that according to the Grossman model, a lower stock of health at the point of retirement would be associated with a shorter life span regardless of the net health effects of retirement. This would not, however, be an effect of retirement per se, but would, to the extent that a low health stock induces retirement, serve as a mechanism for negative health selection into retirement.

2.4 Gerontological theories about aging

Several theoretical perspectives from the gerontological research literature may also inform the discussion about the effects of retirement on health and mortality. Most of these give similar predictions about the direction of the effect. Some of them give predictions in line with the Grossman model, but differ from it with regard to which mechanisms are in play. Several of these theories also have similar theoretical underpinnings, namely the emphasis on identity, roles, role expectations and role loss. In this regard they bear some resemblance to Atchley’s process theory discussed below. Readers interested in the corpus of relevant social gerontological theories are referred to Daatland and Solem (2000).

2.4.1 Activity theory and continuity theory

The activity theory of aging is not a theory about how people age, but about the prerequisites for successful aging. It states that staying social and maintaining one’s activities and attitudes from middle age enhances quality of life and delays the aging process (Havighurst, 2009 [1963]). Inherent in this theory is the view that people should avoid major life course transitions in their later life, and that retirement is a potentially harmful transition unless the stimulating activities people engage in as employees are somehow substituted by other activities in retirement (Ekerdt, 1987). In one sense, then, the theory predicts that retirement should have a harmful effect on health, if any. This prediction is in line with what would be expected if the mechanisms that harm health in the Grossman model dominate. However, this
theory places emphasis on direct health benefits of working rather than seeing work and
health behavior as separate.

Whereas the activity theory may be seen as having some normative implications (or at least
strong recommendations) about how people should age, the closely related continuity theory
attempts to describe how people do age. It states that people tend to aim for, produce and
enjoy continuity in their life with regard to both psychological characteristics, perceptions of
self and social behavior. Continuity is seen as an adaptive strategy. But continuity is not
understood as static sameness, but dynamic consistency and coherence with the past (Atchley,
1989). As in activity theory, discontinuity is seen as a potential threat to mental health, but it
is not clear that retirement should be expected to induce such discontinuities; “Just because
logic suggests that there ought to be an identity crisis connected with retirement, it does not
follow that there will usually be one. Indeed, researchers have indicated that identity crises
connected with retirement are relatively rare” (Atchley, 1989: 187). In other words,
continuity theory predicts that retirement may be harmful to mental (and perhaps also
physical) health if it marks a sharp discontinuity in self-perception or identity, but it leans
heavily towards this not being the case. This prediction of a potential negative health effect of
retirement may be thought of as lasting or relatively short-term. If it is lasting, the prediction
could bear resemblance to one of the possible predictions of the Grossman model and to
disengagement theory. If it is short lived, the theory may be more closely related to the
stressful life event perspective discussed below.

2.4.2 Disengagement theory

For the present purposes, the most important postulate of the disengagement theory is that
disengagement from one’s central role in society tends to lead to crisis and demoralization4.
Within the framework of this theory, which was first formulated by Elaine Cumming and
William Earl Henry in the early 1960s, men’s central role is seen as that of a worker

4 Disengagement theory postulates many things. The summary of the theory consists of nine (or ten) postulates
with ten corollaries.

providing for the family (“instrumental”) and women’s role is understood as that of a wife and
homemaker reducing tension within the family (“socioemotional”). Disregarding this
somewhat outdated gender distinction, the disengagement theory predicts that retirement from
work is an unavoidable step in the disengagement process. As I interpret the theory, this
disengagement will have adverse mental health consequences (and possibly also adverse
physical health outcomes) unless the lost role is substituted (Cumming and Henry, 1961: 210-
218). In other words, the loss of the worker role and the social network, status etc. associated with this role can be understood as a necessary stage in the disengagement process, but nonetheless a stage that should serve to accelerate the mental and physical decline associated with old age. The health trajectories of retirees should show a steeper decline than those of workers as a consequence of the retirement disengagement. Although the retirement disengagement is seen as a part of the general process of disengagement related to aging, the retirement disengagement itself should be seen as an event or transition with potentially adverse health consequences, similar to the Grossman model, activity theory and continuity theory.

It should also be noted that the disengagement theory has been subject to substantial criticism. This criticism has in turn been very neatly summed up and systematized by Arlie Hochschild (1975). Essentially the theory is criticized for being unfalsifiable, for the use of all-inclusive variables or catchall categories to describe variations in forms of disengagement, and for assuming (or imputing meaning and interpretations into) actors’ perception without empirical support; “if conscious reflections were fully taken into account and given weight, I think the authors would find themselves with evidence which refutes the theory, were it posed as a refutable theory” (Hochschild, 1975: 560).

2.4.3 Retirement as a stressful life event

The stressful life event perspective holds that the detachment from gainful employment and the transition into retirement entails a loss of social networks, identity, income and other benefits associated with employment. Essentially, the theory emphasizes the loss of employment, rather than the acquisition of the retirement role. The general idea is that the transition into retirement is a large-scale life change that induces stress and therefore increases susceptibility to illness (Daatland and Solem, 2000:150; Minkler, 1981). In this regard it has much in common with the activity theory (Ekerdt, 1987), but also with some of the other theories discussed in this chapter. The degree to which retirement could be found to work as a stress inducer may depend on the degree of control over- and on the timing of the retirement event and on the point in the retirement process the stress level is measured (Minkler, 1981), but there could be considerable heterogeneity in the extent to which retirement induces stress. Although compatible with a process perspective in the sense that the stress period may be relatively short (Minkler, 1981), I would argue that the view of retirement as a stressful life event
event should on its own be regarded as an event or transition perspective which, similarly to
the Grossman model, predicts a one-directional (negative) effect on health.

2.4.4 The no effect assumption
Both the “stressful life event” perspective and the activity theory have been criticized by
sociologist David J. Ekerdt (1987) as examples of a priori reasoning based on the erroneous
assumption that retirement must have some effect on health:

*Although the research literature contains virtually nothing to encourage the notion
that retirement is likely to harm the physical health of older workers, the idea persists
that retirement increases the risk of illness and death.* (Ekerdt, 1987: 455)

In somewhat strong words, he has argued that the notion that retirement harms health is based
on anecdotes, fallacies, wishful thinking, a priori reasoning and misinterpretations of research
findings. This criticism can easily be extended to the other theories discussed in this chapter,
although they are not explicitly mentioned. In a sense, Ekerdt is arguing in favor of the null
hypothesis, namely that retirement does not impact on health or mortality.

Although not a theory in a strict sense, I include this assumption among the other theories
because it has formed an underlying assumption through most of the work I have done on this
thesis. To elaborate on this point, it is worth noting (as will become clear in the literature
review below) that neither of the theories discussed in this chapter, as applied to retirement
and mortality, have a well-established explanandum (what needs to be explained). There is no
well-established fact or causal relationship in need of an explanation; there is no consensus on
the health and mortality effects of retirement – at least there was no consensus at the point in
time when the different theories were developed. These theories have mostly been employed
by researchers to predict a hypothesized phenomenon (“retirement is harmful”), not as
explanations for an observed one (“how do we explain the observation that retirement
increases mortality risk?”). This should perhaps not be considered in line with ideals about
explanatory theorizing (Elster, 2015: 3-10), but as will be seen from the literature review
below, the lack of an explanandum is hardly surprising given the divergence in research
findings.
2.5 Atchley’s process approach

In his book entitled *The Sociology of Retirement*, Robert C. Atchley (1976) outlines a process perspective on retirement. This theory is given special attention here as it can be taken to predict an effect of retirement on mortality that varies with time in a way that sets it apart from other theories in the field. According to this view, retirement should not be seen just as a single event or change of state. Rather, retirement should be viewed as a process in which the (prospective) retiree passes through a series of phases both before and after the transition to retirement, as illustrated in Figure 2.2, and takes on the social role of retiree through this process. In other words, this theory does not assume that people mechanically (or rationally) respond to changes in opportunities and constraints relating to retirement. Rather, retirement is a process of social role change and adaption that has a psychological effect on the individual.

**Figure 2.2: Phases of retirement (Atchley, X1976: 64).**

<table>
<thead>
<tr>
<th>Remote phase</th>
<th>Near phase</th>
<th>Honeymoon phase</th>
<th>Disenchantment phase</th>
<th>Reorientation phase</th>
<th>Stability phase</th>
<th>Termination phase</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preretirement</td>
<td>Retirement</td>
<td>Retirement event</td>
<td>End of retirement role</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

2.5.1 The phases of retirement

Although interesting in its own right, I will not draw on the entirety of Atchley’s process theory in this thesis. Rather, I will pay special attention to the two phases that may affect health in the period following retirement, i.e. the honeymoon phase and the disenchantment phase.

The first two of Atchley’s retirement phases are labeled the remote phase and the near phase of preretirement. The remote phase may begin a very long time before the actual retirement transition, for instance at or before the start of an occupational career, and it would tend to continue until one realizes that retirement age is nearing. During the remote phase, the thought of retirement is a distant one. The near phase sets in when the aging employee realizes that retirement is drawing near. It is often characterized by more negative sentiments towards retirement, as people may be struck by the realities of facing a sharp reduction in income, or realize that they lack the “leisure skills” necessary for a pleasant and meaningful retirement. The person nearing retirement will also build up expectations or fantasies about
the upcoming life as a retiree, which may serve to smooth the transition if they are realistic, but may make the transition more difficult if the expectations are unrealistically idyllic.

After the actual retirement event (assuming that there is such an event and not just a gradual detachment from work), Atchley’s theory states that the retiree enters a honeymoon phase – a euphoric period where the retiree enjoys his or her newfound freedom and spare time, attempting to do all the things he or she did not have time to do while still working, including leisure activities, physical activities, travelling and spending time with grandchildren. According to Atchley (1976: 68), “[t]he person in the honeymoon period of retirement is often like a child in a room full of new toys”. In a Norwegian context one may imagine (in line with common stereotypes) that recent retirees spend more time outdoors, hiking, skiing, fishing, gardening et cetera, compared to what they would have done if they had still been working.

In relation to health behavior, the honeymoon phase may induce a more healthy and active lifestyle, but for some, the honeymoon phase may also have some adverse health consequences if it for instance involves an increase in alcohol and/or tobacco consumption). The net effect, however, is likely to be positive. The honeymoon is not for everyone, though, and the lifestyle options may be limited or the joy of freedom thwarted by a lack of money, poor health, weak or missing social or family bonds et cetera. In other words, some may skip this phase altogether, and those who are subject to involuntary retirement could be especially prone to never experience the bliss of retirement. The fast-paced and hectic period of activity following retirement soon enough turns into a routine as the honeymoon comes to an end. Depending on the nature of this routine people may either adjust fairly quickly to the new role of being a retiree, or they may enter the fourth phase of the retirement process.

This fourth phase of retirement is aptly labeled the disenchantment phase, and may result from problems adjusting to the life as a retiree. The idea is that the aforementioned expectations about retirement may be unrealistic, leading to disappointment, disenchantment and/or depression for retirees when they are faced with the realities of retirement. In essence, people may get bored, experience a lack of meaning or anomie, or simply have trouble structuring their lives when they no longer have a job or a social network related to work. According to Atchley, factors such as having less money, poor health, problems with managing one’s own life, loss of previous social roles, a formerly strong attachment to work or moving away from one’s community may worsen the backlash from the honeymoon phase
and lead to deep depressions. With regard to health behavior, a disenchantment phase can therefore be assumed to result in both physical and mental health deterioration.

The fifth and sixth phases of retirement are referred to as the **reorientation phase** and the **stability phase**, respectively. The reorientation phase is the process of reemerging from the lows of the disenchantment phase and finding new meaning in the role of retiree. Expectations are adjusted and new arenas of involvement are discovered, for instance at senior centers. For most people, the reorientation phase leads to stability in the sense that the retiree reconciles with the new role and get into a routine and a lifestyle that, at least to some extent, is experienced as meaningful and satisfying. The last phase of retirement is termed the **termination phase**. Despite the somewhat morbid label, this phase does not necessarily involve dying. Rather, this phase marks the transition from the role of retiree. Usually this happens because increased frailty, illness and disability forces one to abandon the role of retiree to take on the role of sick or disabled. But low income resulting from a loss of pension benefits may also cause a transition into a role as economically dependent.

### 2.5.2 Strengths, weaknesses and interpretations of Atchley's theory

The process approach outlined by Atchley is not strictly a theory about retirement and health, but a theory about retirement that has implications for health. Most importantly, it implies that retirement may have consequences for health that vary over time. If one assumes that the honeymoon phase is beneficial for health and that the disenchantment phase is detrimental for health, it could follow that the mortality effects of retirement are not monotonic but time-variant, in the sense that, all else equal, the mortality risk of retirees relative to those who continue working may be reduced in one phase and increase in another. Such an interpretation has been stated or implied repeatedly in the literature (Ekerdt, 1987; Minkler, 1981; Solem, 1987)\(^5\), and as I will show below, there is some evidence to support it. I will return to the mortality implications of the process perspective below, but first, it is necessary to address an important theoretical issue in the process perspective.

The process theory has a theoretical black box; why do people transition into the disenchantment phase? It appears highly plausible that people who voluntarily retire for reasons not related to health will enter into a honeymoon phase. For voluntary retirees, retirement is a fulfillment of their desires, which will obviously make them happy. It will also give them more time to pursue their interests in areas other than work, which should also

\(^5\) The reference to Atchley’s process theory is made only implicitly by Ekerdt.
make them happy, and make them healthier, given that these interests are beneficial to their health. However, it is difficult to see the logical reason for why a large share of retirees would then simply become disenchanted, unhappy or depressed some time after a very happy retirement. What is the psychological mechanism that could cause such a pattern?

The *expectations for retirement* that are built up during the preretirement period would appear to be the key to a plausible explanation of such a pattern. Atchley (1976: 69) in fact highlights unrealistically high expectations (or fantasies) and subsequent disappointment as important triggers for the honeymoon and disenchantment phase, respectively:

*In a sense, the honeymoon period represents a living out of the preretirement fantasy. The more unrealistic the preretirement fantasy turns out to have been, the more likely it is the retiree [sic] will experience a feeling of emptiness and disenchantment*

Put differently, people become happy in the honeymoon phase because they have high expectations and are able to live them out, but they become sad and enter the disenchantment phase when they realize that their expectations were too high. Expectations in this context can be understood as a combination of beliefs and desires, perhaps constituting a case of wishful thinking (Elster, 2015: 125-8), i.e. beliefs formed by desires, with the accompanying dissonance between beliefs and reality. So, what are the problems with this interpretation of the theory?

First, one could ask why people would develop unrealistically high expectations about retirement in the near phase of preretirement when, according to Atchley (1976: 66), “[a]ttitudes toward retirement generally become more negative during the near retirement phase […]”. An answer to this could be that *most* people may remain positive to retirement, or that ”more negative” may still mean that people have positive sentiments toward retirement, only less so than they did before.

Second, one could ask how people can become happy by living out their expectations in the honeymoon phase if these expectations are unrealistically high. Would this not make them impossible to live out in the first place, leading to immediate disappointment? An answer to this might be that the expectations are not unrealistic to fulfill, but unrealistic to maintain. People cannot maintain a high activity level and euphoric state of mind for long periods of time, and some regression to the mean should be expected. This reality leads to disenchantment. Well, then one may ask why people do not just adapt their expectations (see
for instance Elster, 2015: 112) and move directly into the reorientation phase, rather than go through the disenchantment phase first. Such a direct adjustment would be in line with the psychological set point theory (or the “hedonic treadmill”) which tells us that the effects of positive or negative experiences tend to be short-lived and that people quickly return to a set point of happiness (see for instance Diener et al., 2006). An answer in accordance with Atchley’s theory could be that people need time to adjust their expectations to the realities facing them. The disappointment that comes with not being able to maintain their expected lifestyle and level of happiness over time may in itself be enough to induce depression or disenchantment, perhaps reinforced by the lack of a job and daily routine to serve as a distraction. This could prevent a smooth transition from the honeymoon phase. Essentially, this would be equivalent to saying that when people realize that they cannot be really happy but have to settle for moderately happy, they become really sad. Although perhaps not convincing, this explanation is at least plausible.

Lastly, Atchley’s theory assumes that people have systematically faulty expectations about retirement. For the expectation/disappointment mechanism to be effective, people need to have expectations that are systematically biased towards retirement as an unrealistically positive role or state (see Rydgren, 2009: 73 for a discussion on biased beliefs), and this must be the case for each new cohort of retirees. In other words, the experiences of older retirees could not serve to correct these expectations, either because they are not communicated, or because they are ignored. If one is to accept the expectation/disappointment mechanism of the process theory, one also has to accept the premise of biased expectations. Although I am not convinced that this premise is true, I personally see it as plausible. In any case, the hypothesis that retirement is followed by a honeymoon phase and a disenchantment phase that both affect mortality is one worth testing.

2.6 Comparing the predictions of the theories
The theories discussed above differ in several ways. One is the action theoretical assumptions that underpin them. But more relevant for this thesis are the different predictions they make about the direction and temporality of the effect of retirement on mortality. These are summarized in Table 2.3.

Theories about health behavior and work related health risks are ambiguous about the direction of the effect. While retiring from harmful jobs into healthy retirement lifestyles
would reduce mortality, retiring from beneficial jobs into unhealthy lifestyles would have the opposite effect. The predicted effect of retiring is also ambiguous in the Grossman model, and may depend on the relative strength of different mechanisms. If the available time mechanism and/or consumption commodity mechanism dominates, retirement can be assumed to reduce mortality, but the reverse would hold if the investment commodity mechanism and/or income mechanism dominates. Both the activity theory, the disengagement theory and the stressful life perspective predict that retirement increases mortality. The continuity theory can also be interpreted as predicting such an effect.

With regard to temporality, the effects predicted by the Grossman model and the disengagement theory can be assumed to be monotonic, meaning that retirement should increase the age-specific mortality of retirees for all subsequent time intervals, relative to workers. The stressful life event perspective could, on the other hand, be interpreted to predict a relatively short-term effect on age-specific mortality. It is not clear whether the continuity theory, the activity theory or the theories about health behavior and work related health risks predict a short-term or a long term, monotonic effect.

Both with regard to direction and temporality, Atchley’s process theory and the no effect assumption stand out. While the no effect assumption predicts that retirement has no effect on mortality, the process theory (or at least a common interpretation of it) predicts a time-varying effect. Specifically, it predicts that age-specific mortality should first be lower in the period following retirement, and then be higher, relative to those not retired. In the following chapter, I will briefly review the research literature on retirement, health and mortality, before returning to these theoretical predictions in chapter 4.
Table 2.3: Predictions from different theoretical perspectives on the effect of retirement on mortality.

<table>
<thead>
<tr>
<th>Theory/model</th>
<th>Predicted retirement effect on relative mortality risk</th>
<th>Mortality risk following retirement</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>Harmful job X healthy lifestyle</td>
<td>Reduces mortality risk</td>
<td>Monotonic or short-term</td>
<td>Grossman (1972)</td>
</tr>
<tr>
<td>Healthy job X unhealthy lifestyle</td>
<td>Increases mortality risk</td>
<td>Monotonic or short-term</td>
<td>Grossman (1972)</td>
</tr>
<tr>
<td>Grossman model</td>
<td><em>Depends on relative strength of mechanisms</em></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Available time mechanism and/or consumption commodity mechanism dominates</td>
<td>Reduces mortality risk</td>
<td>Monotonic</td>
<td></td>
</tr>
<tr>
<td>Investment commodity mechanism and/or income mechanism dominates</td>
<td>Increases mortality risk</td>
<td>Monotonic</td>
<td></td>
</tr>
<tr>
<td>Continuity theory</td>
<td>Likely no effect. Increases mortality risk if any</td>
<td>Monotonic or short-term</td>
<td>Atchley (1989)</td>
</tr>
<tr>
<td>Activity theory</td>
<td>Increases mortality risk</td>
<td>Monotonic or short-term</td>
<td>Havighurst (1963)</td>
</tr>
<tr>
<td>Disengagement theory</td>
<td>Increases mortality risk</td>
<td>Monotonic</td>
<td>Cumming and Henry (1961)</td>
</tr>
<tr>
<td>Stressful life event</td>
<td>Increases mortality risk</td>
<td>Short-term</td>
<td>Minkler (1981)</td>
</tr>
<tr>
<td>No effect-assumption</td>
<td>No effect</td>
<td>No effect</td>
<td>Ekerdt (1987)</td>
</tr>
<tr>
<td>Process theory</td>
<td>Varies with time</td>
<td>First lowered, then heightened</td>
<td>Atchley (1976)</td>
</tr>
</tbody>
</table>
3 Empirical background

The contention that retirement may have an adverse effect on health has become increasingly popular with the recent categorization of this phenomenon as a stressful "life event." The small number of empirical studies examining the health outcomes of retirement, however, appear neither to support nor to refute this hypothesis. Moreover, the serious methodological problems inherent in most of these studies caution against the generalization of findings. (Minkler, 1981: 117)

This observation was made by Meredith Minkler some 35 years ago. And, regrettably, it appears to still be valid – at least up until very recently – despite the growing number of studies on the relationship between retirement and health. In this chapter, I review the literature on the relationship between retirement, health and mortality. I pay special attention to recent studies that have attempted to circumvent the selection issues and biases that tend to haunt studies in this area and that can thereby make strong claims for causal inference. The studies reviewed here will later inform the formulation of the hypotheses in this thesis.

3.1 Descriptive studies

3.1.1 Association between retirement and health

The research literature abounds with studies investigating the relationship between retirement and health. I will not attempt a thorough review of the entire literature, as this would be very demanding, and because it is not directly relevant for the present paper, since I study mortality rather than health. Rather, I will first briefly discuss a recent literature review published on the topic and subsequently discuss a few research articles published after this review was conducted.

van der Heide et al. (2013) have conducted a systematic review of longitudinal studies investigating the relationship between retirement and health. The researchers found retirement to have a beneficial “effect” on mental health, but found that the results with regard to perceived or self-rated general health, and different measures of physical health were contradictory. The observed association between retirement and relative mental health improvements runs contrary to the disengagement theory and the stressful life event perspective discussed above, and may also be at odds with activity theory and continuity theory (depending on one’s interpretation of these theories). It may, however, be consistent
with a post-retirement “honeymoon” as envisioned by Atchley (1976) or with increased health investments (Grossman, 1972). The review suggests that the contradictory findings may be due to differences in quality, research design, follow-up-periods, context or other factors.

The review by van der Heide et al. (2013) can be said to suffer from a serious weakness; it places considerable emphasis on general quality criteria of the papers reviewed, but it does not consider the internal validity of the studies with regard to claims about causal inference concerning the relationship between retirement and health. This is despite the fact that it strongly implies that the examined associations reflect causal relationships – a claim that could be considered dubious in light of the selection issues discussed below.

A few studies not included in the abovementioned literature review may shed additional light on the retirement and health relationship. First, Stenholm et al. (2014) found physical functioning to decline faster among retirees than among older employees in the US. Second, Coppola and Spizzichino (2014) found retiring to be associated with higher risk of reporting worsened health among men, but not among women in Italy. They suggested that this gender difference reflected men’s stronger involvement in the labor market compared to women, and that retirement was associated with a bigger risk as it involved a greater loss of role and status for men. This would be in accordance with the stressful life event perspective discussed above. Third, Syse et al. (2015) found Norwegian retirees (compared to workers) to be more likely to report mental health improvements, increased physical activity, weight loss, and changes in alcohol intake, and that they were less likely to report mental health deteriorations. In other words, these more recent studies also point in different directions with regard to the association between health and retirement.

Obviously, the association between retirement and poor health that some studies report may be due to the “healthy worker effect” (Li and Sung, 1999; Shim et al., 2013), a special case of selection bias whereby employed people are positively selected with regard to health, while people who retire are negatively selected, or some other selection process. The healthy worker effect is well documented – poor health predicts early work exits (Mein et al., 2000; Rice et al., 2011). But the reverse case that some studies report – that retirement is associated with relative health improvements – is more difficult to explain as reflecting some selection process. However, some slightly less intuitively obvious non-causal mechanisms may produce such results. The first is pointed out by Ekerdt et al. (1983). They show how retrospectively reported health improvements may be attributable to reduced or changing role demands.
related to retirement. The same point may extend to comparisons of pre- and post-retirement health reporting. If your health hampers your functioning at work, and you retire from your job, your health problems may be less acutely felt, without your health really improving. Similarly, reported health improvements may reflect a purely psychological effect; “claims for retirement's beneficial effect on health may [...] be another way of expressing enthusiasm for retirement life” (Ekerdt et al., 1983: 236). The second mechanism is of a more technical kind, and has to do with the operationalization of health changes. When using change scores (i.e. the difference between reported health status before and after some treatment – in this case retirement) as an outcome, a mathematical bias produces effect heterogeneity in the sense that a lower baseline score will be artificially correlated with a more positive change score (Hankins, 2012). Since one can assume that low baseline health scores are correlated with early retirement, this may lead to biased results. A similar biasing mechanism is the one whereby health scales are capped at the top and bottom, so that health improvements among those with good baseline health are hard to detect and vice versa.

3.1.2 Association between retirement and mortality

A brief review of the literature on retirement and mortality shows that some studies have found earlier retirement to be associated with reduced mortality (Brockmann et al., 2009; Munch and Svarer, 2005), while a number of other studies have found early retirement to be associated with increased mortality (Bamia et al., 2008; Carlsson et al., 2012; Kühntopf and Tivig, 2012; Morris et al., 1994; Quaade et al., 2002; Skirbekk et al., 2010; Waldron, 2001; Wolfe, 1983; Wolfson et al., 1993; Wu et al., 2016). Other studies have shown mixed results (Haynes et al., 1978; Litwin, 2007; Tsai et al., 2005).

Shim et al. (2013) have carried out a systematic review of the research literature on retirement as a risk factor for mortality. They conclude that the results are mixed with regard to the association between early retirement and mortality risk, but several of the studies considered compare disability pensioners with workers or do not distinguish between disability pensioners or other health-related retirees and other groups of retirees, or did not account for selection or reverse causality in their research designs. The authors attribute the conflicting results to factors such as the studies only having access to a limited set of control variables, small samples or non-representative (subgroup) samples.

The authors further conclude that there is “strong and sufficient evidence” for retirement as a risk factor for mortality. This conclusion is based on the fact that three high-quality studies
and five medium-high quality studies out of a total of thirteen found evidence in support of this. This conclusion does not, however, extend to all types of retirement considered in the review. If the studies investigating health-related retirement are excluded, the number of high- and medium-high quality studies supporting retirement as a risk factor for mortality falls to one and three, respectively. These findings highlight the importance of distinguishing between different types of pensions, as there is good reason to suspect strong negative health selection into health-related pension schemes such as disability pensions. Such a distinction is made possible by the data used in this thesis.

The review conducted by Shim et al. (2013) can also be said to suffer from the same weakness as the review by van der Heide et al. (2013); it emphasizes formal quality criteria, but does not consider the internal validity of the implied claims about causal effects of retirement on mortality. In the next sections, I will consider studies better suited for making causal claims more carefully.

In sum, the majority of the studies that investigate the association between retirement and mortality point towards early retirement being associated with increased mortality. There are several possible explanations for such a relationship. First, the relationship may be a causal one. Early retirement could increase the risk of dying, for instance through retirement-related lifestyle changes, stress, role loss, or because of some protective effect of employment. Alternatively, the relationship may reflect an instance of reverse causality if becoming fatally ill induces early retirement (Skirbekk et al., 2010). Further, the relationship may be due to a healthy worker effect or some other selection process. Similarly, some attributes, such as a preference for a sedentary lifestyle, may cause both early retirement and a shortened life span. Such an effect would constitute a case of omitted variable bias.

### 3.2 Studies designed for making causal claims

#### 3.2.1 Effect of retirement on health

Several studies have investigated the causal effects of retirement on health. Commonly, they have done this by exploiting the exogenous variation in retirement opportunities created by different types of changes in pensions systems or pension eligibility rules, or variations in pension rights that stem from arbitrary cutoff values for retirement eligibility.
Bingley and Pedersen (2011) used the introduction of an early retirement program in Denmark as an instrument for retirement age in order to estimate health effects of early retirement, and found that early retirement resulted in better health. Both Coe and Zamarro (2011) and Hessel (2016) employed European data and used country-specific retirement ages to instrument for retirement behavior. Both studies found retirement to be beneficial for self-reported health. Coe and Lindeboom (2008) studied the impact of retirement on several health measures, using early retirement windows in the US to instrument for retirement behavior. They found no negative effect of retirement on health, but some indications of a temporary, small and positive effect on self-reported health. Atalay and Barrett (2014) similarly exploited an Australian pension reform to instrument for retirement age, and found retirement to have a positive impact on both subjective and objective health measures for women. Eibich (2015) used financial incentives in the German pension system in a regression discontinuity (RD) design to study the effects of retirement on health. He found retirement to improve subjective health and mental health and to reduce the use of outpatient care. The results further showed that retirement improved health mainly through reduced work related stress, more sleep and more exercise. Both Bound and Waidmann (2007) and Johnston and Lee (2009) used the discontinuity in the probability of being retired at certain ages in England in RD designs to study the impact of retirement on subjective and objective health measures. They found retirement to improve subjective health, but retirement did not affect objective health measures in Johnston and Lee’s study. Hallberg et al. (2015) studied the effects of an early retirement offer given to military personnel in Sweden. They found this offer to reduce inpatient care – an effect that was stronger among those with lower income and low education. Bonsang et al. (2012) used eligibility age as an instrument for retirement age in a study of cognitive functioning among older Americans. They found retirement to reduce cognitive functioning around one year after retirement.

As hinted at above, the use of self-reported health measures in many of these studies may be questioned as it is not obvious that self-reported health is a valid measure of objective health. It has, however, been shown that self-rated health is a reasonably good predictor of mortality and physical health (Holseter et al., 2015; Jylhä, 2009; Lee, 2000; Miilunpalo et al., 1997).

Measurement aside, most of the studies briefly reviewed here point towards retirement actually having a positive effect on self-reported physical health. The evidence is less clear or

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6 Instrumental variables and difference-in-differences methods are described in chapters 7.4 and 7.3, respectively.
points in the opposite direction with regard to objective health measures and cognitive functioning. The fact that these studies tend to find evidence of a positive subjective health effect of retirement, while the studies reviewed by van der Heide et al. (2013) show more contradictory results illustrates the importance of adequately dealing with selection bias in studies of retirement and health-related outcomes. Further, these results could be said to lend support to the criticism put forward by Ekerdt (1987).

3.2.2 Effect of retirement on mortality

Some studies investigating the effects of retirement on mortality have also attempted to circumvent the selection bias into early retirement by using for instance early retirement windows or changes in retirement regulations as instruments for retirement behavior. As opposed to the studies using health as an outcome, however, the results from these studies may appear somewhat confusing.

In the aforementioned working paper by Bingley and Pedersen (2011), the authors also estimated the effect of early retirement on mortality before the age of 80. They found that early retirement led to reduced mortality. Similarly, Bloemen et al. (2013) found earlier retirement to reduce mortality among Dutch men. In their study of Swedish military personnel, Hallberg et al. (2015) also found an early retirement offer to reduce mortality, and the results indicated that this may have been due to a reduced risk of dying from acute myocardial infarction. Kuhn et al. (2010) similarly studied a sample of Austrian blue-collar workers. They used a change in unemployment insurance rules to instrument for retirement age. Their instrumental variables (IV) estimates, however, showed an increased risk of dying before the age of 67 among males (but not among females) as a result of early retirement. Their results also indicated that the increased mortality risk may be associated with cardiovascular disorders due to changes in health-related behavior. In their paper, Coe and Lindeboom (2008) measured mortality after four and six years in addition to their health outcomes. The results of their IV estimations of the mortality effect of retirement were close to zero and a long way from statistical significance. Similarly, a recent study on Norwegian data using the lowering of the minimum retirement age in the contractual early retirement system (AFP) both as an instrument for age of retirement and in a triple differences estimator did not find any statistically significant effect of retirement age on mortality. This result was robust despite the fact that the researchers also ran the analysis on several subgroups, including men and blue-collar workers (Hernæs et al., 2013).
So, does age of retirement affect the age at which you die? Or does retirement status affect mortality risk? In sum, the evidence for a causal relationship between retirement and mortality is rather weak and mixed. One of the studies reviewed here found an increased mortality risk due to early retirement, while other studies found early retirement to reduce mortality and yet other studies have not found any statistically significant effects of retirement on mortality. In other words, it is far from clear whether retirement age or retirement status affects mortality. This thesis attempts to provide some clarity about this issue. But the question could also be asked a bit differently. If the mortality implications of Atchley’s process perspective hold true, the relevant factor is not at what age you retire or whether you are retired, but for how long you have been retired. In such a case one would be better off asking “does the duration of retirement affect mortality?” If retirement first lowers, then increases the mortality risk, the effect would be notoriously difficult to capture. Measuring it would require both exogenously induced retirement and highly detailed information about mortality at different durations following retirement. Many studies reviewed in this section have not had access to, or have not exploited, such detailed information. This thesis tests whether retirement age or retirement status affects mortality. In addition, it adds to the research literature by using highly detailed person-month data on retirement eligibility and mortality in an attempt to put the mortality implications of the process theory to the test.

3.3 Is there evidence for the process theory?
There is some evidence to support the hypothesis that there are retirement phases that are associated with health and mortality. Such empirical support has been found with regard to psychological well-being (Kim and Moen, 2002; Richardson and Kilty, 1991), distress (Nuttman-Shwartz, 2004), life satisfaction and involvement in physical activities (Ekerdt et al., 1985), and mortality (Solem, 1987). Westerlund et al. (2009) and Martin and Doran (1966) also found evidence of a sharp reduction in poor health and illness around the time of retirement, followed by a gradual increase, perhaps consistent with a honeymoon effect. On the other hand, Van Solinge and Henkens (2008) found no support for such phases with regard to well-being, and both Price (2003) and Nuttman-Shwartz (2004) have emphasized the variability of the retirement experience. The results mentioned here may also be driven by selection processes rather than causal ones, though. Also, a cautionary note should be cited regarding the interpretation of research findings as being in line with the process theory:

Ambiguous interpretations of research findings have also appeared in original work. In two studies of retirement and mortality [...] in which it was reported that retirement
was not detrimental to survival, the door was, nevertheless, left ajar to the opposite conclusion by reading year-by-year fluctuations in postretirement mortality rates as possibly indicating periods of stress or disenchantment with retirement. Speculations such as these are available throughout literature reviews. (Ekerdt, 1987: 456)

Further, some of the studies supporting the process approach can be said to also be consistent with one or more of the other theories discussed above, and none have explicitly tested the implications of this theory for health and mortality using methods suited for causal inference.
4 Deduction of hypotheses

[…] the crucial question is whether people retire because they are sick or whether they are sick because they retire

Atchley (1976: 87)

One of the main issues in the research on the relationship between retirement and mortality is the question of health selection and causality. In this chapter, I attempt to develop two sets of hypotheses based on the theoretical and empirical contributions discussed above. The first set of hypotheses relate to the descriptive associations between retirement and mortality. The second set of hypotheses concern the causal effects of retirement on mortality.

It is useful to keep a few points in mind for the remainder of this chapter. Age-specific mortality rates and the probability of dying at various ages are two related measures used to describe age-specific mortality. The age patterns of these two measures are essentially the same (Rowland, 2003: 275-89). Both increase approximately exponentially with age among adults, as illustrated in Figure 4.1. I therefore use the different terms interchangeably. The probability of being dead at any given age, however, is a function of the products of the conditional probabilities of dying at each previous age. This means that, depending on what measure is used, a hypothetical effect of retirement on mortality can be illustrated by two different patterns. One is based on age-specific rates or probabilities, measuring the risk or probability of dying at each age (the flow or the event). The other is the probability of having died before (or being dead at) each age or the proportion who are dead at that age (the stock or state). Personally, I prefer to think about theory in age-specific terms (event). The corresponding cumulative measures (state) are in some situations easier to calculate, and their calculation does not entail endogenous selection (more on this below). In the analyses, I use the two types of measures interchangeably. To distinguish between them can be a difficult exercise. Therefore, I describe both in some detail here. It is also well known that mortality rates differ for men and women, but in the discussions that follow I assume that gender differences are accounted for.
4.1 The association between retirement and mortality

Based on the theoretical discussions and the research reviewed in the previous chapters, there is little reason to assume that the age-specific mortality patterns should be identical for early retirees and the population at large. Rather, there is reason to expect a strong and negative health selection into retirement in accordance with a healthy worker effect (Li and Sung, 1999; Shim et al., 2013). There appears to be consensus in the research literature that disability retirees in particular are negatively selected and suffer much higher mortality than ordinary retirees (Shim et al., 2013). This should not come as a surprise, since poor health is a criterion for disability pension eligibility. But such a selection process may very well also be at work among regular early retirees, since people with health problems who are not eligible for a disability pension may be more prone to retire early with an old age pension, and since people in their late sixties who are eligible for early retirement and suddenly find themselves facing serious illness or deteriorating health may opt for early retirement. The association between retirement and mortality could also be expected in the strongly related case of reverse causality, whereby people afflicted with fatal illnesses would tend to retire early in anticipation of death (Skirbekk et al., 2010). Such health selection and reverse causality processes may also be strong enough to offset any potential mortality-reducing effects of retirement. As seen from the literature reviewed in chapter 3, the majority of descriptive studies point towards increased mortality following retirement, while the majority of studies employing causally oriented research designs do not. This could indicate that such processes may indeed be at work.
The age-specific mortality rates that one could expect to see in the period following retirement should therefore be expected to reflect an elevated mortality risk for retirees relative to people who have not retired. As shown in Figure 4.2a, this pattern may for instance take the form of a lasting difference between the two groups (line A) or a brief elevation in mortality rates (line B). A brief elevation should be expected in the case of reverse causality or health selection due to acute and serious illness. In such cases it is plausible that the mortality differences are largest immediately following retirement, since people who retire early would have acutely elevated mortality risk. A lasting (or even increasing) difference in mortality rates between retirees and workers over time could also suggest health selection, for instance in the form of a healthy worker effect whereby those still working at higher ages are increasingly positively selected with regard to health.

As shown in Figure 4.2b, a sustained difference in mortality rates should in cumulative terms display as monotonically diverging probabilities of being dead at each age between retirees and non-retirees (line A). In other words – the gap should gradually widen. A temporary elevation in the mortality rates among retirees should manifest itself in a permanently elevated probability of being dead at each subsequent time period. Such an elevated probability should more or less run in parallel with the corresponding probability among workers (line B).

*Figure 4.2a: Illustration of the hypothesized mortality rate pattern in relation to retirement age.*

*Figure 4.2b: Illustration of the hypothesized pattern of the probability of being dead in relation to retirement age.*

Based on this brief discussion, I set forth the following two hypotheses regarding the association between retirement and mortality:
H1: The mortality of retirees is higher in the period following retirement, relative to people who have not retired.

H2: This holds true even if disability retirees are excluded.

4.2 The effect of retirement on mortality

As explained in the discussion of theories, retirement may impact mortality in several different ways. Some theories predict retirement to increase mortality. Other theories predict retirement to reduce mortality. Some theories give reason to expect retirement to cause sustained or even continually increasing differences in age-specific mortality between those retired and those working. Other theories suggest that the effect could be short-term. Finally, the process theory suggests a time-varying effect, and Ekerdt (1987) suggested no effect at all.

An important point I wish to make when comparing the different theories and emphasizing Atchley’s process theory is the distinction between theories with regard to direction and temporality. Studies that have investigated the effect of retirement on mortality have generally, implicitly or explicitly, assumed an effect to be a monotonic quantum effect of retirement age. In other words, they have assumed that retirement age affects the number of deaths that occur between the retirement age and some arbitrarily set future age, for instance age 70, and that the effect points in one direction, i.e. that it is either positive, negative or nonexistent. Based on this assumption, they have assessed whether age of retirement affects the risk of dying before this set age. This approach is perfectly in line with what one could expect in accordance with theories that predict that retirement has a monotonic or short-term effect on mortality (theories about health behavior and work related risk, the Grossman model, the activity theory, the continuity theory, the disengagement theory and the stressful life event perspective).

To illustrate the predicted effects of these theories, Figure 4.3a shows four sets of hypothetical mortality rates patterns in thick lines. Theories about health behavior and work related risk could be in line with any of these patterns. Predictions from the Grossman model would be in line with line A and D. Predictions from the activity theory and the continuity theory

\footnote{It could be argued that the Grossman model allows for the effects of retirement induced health investment to vary over time, if, say, retirement causes both a weight loss and a sharp increase in tobacco consumption. In such a case, the short-term effect could be positive for health, while the long term effect may be negative. While consistent with the general idea of health investments, I do not see this view as consistent with the Grossman model.}
correspond to line A and B. The disengagement theory predicts a pattern in line with line A, and the stressful life event perspective predicts a pattern in line with line B.

Figure 4.3b illustrates how the age-specific mortality predictions in Figure 4.3a could be expected to either raise or lower the probability of being dead at subsequent ages. Note that when the number of deaths is measured cumulatively at age 70 (or some other arbitrarily set age), it should be possible to see an effect of retirement as a difference in the number of deaths between those retired and the control group.

**Figure 4.3a: Illustration of predicted mortality rate patterns caused by retirement according to different theories.**

![Mortality Rate Patterns](image)

**Figure 4.3b: Illustration of the corresponding predicted patterns of the probability of being dead caused by retirement.**

![Probability of Being Dead](image)

The alternative view of retirement as a process would, in line with Atchley’s (1976) theory, however, imply that retirement causes a temporary fluctuation in mortality rates in the period directly following retirement. If the honeymoon phase is associated with a healthier, more active lifestyle, this could cause a reduction in the mortality rates following retirement relative to the control group, followed by increased mortality in the disenchantment phase. If this health and mortality interpretation of the process perspective is true, Figure 4.4a could roughly illustrate the expected mortality rate pattern resulting from retirement (thick line). The corresponding pattern in the probability of being dead is illustrated in 4.4b. As may be

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model in its original conception. This is because an incorporation of such a fine-grained concept of health investment would require substantial revisions of the model.

8 Let me briefly propose two mechanisms through which such a result could be brought about. One mechanism could for instance be that latent health problems, such as a cardiovascular disease, could stay latent for a longer time due to a healthier lifestyle following retirement, and that it would eventually manifest itself after the honeymoon is over. Another mechanism could be that the phases are associated with reduced and increased suicide risk, respectively, due to the aforementioned experience of joy followed by depression induced by disappointment or anomie (Durkheim, 1978 [1897], but see Van Poppel and Day, 1996 and Pope, 1976).
seen from this figure, retirement would not necessarily cause a change in the total number of deaths. The number of people dying between the retirement age and some arbitrary later age (here shown as age 70) may very well be roughly the same in the retired and the control group, because the reduction in mortality rates in the honeymoon phase and the increased mortality rates in the disenchantment phase could more or less cancel each other out. In other words, retirement could have a *tempo effect on mortality*, rather than a quantum effect. Such a tempo effect may not be captured by an attempt to measure a quantum effect, unless it is measured in the period of mortality rate fluctuations immediately following retirement. In order to fully capture such an effect, one would not just have to model age-specific mortality, but rather mortality relative to both age and duration since retirement. This requires detailed data on retirement and age- and duration-specific mortality. I have such data, and this allows me to test this theory.

**Figure 4.4a: Illustration of the hypothesized mortality rate pattern caused by retirement in line with Atchley’s theory of retirement phases.**

**Figure 4.4b: Illustration of the hypothesized pattern of the probability of being dead caused by retirement in line with Atchley’s theory of retirement phases.**

Based on the discussion above, I set forth several mutually exclusive hypotheses, H3 through H5:

- **H3a:** Retirement causes a lasting increase in mortality rates.
- **H3b:** Retirement causes a short-term increase in mortality rates.
- **H4a:** Retirement causes a lasting reduction in mortality rates.
- **H4b:** Retirement causes a short-term reduction in mortality rates.
- **H5:** Retirement causes an initial reduction in the age and duration specific mortality rates, followed by an increase.
H3a would be in accordance with theories about health behavior and work related risk, the Grossman model, the activity theory, the continuity theory and the disengagement theory. H3b corresponds with theories about health behavior and work related risk, the continuity theory and the stressful life event perspective. H4a is deduced from the theories about health behavior and work related risk and the Grossman model. H4b would be in accordance with theories about health behavior and work related risk, while H5 corresponds with the process theory. Obviously, there is a null hypothesis here as well, namely that retirement does not affect mortality. This would be in accordance with the no effect assumption.

If there is an effect of retirement on mortality, and particularly if retirement increases mortality risk, the fact that I (for reasons given below) sample cohorts where there is a large difference in labor force participation between men and women leads me to expect that the effect will be larger for men than for women. This is because the role loss associated with retirement in the time period studied in this thesis (the 1970s and onward) is likely greater for men than for women due to men’s breadwinner role in this generation (Birkelund and Petersen, 2016). This would be in line with the view of retirement as a stressful life event, the disengagement theory, the activity theory, the continuity theory, and the discussion in Coppola and Spizzichino (2014). A similar argument may be based on the Grossman model, although without a specified direction. Since women earn less and work more part-time, the changes in income, incentives structure and available time following retirement may be smaller for women than for men. This points towards a larger effect for men. Higher mortality among men and thereby higher baseline risk also points in the same direction. In any case, I can formulate H6:

H6: If retirement increases mortality risk, the effect is stronger for men than for women.

As mentioned in section 2.2.1, it is likely that an effect of retirement on mortality follows a socioeconomic gradient. But the direction is not obvious. Retirement from low-status manual jobs associated with health risks such as exposure to cold weather, harmful chemicals etc. may be beneficial to health, as clearly indicated by Westerlund et al. (2009). But people voluntarily retiring early from such jobs might also be particularly negatively selected. Also, the socioeconomic differentials in lifestyles may imply that people retiring from high-status and well-paid jobs are more inclined to spend the increased spare time on activities that are beneficial for their health. In that case, the increase in available time following retirement could amplify the socioeconomic gradient in health behavior. On the other hand, people in
such occupations are also less likely to retire early. Ideally, it would be interesting to explore effect heterogeneity with regard to different socioeconomic measures, such as income, education and occupation groups. However, due to there being a lot of missing values on both the occupation variable and the income variable, this is difficult. Therefore, I am left with education levels as the best candidate for a socioeconomic indicator. Because people with lower education are more likely to be in jobs that are associated with health risks, and may therefore benefit more from retirement I suggest the following hypothesis:

H7: If retirement affects mortality risk, earlier retirement reduces mortality more, or increases it less, for people with low education, relative to people with higher educational levels.

Finally, as noted before, if retirement indeed has an effect on mortality, I do not expect this effect to be large.
5 Choice of empirical strategy

To test the different hypotheses deduced in chapter 3, I have chosen two different empirical strategies. First, I test hypotheses H1 and H2 concerning the association between retirement and mortality through event history analyses. A discussion of the pros and cons of different approaches to such analyses follows below. Second, I treat a pension reform that affected the 1903 through 1906 cohorts as a natural experiment, and exploit it as a source of exogenous variation in retirement eligibility. I do this in an attempt to provide estimates of the relationship between retirement and mortality that can be given a causal interpretation.

As stated earlier, I take retirement to mean work exits in relation to old age (usually in combination with the uptake of pension benefits). For practical reasons, however, I operationalize retirement as the uptake of benefits and do not condition on work exits. In practice, then, I use the uptake of benefits as a proxy for retirement.

5.1 The association between retirement and mortality

Hypotheses H1 and H2 concern the association between retirement and mortality. Due to strong health related selection into retirement, H1 should be expected to hold true regardless of whether there exists a causal relationship between retirement and mortality. H2 specifically addresses the issue of health-related selection into retirement though disability pensions. It suggests that even when disability pensioners are excluded, the health selection may be strong enough to produce a positive correlation between retirement and mortality. This selection process could work in combination with a causal mechanism, or it could run counter to it (but be strong enough to suppress it).

Ideally, and in isolation, these hypotheses could be tested on recent data on retirement and mortality. But in the context of this thesis, it is more interesting to test these hypotheses on cohorts that retired around the same time as those included in the second part of the analyses. For reasons given below, I test hypotheses H3 though H7 about the causal relationship between retirement and mortality on cohorts that retired in the early 1970s. To make the results from the two parts comparable, I have chosen to test hypothesis H1 and H2 on the 1906 and 1907 cohorts. These became eligible for old age pensions at age 67, and were born close enough in time to be comparable with regard to age-specific mortality. In addition, they became eligible for retirement at or around the same time as those affected by the 1973
pension reform, which I use in the second part of the analysis. In fact, the 1906 and 1907 cohorts were the first cohorts to be fully affected by the reform.

The data I use in this thesis contains detailed person-year information on retirement and mortality for these cohorts. It is also possible to distinguish between several types of pensions. For the present purposes, the possibility to distinguish disability pensioners from other pension benefit recipients is particularly important. To test hypothesis H1 and H2, I apply discrete-time hazard regression methods to the retirement and mortality data, fitting separate models that both include and exclude disability pensioners.

5.2 The effect of retirement on mortality

In order to test hypotheses H3 through H7 and provide estimates of the correlation between retirement and mortality that can plausibly be given a causal interpretation, I need to identify and exploit a source of exogenous variation in retirement age. People may decide for themselves when to retire, to a large extent. And this decision is likely influenced by factors that also affect mortality, such as health. As such, those who retire early and those who retire late are likely different in many regards. This means that to estimate the effect of retirement, it is necessary to identify a situation where it is plausible (under some conditions) to assume that those who retire early and those who retire late are similar on salient properties. In other words, an experiment or a natural experiment is required. True experiments in this area are hard to come by. But to the best of my knowledge, there have been three different large-scale Norwegian pension reforms that may serve as natural experiments. Here, I briefly discuss these reforms in reverse chronological order, and point to the pros and cons of using each of them in an attempt to estimate the causal effect of retirement on mortality. I then try to justify the choice I have made.

5.2.1 The national insurance pension reform of 2011

The pension reform of 2011 comprised of several important elements. Among these was the option of early retirement from age 62 for individuals with sufficient cumulative pensionable income, increased flexibility in the form of the option of combining (full or partial) pension outtake with continued (full or partial) work force participation, and strong economic incentives to postpone retirement in relation to increases in life expectancy (The Ministry of Labor and Social Inclusion/Arbeids- og inkluderingsdepartementet, 2006; Fredriksen and Stølen, 2011).
At first glance, the pension reform of 2011 may be considered a good candidate for the role of a natural experiment. It represents a major and exogenous change in individuals’ opportunities for early retirement, as the entitled retirement age was reduced from 67 to 62. Although many people already had the option of retiring early through contractual early retirement schemes (AFP), a considerable proportion of the affected cohorts did not, and they were effectively given the opportunity to retire up to five years earlier than previous cohorts (Hernæs et al., 2015). This feature of the reform alludes toward a difference-in-differences (DiD) strategy or an instrumental variables (IV) approach whereby being in a cohort affected by the reform at a given age functions as a treatment or instrument for retirement age. The cumulative earnings threshold for eligibility into early retirement of 1,85 G for married and 2 G for unmarried people (Fredriksen and Stølen, 2011) may also be considered a somewhat arbitrary cutoff – a feature that makes this reform attractive for use in a regression discontinuity framework, if one can overcome the issues related to self-selection into eligibility. In combination, the reduction in retirement age and the eligibility threshold may be used in a triple differences (DiDiD) estimator by using the between-cohorts variation in retirement eligibility age resulting from the reform implementation and the within-cohort variation resulting from the lifetime earnings threshold. In fact, Hernæs et al. (2015) have done something similar to this approach in an attempt to estimate the impact of different aspects of the reform on labor supply.

On the other hand, there are some major issues that discourage the use of this reform as I have suggested here. First, the strong incentives to postpone retirement mean that many people may in fact retire later than they otherwise would have, despite being given the opportunity to retire earlier. In fact, the reform has on average been shown to increase the actual retirement age (Fredriksen and Stølen, 2015). In an instrumental variables framework, this means that there is likely to be a substantial number of defiers (people who do not take the treatment because they are offered it or vice versa) (Angrist and Pischke, 2008: 156-7; 2014:112-5; Borgen, 2013). In other words, a lot of people likely postponed their retirement because they were affected by the reform, and despite the reform allowing them to retire earlier. Having a lot of defiers in a sample strongly biases IV estimates (Borgen, 2013). This problem may, however, be possible to overcome by controlling for the effects of such incentives or by excluding individuals who are subject to them. Second, given that one cannot condition upon actual retirement in a DiD framework, the changes in incentives and benefits associated with

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9 An explanation of the concepts of defiers, compliers, always-takers and never-takers is given in section 7.4.
the pension reform make it difficult to ensure that an estimation of the effect of the reform reflects the effect of lowered retirement age and not the effects of changes in benefits and incentives structures. This problem may also be possible to overcome through control or exclusion. Third, the fact that many people were already eligible for early retirement before the reform through the contractual early retirement scheme makes it difficult (but not impossible) to isolate the population that was affected by the reform. Fourth, the fact that the reform was anticipated several years in advance means that people may have been able to self-select into eligibility, for instance through increased labor market participation in the final years before retirement eligibility age in order to reach the earnings threshold sooner. This is more difficult to account for. Fifth, the fact that this reform is fairly recent means that the effect is only observable for a short time period, meaning that only short-term effects may be identified. Finally, few people die in their early sixties. The remaining life expectancy at age 62 was 20.5 years for men and 23.8 years for women in 2011 (SSB, n.a.). This means that even if retirement age actually impacts mortality, it may be difficult to identify a statistically significant short-term effect in these age groups. These issues have in sum led me to opt not to use this reform in this thesis.

5.2.2 Lowering of the contractual early retirement age in the 1990s

Between 1989 and 1998, the minimum retirement age in the contractual early retirement scheme (AFP) was lowered in a stepwise fashion from 67 to 62 years (NOU 1998: 19). As mentioned before, this gradual reduction in the eligible retirement age for one group of workers was used by Hernæs et al. (2013) in their IV and hazard rate triple differences estimation of the effect of retirement on mortality.

The advantages of using this reform in an identification strategy are laid out in detail by Hernæs et al. (2013), and are not discussed here. But I will mention a few drawbacks of using this reform. First, the treatment groups are essentially identified by cohort membership and AFP eligibility (i.e. being employed in an AFP firm). This means that the instrument could be invalid if the trends in cohort mortality differ between those employed in AFP firms and those who are not. The paper showed that this does not appear to be the case. However, it could in theory still be so in the implausible scenario that such differential mortality trends and the effect of retirement age pointed in opposite directions and more or less precisely cancelled each other out. I personally find this unlikely. A more important point concerns the question of whether this reform is the best candidate for identifying a hypothetical causal effect of retirement on mortality. The remaining life expectancy at age 62 was 18 years for men and
22.2 years for women in 1998 (SSB, n.a.). Since few people died in their early 60s in the 1990s, a hypothetical causal effect may be too small to identify in this age group, but may still be possible to identify at higher ages. Such a scenario would not be at odds with the findings of Hernæs et al. (2013), but would point towards there being a mortality effect of retirement in other age groups or periods than those that they studied. Also, the identification of the treatment and control groups in this scenario is very complicated. Lastly, the fact that Hernæs and colleagues have used this reform in their study also means that there would be little to gain from using it again in this thesis, except for the purposes of replication. Using a different sample and a different reform may, however, contribute more to the accumulating research literature on this topic.

5.2.3 Lowering of the retirement age in the national insurance scheme in 1973

In the law on old age insurance was passed in 1937, the statutory minimum retirement age was 70 years (Ministry of Social Affairs/Sosialdepartementet, 1971). In 1973, however, this age limit was lowered to 67 (Amendment to the law on social insurance, 1972)\(^\text{10}\). As a policy experiment, this reform has some appealing features. First, it was a rather simple reform. The main reform element was the lowering of the entitled retirement age by three years, and this change happened at a single point in time – on the 1\(^{\text{st}}\) of January 1973. This sets it apart from the more complex reform of 2011 and the gradual reduction in entitled retirement age in the contractual early retirement scheme in the 1990s. Second, the fact that the reform happened more than forty years ago provides a long observation window and facilitates estimation of the long-term effects of retirement on mortality. Third, the complexity of the 2011 reform makes the identifiable treatment effect a very local one (meaning that the effect can only be estimated for specific subgroups) (Hernæs et al., 2015), and the contractual early retirement reform only affected those covered by the system. But the 1973 reform affected the majority of the working population, meaning that it is theoretically less problematic to generalize the results to different groups (though not necessarily across time). Fourth, the fact that the important eligibility criterion is related to birth cohort membership means that people could not self-select into treatment. People can’t change birth cohort like they can change jobs in response to a policy reform. Finally, the reform happened in a period in time, and affected people at ages, where the mortality was markedly higher than the later reforms discussed above. More people die in their late than in their early sixties, and mortality was higher in 1973 than in the 1990s and after 2011. In fact, the remaining life expectancy at age 67 was

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\(^{10}\) I originally got the idea to use this reform when I read Brunborg et al. (2008) and saw the figures on page 28.
just 12.6 years for men and 15.4 for women in 1973 (SSB, n.a.). The fact that Hernæs et al. (2013) did not find an effect of the reform in the 1990s may be attributable to the fact that they studied the effect of a recent reform affecting an age group with low mortality. In this sense, the 1973 reform serves as a critical case (Flyvbjerg, 2006); if an effect of retirement on mortality cannot be found when using the 1973 reform, it is unlikely that one can be found at all.

A complicating matter is the fact that the 1973 reform was introduced together with a “waiting supplement” (“ventetillegg”) that provided some incentive to delay retirement (NAV, 2011). However, given that this benefit only worked as an incentive to postpone retirement up to age 70 (the pre-reform limit), it is far less prone than the 2011-reform to produce many defiers, although not everyone opted to retire at the earliest opportunity. In theory, people who were affected by the reform, but who did not retire until after age 70, should then be never-takers rather than defiers, since there was no incentive to defy beyond age 70. This is because defiance would mean postponing retirement until age 70 or later, when there was no more waiting supplement. However, people were not allowed to self-select into retirement eligibility (i.e. the treatment groups), but once eligible for retirement, they could decide on their retirement age within the constraints of the system.

Another complicating matter is the fact that not all employees were affected equally by the reform. Some occupations in some firms and government institutions and offices already had lower statutory retirement ages at the time of the reform. With regard to the government employees, the jobs with lower retirement ages are listed in the recommendation from the committee on retirement age (Pensjonsalderkomitéen, 1971). The inclusion of people with lower statutory retirement ages will bias the estimates of the mortality effect of this reform slightly towards zero.

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11 Of course, it is always possible to come up with a just-so story about how the reform may generate defying behavior. Here is an attempt: “In 1973, Peter had planned to retire early with his savings at age 68. The reform gave him incentives to work until age 70, so he did. After all, he was a rational actor with transitive preferences, who valued his leisure more than his wage, but less than his wage plus the waiting supplement. By age 70, Peter had realized that he preferred working to retiring. His preference ordering had shifted, perhaps because old age had deprived him of his rationality. Or maybe it had bereaved him of his spouse and life partner so that he felt little reason to retire. In any case, Peter worked for two more years, until his health failed him, and he retired as a defier at age 72.” The plausibility if this identification strategy partly rests on the implausibility of such untestable just-so stories, i.e. on the likely number of defiers and the extent of their defiance being small (Borgen, 2013).

12 By using data on occupations from the 1970 census, it could have been possible to remove some of this bias. These census data contain information on occupation titles and industry/field (Vassenden, 1987). This could have made it possible to (in broad strokes) exclude individuals who were in occupations less affected or unaffected by
Finally, the fact that this is an old reform makes it less appealing. Estimating the effect of something that happened in 1973 is not exactly newsworthy. The relevance and external validity of the findings to present day elderly may be questioned, since both working lives, lifestyles health, mortality and the availability of health and welfare services have changed considerably since then. However, I would argue that the mechanisms that potentially could produce an effect in the early 1970s may very well still be at work today, though perhaps tempered by other developments. Furthermore, the critical case argument made above makes this reform theoretically interesting, as it could serve as a critical test of the theories postulating an effect of retirement on mortality.

All in all, two estimation strategies based on the 1973 reform appear promising. The first is a DiD-estimation whereby cohorts affected by the reform at different ages are compared to cohorts that are not affected, with variation in treatment status arising from the between-cohort variation in age at the time of the reform implementation. This could provide an estimate of the net effect of the reform (the intention to treat effect) on the cohorts affected.

By also introducing within-cohort variation in eligibility at each age by distinguishing between groups affected and not affected by the reform (for instance because they are unemployed or homemakers) in a triple differences (DiDiD) estimator, it could be possible to get a more precise estimate of an effect of being offered to retire earlier (Angrist and Pischke, 2014:119-20). Such an estimator would measure something more akin the effect of actually being offered to retire (not just being in a cohort where most people were given this offer) while also providing some some safeguard against the potential confounding of calendar year effects (such as a rough flu season etc.). Introducing within-cohort variation in retirement eligibility would in other words reduce bias since people eligible and not eligible for retirement could be assumed to be subject to the same calendar year variations in mortality risk. However, finding and identifying such groups appears to be be easier said than done.

The second promising strategy is to use the reform as an instrument for retirement age. Assuming that which cohorts were affected by the reform is unrelated to the differences the reform. But unfortunately, as shown in chapter 8, the number of missing values on the occupation variable is too large to make such an analysis meaningful.

13 In fact, Norway was hit by a flu pandemic in 1977, commonly referred to as the “Russian flu”. This could potentially bias the results by introducing calendar year effects. However, Mamelund and Iversen (2000) did not find evidence of excess mortality associated with this flu pandemic.

14 One option could be to use housewives as a within-cohort control group. These would not be affected by the reform since they were not employed. But if there are spillover effects of the husbands’ retirement, this could also bias the results.
between cohorts in characteristics that affect mortality (such as education, health etc.), I can assume that the between-cohort variation in retirement eligibility age is exogenous. Further, assuming that the reform also had an effect on the retirement ages of the affected cohorts, it is possible to use this reform-induced variation in retirement age to estimate the effect of retiring earlier (or later). This first assumption is not directly testable, but the second one is, as I will show below.

Policy reform instruments are frequently used in social research, as can be seen in the literature review in chapter 3. This is partly because they are often plausibly exogenous. Policy reforms that affect different segments of the target population differentially through arbitrarily defined eligibility rules or timing can often be assumed to be unrelated to other characteristics that affect the outcome of interest. This makes it easier to argue that a given reform constitutes a valid instrument. An IV approach could also be theoretically more interesting than a DiD approach, as it would (hopefully) yield estimates of the local average treatment effect for the compliers (Angrist and Pischke, 2014: 111-5). It would thus give estimates of the effect of retiring, not just being offered the opportunity to retire. To be precise, I would measure the average effect of retiring a given number of years earlier than one otherwise would have, within the age span of 67 to 70 years in the early- to mid-1970s, because one is allowed to do so because of the reform. This may appear to be a very local and narrow (though well-defined) effect, but it is theoretically interesting because it could be interpreted as a retirement effect, not a reform effect, making it more plausible that the findings may be generalized to other, similar contexts. The point that the treatment effect would be local is important. If the effect can be assumed to vary across contexts, groups or time, one should be careful about generalizing the results. A drawback is that IV estimation is accompanied by large standard errors (Murray, 2006), leading to less precise estimates and an increased risk of committing a type II error (i.e. failing to reject a false null hypothesis).

In sum then, I opt to use the 1973 reform to identify an effect of retirement on mortality. I find this reform to be most appealing, as it gives a long follow-up period, relatively simple identification, population-wide treatment effects and high chances of identifying an effect if there is one. Nonetheless, the abovementioned drawbacks to using this reform should be kept in mind.
6 Data

For the empirical analyses in this thesis, I rely on high quality register data from Norwegian administrative registers, as well as on data from the 1970 census. The original datasets from which my samples are drawn comprise the entire Norwegian resident population that has been alive at some point since 1967.

Approval to use these data was obtained from the privacy ombudsman at Statistics Norway and the sections responsible for the data at Statistics Norway. This process was coordinated by the project manager. The approval is attached in the appendix, together with a note confirming an approval to expand the sample to cover people alive in 1967-1969. All datasets were provided by Statistics Norway.

6.1 Data sources

I use data from population registers (births, deaths, immigrations, emigrations, age, sex and immigrant background), income records (pensionable income), welfare benefit registers (pensions), and census data (occupation, industry and education levels).

The original data on demographic variables from the population registers were organized in two files, each with one record per individual. Only observations with valid project-specific ID numbers were included. One of the population register files contained information on immigrant backgrounds while the other contained information on other characteristics Time-invariant characteristics were coded as categorical variables, while events were coded as dates, months or years. I only used information about events per month and year. For immigration and emigration, only the first immigration date and the last emigration date were used. Immigrant background was coded into three categories; immigrants, Norwegian-born with two Norwegian-born parents, and all others. In some analyses, these last two groups were merged. Income records were also organized with one record per individual, but with one variable per year since 1967.

The data on welfare benefit recipiency was organized as one record per person-event. These data also only had information on events per year. I coded one variable for each pension type (old age pensions, disability pensions and all other pensions types combined) that contained information on the first year of recipiency, as well as one variable indicating the first year each individual received any type of pension. Before merging these data with the other
datasets, I deleted all records of the same individual except one, which then contained detailed data on the year of entry into different pension types.

The census data was also coded with one record per individual. With regard to educational levels, the nine groups in the original data were coded according to the 80-standard (Vassenden, 1987). These have been collapsed into four groups – no education (including preschool), primary education, secondary education/high school, and university/college education. Occupations were also coded according to the 80-standard. In chapter 8, I show the proportions of the samples that belong to each of several broad occupational groups. But the large number of missing values on this variable has led me to opt not to include it in the analyses.

6.2 Merging, recoding and sample restrictions

Merging of datasets and preliminary recoding of variables was done in the SAS Enterprise Guide 7.1 software package. An overview of the different steps in the sample restriction process is provided in Table 6.1. Starting with one of the population register files, I deleted 1610 individuals who were not resident, dead or emigrated (people with missing status codes, people who have disappeared, etc.). The two population register files were then merged using the PROC SQL inner join procedure. This procedure discards all observations not present in both datasets, ensuring that there are no duplicates or people with information from only one of the files. I then deleted 40 individuals whose death times did not match in the two population register files. I also deleted 11 people with missing times of both death and emigration.

The income file was merged with the population register dataset using the PROC SQL left join procedure. This procedure ensures that the population register files are given primacy, so that observations from other files are only merged to observations in the population register files if they can be linked to an individual in these files. In other words, observations in other datasets that could not be matched with an individual in the population register files were discarded, while all individuals in the population register files were retained. All cohorts except those born between 1902 and 1907 were then discarded. All other datasets were then sequentially merged with the combined population register and income files using the PROC SQL left join procedure. Old age pension eligibility age was derived from each individual’s birth year and birth month.
The complete dataset contained information on 255 138 individuals. This equals 99.35 percent of the original sample. Only counting people alive at age 65 reduces this number considerably (by 26 426 individuals). This restriction is imposed in most analyses in this thesis since imposing it means that I avoid issues related to the fact that the registers were established in 1967. Based on this complete dataset, three separate files were produced, exported to Stata format, and then used in the analyses. Analyses were performed with the Stata 13.1 software package. The first file, which I will denote the “wide file” contains one record per individual. It has information on event times in years and months. This file is used to produce much of the descriptive statistics on time-invariant characteristics and to perform the IV analyses. It contains information on 228 712 individuals who lived beyond age 65 when the 1907 cohort is included, and 189 634 individuals who lived beyond age 65 when it is not. In one robustness check, I keep the 1907 cohort when estimating an IV model with a linear cohort trend. The second file, hereby denoted the “long file with years” is organized as a conventional panel dataset with one observation per person-year. Individuals are censored before immigration and after emigration and death. This file is used for the event history analyses. It contains information on 77 422 individuals from the 1906 and 1907 cohorts who lived past age 65. The total number of person-year observations for this group is 1 348 473. The third file can be denoted the “long file in months”. It contains one observation per person-month and information on time-variant variables at each month. This dataset is not censored at death, and each individual is followed until age 75 regardless of whether they die (although I never use observations for ages higher than 70). This dataset contains information on 189 634 individuals from the 1902-1906 cohorts. It is used for the DiD and individual FE models.

In both long files, time-variant statuses (such as not retired/retired) were coded so that they refer to the first full month or year in which the person holds that status. Event variables were coded so that they refer to the time intervals within which the event took place. Let me give an example based on the long file with years. A person who is followed from 1970 to 1975 retired sometime in 1973. The retired-variable would then have the values 0,0,0,1,1. The event variable would have the values 0,0,0,1,0,0.
Table 6.1: Number of people in each cohort and the impact of each sample restriction.

<table>
<thead>
<tr>
<th></th>
<th>Cohort</th>
<th>Number deleted</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1902</td>
<td>1903</td>
</tr>
<tr>
<td>Original dataset (those with valid project ID)</td>
<td>41 387</td>
<td>41 996</td>
</tr>
<tr>
<td>Keeping only those resident, emigrated or dead</td>
<td>41 220</td>
<td>41 781</td>
</tr>
<tr>
<td>Keeping only those with matching death time</td>
<td>41 218</td>
<td>41 773</td>
</tr>
<tr>
<td>Keeping only those with death or emigration time</td>
<td>41 218</td>
<td>41 773</td>
</tr>
<tr>
<td>Sample restricted to those alive at age 65</td>
<td>37 699</td>
<td>37 901</td>
</tr>
</tbody>
</table>
7 Methods
The analyses in this thesis can be divided into two parts. First, I examine the association between retirement age and mortality using event history analyses. These analyses shows how mortality varies with retirement status and retirement duration (H1) and to what extent the association is driven by health selection into disability pensions (H2). In the second part, I employ two different methods suited for analyzing quasi-experimental data. I use these methods to exploit the (presumably) exogenous variation in retirement eligibility age that stems from the lowering of the retirement age in 1973. These analyses show whether and how retirement affects mortality (H3 through H7).

7.1 Event history analysis
For the event history analyses in this thesis, I rely on discrete-time hazard regression models. These models are useful for analyzing mortality data because they can estimate the conditional odds, log-odds or hazards of failure in different time intervals. In many cases, the researcher is interested in knowing the risk of failure (death) in some time interval \( t \) for the population at risk in the time interval. This entails excluding those units that are not at risk (in the case of mortality this could for instance be because they are not resident or because they are dead) and condition on being at risk in the time interval. In an event history analysis, this is achieved by censoring cases not at risk (Allison, 1995: 9-14; Rabe-Hesketh and Skrondal, 2008: 331-333). Note that censoring due to emigration is likely informative in the analyses presented in this thesis (Allison, 1995: 13-14), since it is reasonable to assume that healthier people are more likely to emigrate. But excluding people who eventually emigrate is not a good option either, since that would be to condition on a collider variable (Elwert and Winship, 2014). I simply do not see a good solution to this issue, except noting that the proportions who emigrate at old ages are very low (see Table 8.1) and unlikely to affect the results much.

In a discrete-time hazard regression model, time is measured at discrete intervals, and, commonly, only integer values are used. In the version of the model that I apply here, dummies for each time period are used to account for time. This way I do not have to make any assumptions about the functional form of the relationship between the baseline hazard and time (Rabe-Hesketh and Skrondal, 2008: 334-350). The discrete-time hazard regression model can be estimated as a logistic regression model on censored panel data. Formally, the model can be expressed as
\[ \text{logit}[P(y_{si} = 1|D_{si}, A_{si}, X_{si})] = \beta_1 + D_{si} + A_{si} + \beta_2 X_{si} \]

where \( y \) is the outcome of interest (death), \( i \) indexes individuals, \( s \) indexes age in years starting at age 60 and running until each individual is censored, \( A \) is a set of dummies for each age interval spanning from age 60 to censoring for each individual, \( D \) is a set of dummies for each age interval following retirement, and \( X \) is a vector of both time-varying and time-invariant covariates, including retirement status.

For the analyses of the association between retirement and mortality, I restrict my sample to the 1906 and 1907 cohorts. Both of these cohorts became eligible for old age pensions at age 67, so variation in retirement age is likely endogenous. While not advantageous in its own right, this endogeneity makes it interesting to compare the results to the estimates provided by the DiD, individual FE and IV estimators. Also, it is interesting to know about the relationship between different retirement types and mortality regardless of the causality of the relationship. One reason is because strong health selection into early retirement could be seen as an argument for improving the health of older workers to prolong their working lives.

Since data on the uptake of pension benefits is only available at yearly time intervals, these analyses are based on the dataset containing information on person-years. Due to the limited availability of relevant time-varying control variables for the cohorts under study, the analyses are somewhat limited in scope. However, since these analyses are motivated by hypotheses H1 and H2 concerning the mortality patterns following retirement by different pension types, this is not a major concern. The main objective here is to examine whether or not the mortality of retirees is higher in the period following retirement relative to people not retired, and whether this holds true even if disability retirees are excluded. The question is not whether or how such correlations are related to other time-varying covariates.

In the analyses reported below, I first estimate models where I regress death on retirement duration dummies, age dummies and time-invariant controls. If there is no duration effect, just an effect of being retired regardless of duration, this would yield similar point estimates for each duration dummy. Second, I exclude disability pensioners to see if the association between retirement or retirement duration and mortality is driven primarily by people receiving disability pensions. Acknowledging the issues related to unobserved heterogeneity in logistic regression (Mood, 2010), I report average marginal effects for the variables of interest.
### 7.2 The policy reform experiment

The lowering of the minimum old age retirement age in 1973 meant that the 1906 birth cohort was allowed to retire three years earlier than the 1902 cohort, and that each successive monthly birth cohort born between January of 1903 and December of 1905 was allowed to retire one month earlier than the previous cohort. This provides up to three years’ worth of plausibly exogenous variation in retirement eligibility age. The eligibility age for each birth cohort and their age on the 1st of January 1973 are shown in Figure 7.1.

This design means that there is no untreated control group – only groups that differ with regard to their treatment duration, age or timing (depending on how you look at it). At age 70, everyone is treated. In analyses depending on comparisons of treatment and control groups (the DiD and individual FE estimations) I can only really look for relatively short-term effects of retirement eligibility on mortality. This is potentially a problem for investigating the long term effects of retirement on mortality as hypothesized in H3a and H4a. For such an analysis, the IV approach is necessary, and the estimated effect must be one of retirement age rather than eligibility status. With regard to the potential short-term fluctuations in mortality following retirement, as hypothesized in H5, I expect to be able to identify at least a honeymoon effect in the short term of up to three years if there is one.

**Figure 7.1: Earliest retirement eligibility by monthly birth cohort.**
7.2.1 The effect of the reform on actual retirement

The identification strategy in this thesis rests on the assumption that the 1973 reform, which lowered eligible retirement age, affected actual retirement age. Flittig (1992: 24, 34) has shown that this was indeed the case. The number of old age retirees increased by 28 percent from 1972 to 1973. From Figure 7.2 it is evident that some of this increase was due to a substitution from disability pensions to old age pensions. The increase in the total number of retirees due to the reform is nonetheless substantial.

*Figure 7.2: Number of national insurance retirees by year and type of pension.*
*Source: Flittig (1992: 24). I have edited the figure to translate the text from Norwegian.*

Turning the attention to the cohorts in question and the data used in this thesis, Figure 7.3 plots the mean age of first claiming different retirement benefits by birth cohort. The 1907 cohort is included here for comparison. Since the claiming of benefits is measured only at the end of each year, the (presumably systematic) birth month variation in mean retirement age is not visible in these data. Therefore, the level of detail is lower in this figure than in Figure 7.1, and the effect artificially appears to be shifted by half a year.
Comparing the plotted average retirement ages for old age pensions in Figure 7.3 with the line marking the eligibility age in Figure 7.1, the marked effect of the reform on actual retirement is easy to see. Between the 1902 and the 1906 cohorts, average old age retirement age fell by more than two years for both sexes. This is good news for the identification strategy. It should be noted that attrition due to mortality is not accounted for here. Some people in the earlier cohorts died after age 67 but before they reached their eligibility age, and therefore could not retire. They do not have a retirement age and are therefore not counted in the cohort mean. People in later cohorts who died at the same age, but who could and did retire first, are counted. This may bias the means, but due to the potential endogeneity, it is not clear which direction the bias would point. However, conditioning on being alive at age 70 hardly alters the results (not shown), so this is likely not a major reason for concern.

A complicating matter is the fact that old age pensions and other pensions (mainly disability) to some extent are substitutes. This means that the effect on average pension age for all types of pensions of lowering the old age pension eligibility age is tempered by people substituting other pensions for old age pensions without actually retiring earlier. Because of this substitution, it is difficult to estimate the effect of retirement on mortality using only the variation in old age retirement ages resulting from the reform. Rather, I should not condition on type of retirement, but use the variation in all types of retirement benefit uptake. However, looking at all pensions taken together, there is a downward trend in the average retirement age between consecutive cohorts in addition to the apparent reform effect. This trend stretches well beyond the cohorts plotted in Figure 7.3 (not shown). This means that some of the variation between cohorts in mean retirement age may be due to cohort- or time related trends rather than the retirement reform. I therefore also test whether such trends bias the results.
7.3 Difference-in-differences
Most of the theoretical contributions discussed in chapter 2, suggest that retiring should have some effect on mortality. Unless the effect has the form suggested by H5, I should be able to measure an effect of retirement – positive or negative – on the mean probability of being dead in the period after retirement.

In the difference-in-differences (DiD) framework presented here, I cannot condition on actual retirement, as uptake is likely endogenous. Therefore, I am forced to treat retirement eligibility as a proxy for retirement in order to provide estimates that can be given a causal interpretation. Put differently, I measure an average effect of retirement eligibility, rather than an effect of retirement. As I have shown above, the reform that lowered the eligibility age appears to have had a strong effect on actual retirement behavior, so eligibility age appears to be an acceptable proxy for retirement age.

Theoretically, I assume that the effect of being eligible for retirement on mortality is mediated by the effect of eligibility on retirement uptake. Given due precautions, then, the results can be considered a somewhat diluted retirement effect or a reduced form effect, whereby the estimates measure the average effect of being treated (eligible) for those offered the treatment (including both those who accept the offer and those who do not), compared to those not offered the treatment (including both those who do not retire and those who retire despite not...
being eligible). Alternatively, the results can be interpreted as the net reform effect – the effect of being offered to retire earlier. While the diluted retirement effect is the most theoretically interesting interpretation, the net reform effect interpretation is the most policy relevant. Since these analyses do not condition on actual uptake of old age pensions, there is little reason to worry about the potential source of confounding that arises from the fact that old age pension and disability pension are likely to be substitutes.

The DiD estimator relies on the assumption that in the absence of treatment, the time trends in the treatment and control groups would be parallel. Using a DiD approach, I do not need to worry too much about differences between the treatment and control groups as long as this assumption holds. The DiD estimator exploits this parallel trends assumption to estimate the effect of a policy change by estimating the difference between the observed outcome in the treated group and the outcome that one would expect in the treated group if the trend in this group had moved in parallel with that of the control group (Angrist and Pischke, 2008: 227-241; 2014: 178-208).

Let me exemplify. A standard DiD approach uses aggregate data on different groups at two or more points in time to measure the impact of a policy change or some similar change that affects the treatment group but not the control group. The effect is measured as the difference between the treatment and control group in differences between the pre- and post-treatment outcomes. Hence the name. A typical application of this design could be measuring the impact of some reform that affects a specific geographical area, for instance a labour market regulation in US state A, on an outcome of interest, such as unemployment. This could be done by comparing the difference between pre and post reform unemployment rate in state A to the difference in the unemployment rates at the same points in time in the neighboring state B (Angrist and Pischke, 2014: 178-208).

In this thesis, I apply this DiD logic in an OLS model, but with some modifications. The first is that I use individual level data, rather than aggregate measures. This data is a panel, rather than a cross- section15. Treatment and control groups are defined by monthly birth cohort membership, since eligibility age varies between cohorts. The time dimension in this setup is not calendar time but age. The reason for this is that since the probability of being dead increases nonlinearly with age, I cannot assume that the cohort trends in time-specific

15 Actually, since practically the entire population (defined as the 1902-1906/1907-cohorts) is sampled, the data is both a panel and a repeated cross-section.
mortality would be parallel. The difference in the probability of being dead at age 70 and 67 are larger than the difference in the probability of being dead at ages 67 and 64. But by regarding age as the time dimension, I can assume that in the absence of treatment, the mortality patterns by age would be similar in the different cohorts (more on this below). Including cohort dummies will also capture the time trends in mean cohort mortality. The parallel trends assumption is violated if there are calendar year effects that impact mortality in all cohorts in a given time period, or if cohort membership and age interact so that mortality at certain ages is higher in some cohorts relative to the cohort mean than in other cohorts.

I use the 1902 cohort as a control group for the age span 67-70, because this cohort was not eligible for old age pensions before they turned 70. The treatment group is the 1906 cohort because they are treated (eligible) throughout this age span. All the cohorts in between start off in the control group, but month by month, as the individuals age, one monthly birth cohort moves from the control group into the treatment group. It is the resulting between- and within-cohort variation in treatment status that I exploit. At age 70, everyone is treated, so there is no longer a control group. The grouping by birth cohorts allows for multiple groups with detailed variation in retirement timing. The fact that I have data on monthly eligibility status and whether each person is alive or dead means that I have multiple data points for each individual.

The DiD setup used here has four main components. The first is a dichotomous outcome variable, namely whether person \( i \) is dead or alive at age \( a \). This variable takes the value one if the individual is dead, missing if the person is non-resident, and zero otherwise. In other words, individuals are not censored at death. Rather, being dead is a state that I wish to estimate the probability of being in. I want to estimate the proportion who are dead at each age and in each cohort, and whether this proportion varies with retirement eligibility. This distinction is important. Censoring at death introduces an endogenous condition (conditioning on the outcome). If I censored at death, those who die at age 71 are excluded from the calculation of mortality at age 72. But if mortality at age 71 is affected by the reform, I do not wish to make such an exclusion, because it means that the mortality at age 72 would be calculated on an endogenously selected subsample. Therefore, for the purposes of causal inference, it is preferable to estimate the effect on the probability of being dead rather than the conditional probability of dying.
The next component is a set of group dummies that identify each group in the data set. I mainly rely on dummies for each monthly birth cohort, except for the 1902 control cohort. To reduce random variation in the reference/control group, I include only one dummy for the entire 1902 cohort. These dummies provide cohort fixed effects, meaning that the mean differences in mortality between the cohorts are removed. In one part of the analysis, I exchange the cohort fixed effects with individual fixed effects to account for all between-individual variation in mortality not captured by the cohort dummies. There is no reason to expect the results to change when individual fixed effects are introduced, but if the results do not differ, this will provide some confidence that the DiD estimator is performing as it should.

Another component is a set of age dummies that capture the mean probability of being dead at each age (absent of the treatment). I include one dummy for each age in months. If the parallel trends assumption is true, these dummies should serve to remove the age variation in mortality not related to treatment status or cohort membership, since they effectively subtract the mean probability of being dead at each age. Finally, the treatment variable takes the value one if person \( i \) is eligible for retirement at age \( a \), and zero otherwise. In addition, I include dummies on immigrant background and educational levels. Formally, what I estimate separately for each sex is the following linear probability model:

\[
p(\text{dead})_{iac} = \beta_1 + \beta_2 X_i + V_i + C_i + A_{ia} + \beta_3 T_{iac} + \varepsilon_i
\]

where \( i \) indexes individuals, \( a \) indexes age in months and \( c \) indexes monthly birth cohorts. \( X \) is a set of observed characteristics (namely, dummies for immigrant status and educational level), while \( V \) represents unobserved characteristics that may vary across individuals. \( C \) is a set of dummies for each monthly birth cohort, \( A \) is similarly a set of dummies for monthly age, and \( T \) is the treatment indicator. The \( \beta \)'s represent regression coefficients.

As can be deduced from the previous paragraph, I include fairly detailed control for time related variables. Including dummies for both birth month and age in months may appear excessive, but it increases the likelihood that the remaining variation in mortality is due to retirement eligibility. The reason why it is even possible to include dummies for both cohort and age is that I do not include their interaction. Ideally, the analyses should also include control for period-varying mortality effects (calendar year effects). Unfortunately, this is not feasible, since cohort membership, age and time are perfectly collinear. I therefore had to choose between controlling for age and controlling for time. Because of the reasons given above and because mortality varies more with age than with time, I chose the former. This
dilemma could be solved by introducing within-cohort variation in retirement eligibility in a difference-in-differences-in-differences (DiDiD) estimator. This would require identifying a group not affected by the reform. I have not been able to identify such a group. This is a potential weakness of this thesis, as the risk of bias by calendar year effects would be reduced if such a group was included.

In the DiD model described here, a potential source of bias is the unconventional censoring (or lack thereof) of individuals who die. People who die in the DiD model are not censored. Therefore, people who die before their retirement eligibility age are included in the calculation of the probability of being dead in the period after they would have reached this age (had they been alive). They cannot be excluded, because they contribute to estimating the mortality in the control group. Hence, those not at risk of dying because they are dead are included in the calculations. To check if this is a major issue, I also estimate DiD models with individual fixed effects (similar to the models presented here), but with censoring after death. In addition to being informative about the effect of eligibility on mortality, these models are a robustness check for the DiD models. This is because they essentially estimate the conditional probability of dying at each age for the population at risk (i.e. those who are resident and alive) and is not subject to the same censoring issues as the DiD model. On the other hand, it is sensitive to the fact that conditioning on being alive is endogenous. This is why I include these estimations as a robustness check and not as part of the main results.

Note finally that the DiD model and the hazard model are strongly related. The probability of being dead at each age $a$ is defined as the inverse of the survivor function $S$, and the survivor function is the product of the inverse age-specific mortality hazards in each previous age interval:

$$P(dead)_a = 1 - S_a = 1 - \prod_{s=1}^{a} (1 - h_s)$$

where $h$ is the hazard and the subscript $s$ is an indicator of time intervals until censoring at age $a$ (adapted from Rabe-Hesketh and Skrondal, 2008: 334-342).

### 7.4 Instrumental variables

In this thesis, I also want to estimate the effect of retirement age on mortality. In an ideal experimental setting, I would be able to manipulate and randomize the retirement age. In such
a case, an OLS regression would yield unbiased estimates of the causal effect of retirement age on mortality. In practice, however, the variation in retirement age is not random, but endogenous to mortality. Thus, what I have is a situation where I wish to estimate the causal effect of an endogenous regressor on an outcome. Simply regressing the outcome (death) on the endogenous regressor (retirement) will likely yield positively biased results. This is because retirement age is likely influenced by a number of factors that also impact mortality, such as health, occupation, income, sex etc. In other words, the endogenous regressor is likely correlated with the error term.

Instrumental variables (IV) estimation is a technique for dealing with such endogeneity. Some of the variation in an endogenous regressor may be due to variation in some third factor unrelated to the outcome of interest except through the endogenous regressor. In such a case, this exogenous variation in the endogenous regressor can be exploited to estimate its causal effect. In other words, I can attempt to isolate the variation in the endogenous regressor that is due to variation in the exogenous third factor (the instrument) but unrelated to the error term. This is usually done in a two-stage least squares (2SLS) model. In the first stage of the 2SLS model, the variation in the instrument is used to predict values on the endogenous regressor. This stage ensures that the variation in the (predicted) endogenous regressor is due to variation in the instrument. In the second stage, the predicted values of the endogenous regressor are used to estimate its causal effect on the outcome. Formally, this can be written as

\[
\begin{align*}
1) \quad X_i &= \alpha + \beta Z_i + \gamma C_i + \epsilon_i \\
2) \quad Y_i &= \theta + \mu \hat{X}_i + \rho C_i + \epsilon_i
\end{align*}
\]

where \(X\) is the endogenous regressor, \(Y\) is the outcome, \(\alpha\) and \(\theta\) are constant terms, \(Z\) is the instrument, \(\hat{X}\) denotes the predicted values for \(X\) for each individual \(i\). These values are given by estimating the first stage: \(\hat{X}_i = \alpha + \beta Z_i + \gamma C_i\). \(\beta\) and \(\mu\) are regression coefficients for \(Z\) and \(\hat{X}\), respectively. \(C\) is a set of control variables that are included in both stages, while \(\gamma\) and \(\rho\) are the regression coefficients for the \(C\)s in the first and second stage, respectively. \(\epsilon\) and \(\epsilon\) denote error terms (Angrist and Pischke, 2014: 98-146).

In this thesis, I use the 1973 pension reform as an instrument for retirement. More precisely, I use retirement eligibility age as an instrument for actual retirement age in order to estimate the effect of retirement age on the probability of dying before various ages. Variation in
retirement eligibility age stems from the reform, since the reform gave everyone born between January 1903 and December 1905 the right to retire in 1973, and everyone born in 1906 the right to retire at age 67.

There are two important assumptions made in an IV estimation. The first is that the instrument is relevant, meaning that it affects the endogenous regressor. This assumption is testable, and as I have shown above, the 1973 pension reform appears to have had a strong effect on actual retirement age in the population. The second assumption is that the instrument is valid. This requires that it is unrelated to other factors that may affect the outcome, meaning that the variation in the instrument is exogenous (the independence assumption), and that the only causal path between the instrument and the outcome is through the endogenous regressor (the exclusion restriction). This last assumption means that there should be no other causal channel connecting the instrument and the outcome (Angrist and Pischke, 2014: 98-146). Unfortunately, this assumption is not directly testable, but the researcher can provide theoretical and empirical arguments in support of it and discuss potential issues.

In this thesis, an important potential violation of the exclusion restriction stems from the fact that the retirement eligibility instrument is derived from birth cohort membership. This means that any link between birth cohort membership and mortality that does not go through the eligibility instrument may bias the results. For instance, if later cohorts were more or differently educated than earlier ones, and education affects mortality, I would have to adequately control for such educational differences in the models. In the next section, I discuss some possible sources of bias in the IV estimation.

IV estimators are known for producing fairly local average treatment effects. Imagine that a group of test subjects are part of an experiment. This experiment is not a double blind one. The test subjects know perfectly well whether or not they are given the treatment. Among those offered the treatment, most accept it, but some do not. In the control group, no one is offered the treatment. Therefore, most are untreated, but some manage to get hold of the treatment on their own, and take it anyway. Each test subject can then be seen to belong to one of four groups. The first is commonly referred to as the compliers. These comply with the treatment, meaning that they take the treatment if they are offered it, but do not take it otherwise. Second, the always-takers will always take the treatment, regardless of whether they are in the treatment group or the control group. Never-takers, on the other hand, never
accept the treatment, regardless of group membership. Finally the defiers are people who do not accept the treatment if they are offered it, but who take the treatment if they are not.

As mentioned before, having a large proportion of defiers in a sample strongly biases IV estimates (Borgen, 2013). In an IV estimation, the estimated effect is the average treatment effect for the compliers – the people who take the treatment because they are offered it, but who otherwise would not have taken the treatment. In other words, the effect is the effect on the people who were affected by the instrument (Angrist and Pischke, 2014: 98-146). While not intuitive, this makes sense when considering the fact that it is impossible to measure the effect of taking some treatment on someone who would never take it. Also, the effect on the compliers is the most policy relevant effect, since this group would be the one (along with the defiers) that would be affected by a reform.
8 Descriptive statistics and checks for balance

For the main event history analyses presented in chapter 9, the sample is restricted to individuals born in 1906 and 1907. For these analyses, I employ the long file in years described in chapter 6. For the IV and DiD analyses, I sample individuals born between January 1st 1902 and December 31st 1906 who were alive at age 65. With regard to analyses and statistics that include information on education and occupations, the samples are also restricted to individuals registered with this information in the 1970 census. While the DiD estimations utilize the long file in months, the IV analyses are based on the wide file. Both of these files are also described in chapter 6.

8.1 Descriptive statistics

Table 8.1 shows descriptive statistics for the two samples. All statistics are shown disaggregated by sex, since all analyses are done separately for men and women. All statistics are also shown conditional on being alive at age 65. This condition is included to avoid issues related to the fact that the registers contain information starting from 1967. Both samples have a slight majority of women. The number of people who immigrated or emigrated in their late 60s or later is very low. This means that censoring due to immigration and emigration is unlikely to be a major concern.

The number of immigrants in the samples is very low, as is the number of “others”. Therefore, I am forced to merge the “others”-group with the Norwegian-born with two Norwegian-born parents group in the discrete-time hazard models to ensure that the models converge. With regard to educational levels, there are few missing cases. The occupation variable has a lot of missing values – especially for women. This is the reason why I have chosen not to include it in my models and why I have decided to investigate effect heterogeneity with regard to education rather than occupation (the same argument applies to income).

Further, the vast majority received some kind of pension benefit, and the vast majority of those who did, received old age pensions. Many of those who did receive old age pensions also received some other type of pension first, such as a disability pension or a survivor’s pension. Relatively few people died or emigrated without having received any kind of pension benefit. It is also possible to discern from Table 8.1 that different pension types are related to different retirement ages. In the table here, mean retirement age is calculated for all groups, and then successively for more restricted subsamples by excluding people who also received other types of pensions. Note that measuring the mean retirement age for old-age pensions (as
plotted in Figure 7.3) and measuring the mean retirement age for old-age pensioners by excluding people who ever received other types of pensions gives two different estimates. This is because a lot of people first received some other type of pension, and later received an old age pension.

The mean death age conveys that, conditional on being alive at 65, the women and men in the sample on average lived for 18 and 14 more years, respectively. This is close to the actual remaining life expectancy in this period (SSB, n.a.). The standard deviations for these estimates show that there is a lot of variation in longevity.
Table 8.1: Descriptive statistics by sex for the two samples.

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<th>1906-1907 sample</th>
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<tr>
<td></td>
<td>Women</td>
<td>Men</td>
<td>Total</td>
<td>Women</td>
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<tr>
<td>Number in 1902 cohort</td>
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<td>17 656</td>
<td>37 699</td>
<td></td>
</tr>
<tr>
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<td>17 713</td>
<td>37 901</td>
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<td>Number in 1904 cohort</td>
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<td>37 707</td>
<td></td>
</tr>
<tr>
<td>Number in 1905 cohort</td>
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<td>17 442</td>
<td>37 731</td>
<td></td>
</tr>
<tr>
<td>Number in 1906 cohort</td>
<td>20 572</td>
<td>18 024</td>
<td>38 596</td>
<td>20 519</td>
</tr>
<tr>
<td>Number in 1907 cohort</td>
<td></td>
<td></td>
<td></td>
<td>20 784</td>
</tr>
<tr>
<td>Total number</td>
<td>101 361</td>
<td>88 273</td>
<td>189 634</td>
<td>41 303</td>
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<table>
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<tr>
<th></th>
<th>Number who immigrated between ages 65 and 70</th>
<th>Number who immigrated at ages 70+</th>
<th>Number who emigrated between ages 65 and 70</th>
<th>Number who emigrated at ages 70+</th>
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<td></td>
<td>116</td>
<td>204</td>
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<td>465</td>
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Percent in different immigrant categories

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<tr>
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<th>Norwegian-born with two Norwegian-born parents</th>
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<th>Others</th>
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<tr>
<td></td>
<td>97.46</td>
<td>2.44</td>
<td>0.10</td>
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Percent with different education levels from the 1970 census

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<th>None</th>
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<td>0.65</td>
<td>80.03</td>
<td>13.03</td>
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Percent in occupation groups from the 1970 census

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<tr>
<th></th>
<th>Technical, scientific, humanistic and artistic</th>
<th>Corp. and organiz. management, administration</th>
<th>Office and clerical work</th>
<th>Trade work and sales</th>
<th>Agriculture, forestry and fishing</th>
<th>Mining</th>
<th>Transport and communications</th>
<th>Industry and construction</th>
<th>Services</th>
<th>Military jobs</th>
<th>Missing</th>
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<td>2.40</td>
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<table>
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<td>73 252</td>
<td>19 038</td>
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Mean retirement age – all pensions

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<thead>
<tr>
<th></th>
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<th>Mean retirement age – excl. disability pensioners</th>
<th>Mean retirement age – incl. only old age pensioners</th>
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<td>67.32</td>
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Mean death age

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<th>Mean death age</th>
<th>Standard deviation</th>
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<td>8.33</td>
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<td>79.12</td>
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<td>81.20</td>
<td>8.31</td>
</tr>
<tr>
<td></td>
<td>79.23</td>
<td>8.37</td>
</tr>
</tbody>
</table>

Number of person-months, ages 65-70 in long file with months

<table>
<thead>
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<th></th>
<th>Number of person-months, ages 65-70 in long file with months</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>6 183 021</td>
</tr>
</tbody>
</table>

Number of person-years in long file with years

<table>
<thead>
<tr>
<th></th>
<th>Number of person-years in long file with years</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>797 116</td>
</tr>
</tbody>
</table>
8.2 **Between-cohort variation in some potential confounders**

The DiD identification strategy in this thesis does not hinge on a perfect balance between the different cohorts/treatment groups in the sample. It is sufficient to provide a convincing argument that the age-specific mortality trends in the different cohorts would run in parallel in absence of the treatment (the lowering of the retirement eligibility age).

For the IV estimation, however, it is important that the different cohorts are well-balanced with regard to potential confounders. Since the instrument is derived from monthly birth cohort membership, it is important that these cohorts do not differ systematically on properties that may affect mortality. Such differences would violate the independence assumption. For instance, if education affects mortality and varies systematically between the cohorts that are subject to different retirement eligibility rules, this would lead to biased estimates of the effect of retirement on mortality. In other words; the instrument would be invalid. Unfortunately, not all relevant variables are observable. Some variables, such as health behavior or childhood nutrition may vary between cohorts, but are not observed. Other variables may be observed (registered) but not included in the available datasets. In other words, I can never achieve full certainty about the validity of the instrument. What I can do is to check for balance on observed characteristics and perhaps find some reassurance if these do not vary systematically between cohorts. This means that systematic differences between cohorts on observables should be seen as grounds for concern. No differences, however, is not in itself a confirmation that everything is fine. It just means that no obvious concerns have been identified. Below, some proportions in the different yearly birth cohorts are shown.

The possibility of confounding by cohort-varying characteristics is an important reason why it is important to use both the IV and DiD approach. In a sense, the DiD-estimation can be seen as a robustness check for the IV estimation, since it takes advantage of the panel structure in the dataset and therefore controls for time-invariant but cohort-varying confounders such as sex, immigrant status, education, family background etc. But although I can never be sure, the fact that these cohorts are born close in time means that I see little reason to expect large differences between them on potentially important confounders. Also, such direct effects is a bigger issue if the instrument is weak than if it is strong. Perhaps somewhat prematurely, I can assure the reader that the instrument is very strong.

When looking at the figures below, it is worth noting that they are shown with spikes marking 95 % confidence intervals. The reason why the confidence intervals are not visible is that their...
width is narrower than the default line width in Microsoft Excel, leaving them both invisible and largely redundant. The reason for the different number of missing values associated with the different variables is that sex and immigrant background are taken from the population registers, income is taken from the tax registers, whereas educational level is taken from the 1970 census. The variables from the census data and the income variable have the most missing observations. Also note that the statistics shown are conditional on being alive at age 65.

From Figure 8.1a it is evident that the different cohorts do not vary with regard to sex composition. The proportions of men and women in each cohort are, for all practical purposes, the same. This also goes for the proportion of immigrants, as shown in Figure 8.1b. Only two groups – immigrants and people born in Norway with two Norwegian-born parents – are shown, because the proportions in the other three categories in the dataset (Norwegian-born children of immigrant parents etc.) are close to zero. As can be seen from Figure 8.1c, a slight trend towards increasing proportions with primary and secondary education can be observed through successively later cohorts. However, this trend is largely a result of the lower proportions with missing values in later cohorts. If these are excluded (Figure 8.1d), the trend in the proportion with only primary education is reversed and much weaker. Excluding missing values also reduces the trend in the proportion with secondary education.

Both with regard to immigrant composition and educational levels, the minor differences between the first and the last cohort are statistically significant. However, the differences are so small that the statistical significance may be attributed to the large sample sizes rather than to actual, substantial differences between the cohorts. Although substantially negligible, these differences between cohorts nevertheless mean that it may be important to control for potential confounders that can be thought to both vary with cohort membership and affect mortality in the IV estimation. It also means that one should be careful when interpreting the results, as there may be other biasing differences between the cohorts. All in all, however, the small differences with regard to sex, immigrant status and education suggest that there is no immediate cause for concern. Rather, this comparison of proportions suggests that the cohort-derived eligibility instrument is not systematically related to other characteristics.

A more pronounced issue arises from the differences between cohorts with regard to income. Having no pensionable income can be seen as a proxy for retirement. Figure 8.1e shows that a substantially lower proportion received no pensionable income at age 66 in the three latest
cohorts. However, income at age 66 is likely endogenous to the retirement reform. It may be a result of adaption to changing retirement opportunities. Let me explain briefly. For people who are eligible for both types of pension, disability pension and old age pension are substitutes. If you are soon to be 66 years old and become ill, you may apply for a disability pension or simply continue working until you become eligible for old age pension. If you are not going to become eligible for old age pension before you turn 70 years old, you may be more inclined to choose the former than if you will become eligible for old age pension at age 67. If having a pensionable income is indeed endogenous, or a collider variable (Elwert and Winship, 2014), I should not control for it even if it is unevenly distributed between cohorts. As can be seen from Figure 8.1e, the observed pattern is consistent with income at age 66 being endogenous to retirement eligibility, as lower proportions had zero income in the affected cohorts.
Figure 8.1a: Proportion women in each cohort\(^{16}\).

Figure 8.1b: Proportion Norwegian-born to Norwegian-born parents and immigrants in each cohort.

Figure 8.1c: Proportions with different educational levels in 1970 in each cohort, counting missing values.

Figure 8.1d: Proportions with different educational levels in 1970 in each cohort, excluding missing values.

Figure 8.1e: Proportion with non-zero pensionable income at age 66 in each cohort.

\(^{16}\) In these and later figures I have not been able to change the decimal separator from “,” to “.”. Sorry about that.
8.3 Parallel trends in the probability of being dead?

The DiD approach rests on the assumption that in the absence of differences in retirement eligibility, the cohort trends in mortality by age would be parallel. The counterfactual mortality trends of the cohorts in question cannot be observed, and therefore this assumption cannot be tested directly. What I can do is to check if the trends are parallel in the period before some of the cohorts were affected by the reform, and if the trends in the 1906 and 1907 cohorts, which were allowed to retire at the same age, are parallel throughout the age span in question.

Figure 8.2 plots the proportions who are dead at the beginning of each age (measured in months) by birth cohort. The numbers are estimated conditional on being alive at age 65. The cohort mortality trends in ages that are comparable are drawn in thick lines, while the trends are drawn in thin lines in periods where they are (presumably) not directly comparable due to differences in retirement eligibility. The 1906 and 1907 cohort become eligible at the same age (67 years/804 months), so these are comparable throughout the age span (up to 70 years/840 months). The other cohorts are only comparable in the period leading up to the earliest retirement eligibility (67 years/804 months). As can be seen from the figure, the trends are not perfectly parallel. However, the deviations are relatively small in the period up to age 67, and although the lines showing the 1906 and 1907 cohort mortality trends diverge slightly at the end of the period, they move closely together throughout the period. In other words, there appears to be close to parallel trends in the age span 65-67 for all cohorts and in the age span 67-70 for the 1906 and 1907 cohorts. But the fact that these trends are not perfectly parallel could bias the results. Therefore, one should interpret the results with due caution.
Figure 8.2: Proportions who are dead at the beginning of each age (in months) by birth cohort, conditional on being alive at age 65. Numbers on the X-axis refer to age in months.
9 Results

9.1 The association between retirement and mortality
Here, I present the main results from the event history analyses. As mentioned, all estimates are produced using discrete-time hazards models on censored panel data with one observation per person-year. Results for the variables of interest are presented as average marginal effects to facilitate comparisons across models.

Retirement age is not included as a covariate in these models. This is because people who die before they retire do not have a retirement age, so these people would not be included in the analyses if retirement age was included. Age and retirement status by duration are included as dummies, however. This means that information on people who do not retire is also included since they are counted as alive and not retired until they die.

9.1.1 The association between retirement duration and mortality
Hypothesis H1 states that mortality should be elevated among retirees relative to people not retired in the period following retirement. In the discrete-time hazards models presented in Table 9.1, death is regressed on dummies for age starting at 60, dummies for retirement duration starting at 0 for those not retired and continuing from 1 in the first year of retirement until top coding, and two time-invariant covariates (a dichotomous indicator for immigrant status and dummies for educational levels). To facilitate model convergence, ages above age 96 are top coded to 96, and retirement durations of more than 21 years are similarly coded to 21. The models are estimated for all pension types combined. For readability purposes, only the coefficients for retirement duration are shown. Because of the top coding, I only show results for durations up to 20 years.

As can be seen from Table 9.1 (the results are plotted in Figure 9.1), the probability of dying increases almost linearly with retirement duration even when age fixed effects are included to capture the baseline hazard. The average marginal effects are generally around two times as large for men as for women.

In other words, mortality is elevated and continually increasing among retirees relative to people not retired. This elevated mortality is in line with H1, but may have several explanations. One of these, as noted in the literature review by Shim et al. (2013), is that when disability pensioners are included, negative health selection into retirement through disability pensions may be a serious issue. The detailed data on pension types allows me to test whether this is the case.
Table 9.1: Average marginal effects from discrete-time hazard regression models of the probability of dying by retirement duration.

<table>
<thead>
<tr>
<th>Retirement duration (years)</th>
<th>Men</th>
<th></th>
<th>Women</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Average</td>
<td>Standard</td>
<td>Average</td>
<td>Standard</td>
</tr>
<tr>
<td></td>
<td>marginal</td>
<td>error</td>
<td>marginal</td>
<td>error</td>
</tr>
<tr>
<td>1</td>
<td>0.0222</td>
<td>0.0011</td>
<td>0.0124</td>
<td>0.0010</td>
</tr>
<tr>
<td>2</td>
<td>0.0260</td>
<td>0.0012</td>
<td>0.0140</td>
<td>0.0011</td>
</tr>
<tr>
<td>3</td>
<td>0.0325</td>
<td>0.0014</td>
<td>0.0174</td>
<td>0.0012</td>
</tr>
<tr>
<td>4</td>
<td>0.0422</td>
<td>0.0016</td>
<td>0.0210</td>
<td>0.0012</td>
</tr>
<tr>
<td>5</td>
<td>0.0476</td>
<td>0.0017</td>
<td>0.0214</td>
<td>0.0013</td>
</tr>
<tr>
<td>6</td>
<td>0.0535</td>
<td>0.0019</td>
<td>0.0276</td>
<td>0.0014</td>
</tr>
<tr>
<td>7</td>
<td>0.0629</td>
<td>0.0021</td>
<td>0.0292</td>
<td>0.0015</td>
</tr>
<tr>
<td>8</td>
<td>0.0764</td>
<td>0.0024</td>
<td>0.0361</td>
<td>0.0016</td>
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<tr>
<td>9</td>
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<td>0.0026</td>
<td>0.0395</td>
<td>0.0017</td>
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<tr>
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<td>0.0028</td>
<td>0.0456</td>
<td>0.0018</td>
</tr>
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<td>0.0568</td>
<td>0.0020</td>
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<td>0.0037</td>
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<td>0.0623</td>
<td>0.0022</td>
</tr>
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<td>15</td>
<td>0.1303</td>
<td>0.0042</td>
<td>0.0670</td>
<td>0.0023</td>
</tr>
<tr>
<td>16</td>
<td>0.1420</td>
<td>0.0046</td>
<td>0.0719</td>
<td>0.0024</td>
</tr>
<tr>
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<td>0.0788</td>
<td>0.0026</td>
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<td>0.0054</td>
<td>0.0803</td>
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<tr>
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<td>0.0811</td>
<td>0.0027</td>
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<tr>
<td>20</td>
<td>0.1696</td>
<td>0.0063</td>
<td>0.0868</td>
<td>0.0029</td>
</tr>
</tbody>
</table>

Age FE: yes, yes
Immigrant dummy: yes, yes
Educational levels: yes, yes

N (person-years) 743,501 1,007,991
-2LL -170,628.62 -192,028.75
P > Chi² 0.0000 0.0000
Pseudo R² 0.1271 0.1496

9.1.2 Distinguishing between pension types
Hypothesis H2 states that even if disability retirees are excluded, mortality should still be elevated among retirees relative to people not retired. I test this by excluding everyone who ever received disability pensions. The main results from discrete-time hazard regression models are presented as average marginal effects in Table 9.2. Even after excluding disability pensioners – a group where there is reason to expect strong health-related selection into retirement, retirement is still associated with elevated and continually increasing mortality. The patterns here are similar to those from Table 9.1, except that the increase in relative mortality seems to occur at a slower pace when we exclude disability pensioners.
Table 9.2: Average marginal effects from discrete-time hazard regression models of the probability of dying by retirement duration. Disability pensioners excluded.

<table>
<thead>
<tr>
<th>Retirement duration (years)</th>
<th>Men Average marginal effect</th>
<th>Men Standard error</th>
<th>Women Average marginal effect</th>
<th>Women Standard error</th>
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<td>0.0135</td>
<td>0.0019</td>
<td>0.0076</td>
<td>0.0021</td>
</tr>
<tr>
<td>2</td>
<td>0.0167</td>
<td>0.0021</td>
<td>0.0074</td>
<td>0.0021</td>
</tr>
<tr>
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<td>0.0024</td>
<td>0.0101</td>
<td>0.0021</td>
</tr>
<tr>
<td>4</td>
<td>0.0328</td>
<td>0.0028</td>
<td>0.0119</td>
<td>0.0022</td>
</tr>
<tr>
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<td>0.0115</td>
<td>0.0021</td>
</tr>
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<td>0.0032</td>
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<td>0.0023</td>
</tr>
<tr>
<td>7</td>
<td>0.0493</td>
<td>0.0036</td>
<td>0.0169</td>
<td>0.0022</td>
</tr>
<tr>
<td>8</td>
<td>0.0619</td>
<td>0.0041</td>
<td>0.0198</td>
<td>0.0023</td>
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<tr>
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<td>0.0206</td>
<td>0.0023</td>
</tr>
<tr>
<td>10</td>
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<td>0.0236</td>
<td>0.0023</td>
</tr>
<tr>
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<td>0.0273</td>
<td>0.0024</td>
</tr>
<tr>
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<td>0.0818</td>
<td>0.0051</td>
<td>0.0299</td>
<td>0.0024</td>
</tr>
<tr>
<td>13</td>
<td>0.0949</td>
<td>0.0057</td>
<td>0.0317</td>
<td>0.0025</td>
</tr>
<tr>
<td>14</td>
<td>0.0926</td>
<td>0.0059</td>
<td>0.0313</td>
<td>0.0024</td>
</tr>
<tr>
<td>15</td>
<td>0.0887</td>
<td>0.0059</td>
<td>0.0313</td>
<td>0.0025</td>
</tr>
<tr>
<td>16</td>
<td>0.1056</td>
<td>0.0068</td>
<td>0.0348</td>
<td>0.0025</td>
</tr>
<tr>
<td>17</td>
<td>0.1180</td>
<td>0.0076</td>
<td>0.0388</td>
<td>0.0026</td>
</tr>
<tr>
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<td>0.1192</td>
<td>0.0080</td>
<td>0.0384</td>
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</tr>
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<td>0.1139</td>
<td>0.0082</td>
<td>0.0364</td>
<td>0.0026</td>
</tr>
<tr>
<td>20</td>
<td>0.1167</td>
<td>0.0088</td>
<td>0.0386</td>
<td>0.0027</td>
</tr>
</tbody>
</table>

Age FE: yes, yes
Immigrant dummy: yes, yes
Educational levels: yes, yes

N (person-years) | 557 862 | 824 547
-2LL             | -122 933.97 | -150 596.62
P > Chi²         | 0.0000     | 0.0000
Pseudo R²        | 0.1296     | 0.1605

The patterns become clear when the results from Tables 9.1 and 9.2 are plotted in Figure 9.1 (spikes indicate 95% confidence intervals). For both men and women, there appears to be a strong selection effect that is considerably reduced when disability pensioners are excluded. But the association between retirement and mortality is still present and strong. Thus, both H1 and H2 are largely supported by the data.

The fact that the association between retirement and mortality continues to be strong after excluding disability pensioners is in line with findings reported by Shim et al. (2013). This may reflect a causal relationship or some selection process – or both. It is worth noting, though, that at higher ages (and longer durations) those not retired (and presumably still working) become an increasingly positively selected group with regard to health. This is what
is commonly referred to as the healthy worker effect. This effect would suggest that a strong selection process into retirement is driving these results at longer durations. In the following sections, I attempt to disentangle the causal relationship between retirement and mortality by exploiting the 1973 pension reform.

Figure 9.1: Average marginal effects by retirement duration from discrete-time hazard models of the probability of dying. Including and excluding disability pensioners.

9.2 The effect of retirement eligibility on mortality
For the age span 67-70 years, I can estimate the average effect of being eligible for retirement using a DiD estimator. This should provide some information about the direction of the effect (H3 though H4) and the relative strength of the effect among women and men (H6) in the short run. In Table 9.3, two such estimates are given for each sex. In models 1 and 3, the controls include immigrant status, educational level dummies, monthly birth cohort fixed effects (the 1902 reference cohort is pooled) and age fixed effects in months. This is the panel data adaption of the standard DiD estimator described above. In model 2 and 4, individual fixed effects are included together with age fixed effects, while the time-invariant controls are omitted. These individual FE models are essentially a different specification of the DiD model, using a more fine-grained control for time-invariant unobserved variables. All models are conditioned on being alive and present at age 65, and individuals are followed from age 65 (or immigration if they immigrate later) to age 70 (or emigration if they emigrate earlier) regardless of whether they are alive or dead.
The two model types give identical results. This is good news, but should not be a surprise, as the two estimators essentially measure the same thing. In both model types, retirement eligibility is estimated to increase the average probability of being dead within the age span 67 to 70 by about 2.7 percentage points among men and 1.4 percentage points among women. These effect estimates are large enough to be substantively meaningful, especially if the effect is driven by actual retirement (the compliers), so that the estimates provided here measure something akin to a reduced form effect. Below, I run a robustness check to test if the estimates are sensitive to the unconventional (lack of) censoring.

Table 9.3: Difference-in-differences and individual fixed effects estimates of the effect of retirement eligibility on the probability of being dead in the age span 67-70 years.

<table>
<thead>
<tr>
<th></th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Model 1</td>
<td>Model 2</td>
</tr>
<tr>
<td>Coefficient</td>
<td>(Robust SE)</td>
<td>Coefficient</td>
</tr>
<tr>
<td>Being eligible for retirement</td>
<td>0.0273 (0.0003)</td>
<td>0.0273 (0.0002)</td>
</tr>
<tr>
<td>Educational levels</td>
<td>yes</td>
<td>no</td>
</tr>
<tr>
<td>Immigrant</td>
<td>yes</td>
<td>no</td>
</tr>
<tr>
<td>Monthly age FE</td>
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<td>yes</td>
</tr>
<tr>
<td>Monthly birth cohort FE</td>
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<td>no</td>
</tr>
<tr>
<td>Individual FE</td>
<td>no</td>
<td>yes</td>
</tr>
<tr>
<td>Person months</td>
<td>5 080 002</td>
<td>5 073 948</td>
</tr>
<tr>
<td>N (individuals)</td>
<td>83 190</td>
<td>97 342</td>
</tr>
<tr>
<td>P &gt; F</td>
<td>0.0000</td>
<td>0.0000</td>
</tr>
<tr>
<td>$R^2$ / $R^2$ overall</td>
<td>0.0423</td>
<td>0.0357</td>
</tr>
</tbody>
</table>

9.2.1 The effect of retirement eligibility duration – is there a honeymoon?

In accordance with the mortality interpretation of Atchley’s process theory (H5), mortality rates should be expected to fluctuate so that the probability of being dead first drops following retirement, and then increases towards (or even beyond) zero. The results from the DiD and individual FE-estimators above seem to contradict this, but they may not tell the full story. The relatively coarse model specification averages the effect across a three-year age span. This may conceal minor short-term fluctuations in mortality immediately following retirement if the reduction in mortality rates in the honeymoon phase is small relative to the subsequent increase.
One way to test this is to substitute the dichotomous treatment indicator in the DiD model for a set of dummies measuring the number of months that have passed since individual $i$ became eligible for retirement. Again, I cannot use actual retirement behavior as a treatment in this setup, but as I have shown, the reform had such a strong effect on actual retirement that it may be used as an exogenous proxy. In a sense, then, these analyses also measure something akin to a reduced-form effect.

For readability, the results from this analysis are plotted in Figure 9.2. Vertical spikes mark 95% confidence intervals. The effect apparently increases almost linearly with time. In other words, there is no support to be found for a honeymoon effect with regard to mortality, and H5 can be safely rejected. The fact that the lines do not start at zero is an issue, but may be attributable to the reform also indirectly affecting retirement at earlier ages. Alternatively, it may be that the treatment indicator captures some between-cohort differences in mortality.

Figure 9.2: Plot of the estimated effect of being eligible for retirement for a given number of months on the probability of being dead in the age span 67-70 years.

9.3 The effect of retirement age on mortality
Table 9.4 shows separate OLS and IV models for men and women for the effect of retirement age on the probability of dying before age 80. The dataset used for these analyses contains only one observation per individual (the wide file). The estimates are conditional on being alive at age 65. This condition hardly affects the results. The regression coefficients from the OLS models indicate that retiring one year later is associated with a 4.2 percentage points reduction in the probability of dying before age 80 among men and a 2.2 percentage points
reduction among women. This is a substantial effect, especially for men, and also if we take into account the fact that variation in retirement age may be much larger than one year.

The IV estimates show that the first stage is strong. The F-statistics are much larger than the conventional minimum of 10 (Angrist and Pischke, 2014: 145). Becoming eligible for retirement one year earlier is associated with retiring approximately three quarters of a year earlier. In the second stage estimates, there is a statistically significant effect of retirement age on mortality, but the coefficients are much smaller than in the OLS models. After self-selection into retirement is (hopefully) accounted for, the estimates show that retiring one year later causes a reduction in the probability of dying before age 80 of 1.5 percentage points among men and 0.5 percentage points among women. In other words, the OLS estimates are strongly biased, likely due to health related selection into early retirement. This bias is apparent despite the fact that these OLS models are estimated on a sample where a large proportion of the variation in retirement age is presumably exogenous. The causal effect of retirement on mortality appears to be relatively small, but if the relationship is linear, the effect of retiring three years earlier would still be substantial. The direction and magnitude of the effects are consistent with the results from the DiD and individual FE models presented in Table 9.3.

Table 9.4: OLS and IV results for the effect of retirement age (in years) on the probability of dying before age 80.

<table>
<thead>
<tr>
<th></th>
<th>Men</th>
<th></th>
<th>Women</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>OLS</td>
<td>IV</td>
<td>OLS</td>
<td>IV</td>
</tr>
<tr>
<td></td>
<td>Coefficient (Robust SE)</td>
<td>Coefficient (Robust SE)</td>
<td>Coefficient (Robust SE)</td>
<td>Coefficient (Robust SE)</td>
</tr>
<tr>
<td>First stage</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Eligibility age (years)</td>
<td>0.7497 (0.0067)</td>
<td>0.7726 (0.0069)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Educational level</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
</tr>
<tr>
<td>Immigrant</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
</tr>
<tr>
<td>F-statistic</td>
<td>3010.09</td>
<td>2402.46</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adjusted R^2</td>
<td>0.1680</td>
<td>0.1243</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Second stage</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Retirement age (years)</td>
<td>-0.0420 (0.0007)</td>
<td>-0.0147 (0.0020)</td>
<td>-0.0220 (0.0006)</td>
<td>-0.0047 (0.0017)</td>
</tr>
<tr>
<td>Education level</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
</tr>
<tr>
<td>Immigrant</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
</tr>
<tr>
<td>N = 79 397</td>
<td></td>
<td></td>
<td>N = 95 624</td>
<td></td>
</tr>
</tbody>
</table>
In sum, the results presented in Table 9.4 together with those in Table 9.3 and Figure 9.2 point toward an effect of retirement akin to those suggested by either H3a or H3b. H6 also receives strong support; the effect appears to be between two and three times as strong for men as for women. There is no support to be found for H4a or H4b; apparently, retirement or early retirement does not reduce mortality but increases it. But it is still interesting to know if the effect is long-term (H3a) or short-term (H3b).

9.3.1 IV estimates of the effect of retirement age on the probability of dying before different ages

Age 80 is an arbitrarily chosen age of measurement. It may very well be the case that the effect is larger or smaller in the short or in the long term. To test this, I have estimated one IV model identical to those in Table 9.4 for each sex and at each age between 71 and 100 years. This allows me to examine both the short-term and the long-term effects of retiring earlier or later. Unfortunately, the results cannot be displayed in relation to retirement age as would be required to see the duration effects discussed above directly. But some conclusions can nonetheless indirectly be drawn about the duration effects.

Figure 9.3 plots the IV point estimates of the effect of retiring one year earlier on the probability of dying before different ages for men and women, with corresponding 95% confidence intervals. All estimates are conditioned on being alive at age 65. Notice that the first stages are not shown. The reason is that they are identical to the ones in Table 9.4. As can be seen from the figure, the effect appears to be larger at younger ages and to attenuate as time passes by, until it reaches a precisely estimated zero. This is perhaps not surprising since the probability of being dead approaches 1 as age increases, so that there is little room for retirement effects at older ages. Nevertheless, the effect estimates are actually quite large at younger ages. This could indicate a rather strong short-term effect of retirement on mortality. In sum, the results appear to favor H3b (predicting a short-term effect) over H3a (predicting a lasting effect). But the pattern plotted in Figure 9.3 may be an artefact of the probability of being dead approaching 1 at higher ages. In order to really test this properly it would be preferable to estimate age-specific mortalities. I return to this point below. I will also discuss some precautions that should be taken when interpreting the results and report the results from a robustness check.
9.3.2 IV estimates of age-specific mortality

To properly test H3b it would be preferable to estimate the effect of retirement age on age-specific mortality. Using the IV setup in the previous section with some modifications, it is possible to do this. For this analysis, I condition on being alive at the beginning of each age in years and estimate the effect of retiring one year later on the probability of being dead at the end of that age. Now, this condition is obviously endogenous. If the probability of dying at age 72 is affected by retirement, then the probability of dying before age 72 is likely also affected by retirement and so on. Therefore, the results should be interpreted with caution. Nevertheless, the approach presented here provides conditional age-specific probabilities of dying, and if they tell the same story as the numbers presented in Figure 9.3, they may still inform on the age specific mortality effects that drive the results. For this analysis, I also look at lower ages (and thus do not condition on being alive at age 65), but have only included ages up to 89. Note that this approach does not properly account for emigration and immigration. That is likely a minor issue here, though.

Figure 9.4 plots the effect of retiring one year later on age-specific probabilities of dying, together with 95% confidence intervals. There is see a clear and precisely estimated effect of retirement age on age-specific mortality for ages up to 71 years. In other words, postponed retirement appears to lower the probability of dying in the short run. Inversely, earlier retirement increases mortality in the short run. At higher ages there is a lot of apparently
random variation and no evident effect. The large confidence intervals at higher ages reflect the successively smaller samples of people still alive. The fact that the point estimates tend to be more positive in this period (particularly for women) may be driven by the fact that if more people in the early retirement group die early, the remaining members in this group may be more positively selected and thus have a lower probability of dying at each age. The fact that an effect can be identified at ages below the earliest retirement eligibility (age 67) is not necessarily problematic, since the reform apparently also affected early retirement through disability pensions (as shown in Figure 7.3). In sum, and taken together with the results in Figure 9.3, this analysis provides support for H3b. Retirement apparently causes a relatively short-term increase in mortality. The attenuation in the effect appears not to be primarily driven by the limited room for effects at higher ages due to mortality approaching 1. But in the next chapter, I will discuss an important caveat to this interpretation.

Figure 9.4: The effect of retiring one year later on the age-specific probability of dying.

![Figure 9.4](image)

9.4 Does the effect of retirement on mortality follow an educational gradient?

Since retirement evidently increases mortality, hypothesis H7 states that earlier retirement should increase mortality less for people with low education than for people with higher education. With reversed signs, this equates to later retirement reducing mortality less for people with low education. To test this, I have estimated separate IV models for each sex and
each education level group, except those with no education. The outcome is the probability of dying before age 75. The results from this analysis are given in Table 9.5, and the evidence for H7 is mixed.

For men, retiring one year later is estimated to reduce the probability of dying before age 75 by 2.5 percentage points among those with only primary education. The corresponding estimates are 1.9 percentage points for those with secondary education and a non-significant 0.2 percentage points for those with higher education. In other words; for men, the educational gradient in the effect of retirement on mortality goes in the opposite direction of the pattern postulated in H7.

Among women, the numbers tell a different story. Postponed retirement is estimated to reduce mortality more among women with higher education than among women with lower education. The reduction in mortality before age 75 is estimated to one percentage point among those with primary education, 1.4 percentage points among those with secondary education and a less precisely estimated 1.7 percentage points for those with higher education. But note that the confidence interval for the effect estimate among the women with secondary education encloses the estimates for the other two groups. In sum, H7 receives some support among women but not among men.

Table 9.5: IV results for the effect of retirement age (in years) on the probability of dying before age 75 for each sex and educational level.

<table>
<thead>
<tr>
<th></th>
<th>Primary</th>
<th>Men</th>
<th>Uni./college</th>
<th>Women</th>
<th>Uni./college</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Coefficient (Robust SE)</td>
<td>Coefficient (Robust SE)</td>
<td>Coefficient (Robust SE)</td>
<td>Coefficient (Robust SE)</td>
<td>Coefficient (Robust SE)</td>
</tr>
<tr>
<td>First stage</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Eligibility age</td>
<td>0.7937 (0.0080)</td>
<td>0.6577 (0.0144)</td>
<td>0.4636 (0.0191)</td>
<td>0.7906 (0.0077)</td>
<td>0.6900 (0.0176)</td>
</tr>
<tr>
<td>Immigrant</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
</tr>
<tr>
<td>F-statistic</td>
<td>3257.50</td>
<td>694.66</td>
<td>619.87</td>
<td>3494.70</td>
<td>515.00</td>
</tr>
<tr>
<td>Adjusted R²</td>
<td>0.1371</td>
<td>0.1283</td>
<td>0.1056</td>
<td>0.1118</td>
<td>0.1025</td>
</tr>
<tr>
<td>Second stage</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Retirement age</td>
<td>-0.0248 (0.0019)</td>
<td>-0.0186 (0.0048)</td>
<td>-0.0018 (0.0105)</td>
<td>-0.0099 (0.0014)</td>
<td>-0.0137 (0.0036)</td>
</tr>
<tr>
<td>Immigrant</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
</tr>
</tbody>
</table>

N = 60 156 N = 13 683 N = 5 139 N = 79 544 N = 12 925 N = 2 508
9.5 Robustness checks
The results presented above may be sensitive to model specifications, the way the data has been organized, or may simply be artefacts of confounding trends or unobserved differences between groups. I therefore run a couple of robustness checks to test some of the objections that may be raised about the validity of the findings.

9.5.1 Are the DiD results driven by the unconventional censoring?
One might interject that the results in the DiD model may stem from the unconventional (lack of) censoring. The models measure the proportion dead or the probability of being dead (the state) rather than the probability of dying (the event). In the absence of censoring at death, the results may be driven by deaths occurring before the treatment. This can be tested by estimating the DiD models with censoring at death. Table 9.6 displays the main results from such models. As can be seen, the results here are consistent with those from the DiD estimator. The small coefficient sizes reflect the fact that these models estimate the additional conditional probability of dying at each monthly time interval for those eligible for retirement. In sum, these models show that the unconventional censoring is unlikely to affect the direction of the results. But keep in mind that these results are estimated with the endogenous condition that only people alive at each time interval are included, so they should not be given a causal interpretation.

Table 9.6: DiD-estimations with individual fixed effects on censored data of the effect of retirement eligibility on dying in each monthly age interval between ages 67 and 70.

<table>
<thead>
<tr>
<th></th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Coefficient (Standard error)</td>
<td>Coefficient (Standard error)</td>
</tr>
<tr>
<td>Being eligible for retirement</td>
<td>0.0004 (0.0001)</td>
<td>0.0001 (0.0000)</td>
</tr>
<tr>
<td>Monthly age FE</td>
<td>yes</td>
<td>yes</td>
</tr>
<tr>
<td>Individual FE</td>
<td>yes</td>
<td>yes</td>
</tr>
<tr>
<td>Person months</td>
<td>4 865 721</td>
<td>5 814 972</td>
</tr>
<tr>
<td>N (individuals)</td>
<td>83 317</td>
<td>97 456</td>
</tr>
<tr>
<td>P &gt; F</td>
<td>0.0000</td>
<td>0.0000</td>
</tr>
<tr>
<td>$R^2$ within</td>
<td>0.0024</td>
<td>0.0012</td>
</tr>
<tr>
<td>$R^2$ between</td>
<td>0.6426</td>
<td>0.6475</td>
</tr>
<tr>
<td>$R^2$ overall</td>
<td>0.0004</td>
<td>0.0002</td>
</tr>
</tbody>
</table>
9.5.2 Are the IV results driven by cohort trends?
The instrument in the IV analyses is derived from cohort membership. This means that cohort trends in unobserved characteristics that affect mortality would constitute a violation of the exclusion restriction. This would lead to biased IV estimates. One example of such a violation is if birth year affected childhood nutrition, which may again affect mortality. As mentioned in section 7.2.1, there is also a cohort trend in retirement age which may affect the results. The DiD models are not very sensitive to such trends, since they do not condition on uptake and include cohort fixed effects. To test whether cohort trends drive the results, I have estimated IV models similar to those presented in Table 9.4, but including a linear term for birth year. To capture the nonlinearity in the relationship between mean pension age and cohort, I also included the 1907 cohort in these models. The results show that the effect estimate for men increases (to -0.0223), but that it is no longer statistically significant (P = 0.065). The effect estimate for women also increases (to -0.0254), but so does the p-value (P = 0.057). Apparently, the effect is not driven by a linear cohort trend, but suppressed by it. But controlling for such a trend greatly reduces statistical power. The reduction in power is not surprising given the strong correlation between eligibility age, retirement age and cohort. In other words, this analysis does not give good reason to distrust the main results.
10 Discussion

The results presented in the previous chapter are complex and their interpretation requires some clarification and context. Therefore, I here provide a discussion of the results, their potential interpretations and implications, and how they relate to the established research literature. I then suggest some angles and topics for future research.

10.1 How is retirement associated with higher mortality?

Looking at the results presented in the previous chapter, it is possible to draw several conclusions about the association between retirement and mortality. First, retirement is associated with elevated and continually increasing mortality risk. Comparing retirees with workers shows large and diverging differences in mortality between the two groups over time. This result is in accordance with the theoretical expectation that regardless of the causal effect of retirement on mortality, strong and negative health selection into retirement should result in higher risk among retirees. In other words, hypothesis H1 received strong support.

Because disability pension is a common entry into retirement for people with poor health, it is reasonable to assume that at least some of the association between retirement and mortality is driven by negative health selection through disability pensions. However, there is also good reason to assume that even if disability pensioners are excluded, the health selection effect would still be strong. This assumption is particularly plausible if one considers the fact that those not retired at higher ages and past earliest retirement age are increasingly positively selected. The exclusion of disability retirees from the sample supports this assumption, and thus supports H2. Excluding disability pensioners markedly weakens the association between retirement and mortality, but does not remove it.

The fact that the results from the event history analyses showed a strong and increasing association between retirement and mortality, while the IV estimation showed a small and short-term effect, suggests that the event history results are highly biased and primarily driven by some strong selection process. The fact that there was no apparent spike in mortality following retirement (keeping in mind that age was measured in years at integer values) further suggests that this process is likely not primarily an effect of terminally ill persons retiring in anticipation of death. Rather, a healthy worker effect and/or an omitted variable
bias\(^{17}\) seem to be the most plausible explanations for the observed strong and increasing association. But as the DiD, individual FE and IV results show, some of the observed association appears to be due to a causal effect.

### 10.2 The effect of retirement on mortality – drawing a detailed picture

In the analyses in chapter 9, I attempt to draw a very detailed picture of how retirement affects mortality over time. The fact that analyses with such a fine-grained time scale are even possible is in itself a testimony to the quality and detail of the Norwegian register data. The possibility of estimating the effect in the long term up to the age of 100 was made possible because using the 1973 reform allowed for a long observation window. The need for such detailed analyses of the temporal pattern of the retirement effect arose from the theoretical discussions. As I have shown, a number of theories predict different effects of retirement on mortality with regard to both direction and temporal profile. But the differences in the predictions are both intricate and subtle. Exploiting the detailed information on time variations in retirement eligibility, retirement and mortality allows for a deductive approach, whereby the hypotheses that do not fit the results are abandoned.

The results are relatively clear, and are summarized in Table 10.1. The DiD and individual FE results show that being eligible for retirement in itself increases mortality risk, most likely through retirement. This gives reason to reject H4a and H4b. A similar analysis with treatment duration dummies shows that there is apparently no honeymoon effect with regard to mortality following retirement. Thus, it is safe to reject H5 as well. The IV estimations support the main findings from the DiD and individual FE models. They also show that the effect is apparently a short-term one. This last finding favors H3b over H3a. But this last conclusion requires a few reservations (discussed below).

These findings are consistent with theories that predict short-term changes in health behavior following retirement, theories postulating some protective effect of employment, and theories that conceptualize retirement as a stressful life event or a continuity break. All analyses show that the effects of retirement eligibility or retirement are much larger for men than for women – between two and three times as large. This supports H6. But it should also be noted that the observed differences between men and women may be strongly related to the gender roles

\(^{17}\) The omitted variable may be preferences, personality traits, genetic inheritance, occupation or other factors affecting both health and proneness to retire early.
that characterized the cohorts studied here. As men and women have become more equal over time, it is likely that such gender differences have attenuated.

Finally, there appears to be some effect heterogeneity along an educational gradient. For men, the reduction in mortality resulting from postponed retirement appears to be larger among those with lower education, while the reverse appears to be true for women. This means that H7 can be soundly rejected for men, but that this hypothesis receives some support among women. These heterogeneous effects are consistent with retirement affecting mortality by inducing a discontinuity or role loss, if the role as a worker or employee is most important among low-educated men and highly educated women. This mechanism is not implausible given the gender roles in the generation under study. Aging men in the 1970s with low education may be expected to be more invested in their traditional breadwinner role. Similarly, highly educated women born around the turn of the century may be a very select group, and they may have not adhered to traditional gender norms but had a strong personal attachment to their work. But this is merely post-hoc speculation and should not be given too much weight.

The conclusion that the effect of retirement on mortality is short-term should be drawn with some caution. Its validity really boils down to whether the effect is one of retirement age, retirement duration or retirement status. It is possible to imagine three different types of effects of retirement. One is that being retired affects mortality, regardless of the age at which you retired or the duration for which you have been retired. This effect would simply entail a shift in the mortality rate curve. On the other hand, it is possible that retirement duration is most important, either because retirement causes a short-term bump in the mortality rates, or because retirement changes the slope of the increase in mortality risk with age. Third, retirement age may be important (perhaps in interaction with the two other effects). This would mean that the age at which you retire matters, over and above the effect of retirement status and duration.
**Table 10.1: Summary of the support for the different hypotheses.**

<table>
<thead>
<tr>
<th>Hypothesis</th>
<th>Supported</th>
<th>Not supported</th>
</tr>
</thead>
<tbody>
<tr>
<td>H1: The mortality of retirees is higher in the period following retirement, relative to people who have not retired.</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>H2: This holds true even if disability retirees are excluded.</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>H3a: Retirement causes a lasting increase in mortality rates.</td>
<td></td>
<td>X</td>
</tr>
<tr>
<td>H3b: Retirement causes a short-term increase in mortality rates.</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>H4a: Retirement causes a lasting reduction in mortality rates.</td>
<td></td>
<td>X</td>
</tr>
<tr>
<td>H4b: Retirement causes a short-term reduction in mortality rates.</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>H5: Retirement causes an initial reduction in the age and duration specific mortality rates, followed by an increase.</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>H6: If retirement increases mortality risk, the effect is stronger for men than for women.</td>
<td></td>
<td>X</td>
</tr>
<tr>
<td>H7: If retirement affects mortality risk, earlier retirement reduces mortality more, or increases it less, for people with low education, relative to people with higher educational levels.</td>
<td>Women</td>
<td>Men</td>
</tr>
</tbody>
</table>

This distinction is important because the DiD model measures the effect of retirement eligibility status and retirement eligibility duration in the short run by comparing those eligible to the control group who are not. But it can neither inform about the effect of retirement age nor the long-term effect. The IV model gives information on the effect of retirement age on age-specific mortality, but it is not informative about the effect of retirement status or duration because there is no control group that has not retired. The fact that the IV model finds effect of retirement age on mortality at low ages may be attributable to retirement age having a short-term effect. But about equally plausible is the interpretation that this effect is driven by differences in retirement status between cohorts affected and not affected by the reform in the age span that the reform affected. The attenuation of the effect after age 71 could then simply be due to the fact that by this age, virtually everyone has retired, so that those who retired later start “catching up”\(^\text{18}\). Using the identification strategy chosen in this thesis, it is difficult to be sure what effect is the true one. I have chosen to interpret the results as supportive of a short-term effect. But I acknowledge that seeing the results as a retirement status effect or a retirement age effect are plausible interpretations of the findings.

\(^{18}\) To clarify, a thought experiment may be useful. Let us imagine an extremely stingy welfare state (or one with a penchant for randomization) flipping a coin to decide who may retire at age 70 and who may not retire before age 80. And let’s say that we observe similar effects as those reported here throughout the entire 10-year age span. Would the differences in mortality then be driven by some being retired and others not being retired, or by some retiring at an earlier age than others?
The implications of the different effects also vary. If the effect is one of retirement status or retirement age, reductions in retirement age would expose those who retire earlier to elevated mortality risk for a longer duration. This would lead to more people dying at all subsequent ages. A short-term duration effect would not increase mortality at all ages, but simply shift the bump in mortality risk to lower ages. In other words, the short-term duration effect is the less dramatic one.

Keeping these reservations in mind, there are a couple of things that may be stated quite firmly. Retirement increased mortality somewhat in the context and period studied here. And there is apparently no honeymoon effect with regard to mortality.

In addition to the main results, this thesis also draws attention to the importance of institutions, policies and welfare state programs and their profound impact on people’s lives. As discussed previously, being retired may be a social role, but it is a role that is strongly influenced by the institutional context. By allowing people to retire earlier, the welfare state enabled a large-scale shift in the timing of people’s work exits. The reason why the IV analyses presented here were even possible is that people respond to such changes in welfare state regulations. In broader terms; people adapt their behavior to changing opportunity structures, and those opportunity structures are strongly shaped by welfare state institutions such as the national insurance scheme.

10.3 The research literature and the importance of context

How do the results fit into the research literature? As I have shown in chapter 3, there is more or less consensus that retirement is associated with elevated mortality risk. The results from the event history analyses are very much in line with these findings and the conclusions drawn by Shim et al. (2013). The finding that excluding disability pensioners reduces the estimated association for both women and men highlights the importance of distinguishing between pension types. The fact that the results generally differ between the sexes also show the importance of being sensitive to differences between men and women in the analyses. The finding that most of the association between retirement and mortality is not causal but likely driven by some selection process shows how important it is not to interpret associations as reflecting causal processes. Without adequately dealing with selection bias, one runs the risk of overstating or otherwise misstating an effect.
With regard to the estimates of the causal effects of retirement on mortality, these results partly contradict previous findings. Bingley and Pedersen (2011) found that early retirement led to reduced mortality before age 80. Bloemen et al. (2013) and Hallberg et al. (2015) also found earlier retirement to reduce mortality. Coe and Lindeboom (2008) and Hernæs et al. (2013) found no effect. Only one study, by Kuhn et al. (2010), has presented findings that are similar to those presented here. One plausible explanation for the inconsistent findings in the research literature might be that the contexts, samples and methods differ. Societies may differ in many aspects that might condition the effect of retirement on mortality. Such differences may be found with regard to for instance culture, pension schemes, labor markets, welfare services, or health systems. Also, societies change, so that even within the same national contexts, the effects of retirement may change over time. This could be driving the difference in conclusions drawn by and Hernæs et al. (2013) and those presented here. Also, different studies have studied different occupational groups and age groups. If the effects vary with occupation and age, one should perhaps not be surprised that the findings diverge.

A closer look at samples, research designs, age groups and country and period contexts in these studies does not give any clear clue as to why the results are contradictory, though. Nor does it aid in the understanding of why the results in this thesis contradict most of them. Table 10.2 compares the studies mentioned above and the present thesis with regard to country contexts, samples, time periods, ages, main methods employed and the direction of the main results. There does not appear to be any clearly identifiable pattern. Apparently, context may matter, but not in any predictable way.

Also, this thesis does not tip the balance in favor of one conclusion. If anything, it adds to the confusion by drawing the conclusion that was least frequently found in the established literature. Among the studies presented here, three found retirement to reduce mortality, two found no effect, while one found retirement to increase mortality (3/2/1). Adding the present thesis to the summary changes the score to 3/2/2.

19 Let me be absolutely clear: although I point out that the research design used in this thesis has some advantages, I readily acknowledge that the design employed by Hernæs et al. (2013) is better in some regards. Particularly, their use of a triple differences estimator is strength.
<table>
<thead>
<tr>
<th>Authors</th>
<th>Country</th>
<th>Sample</th>
<th>Time</th>
<th>Ages affected</th>
<th>Methods</th>
<th>Effect of retirement</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bingley and Pedersen (2011)</td>
<td>Denmark</td>
<td>Blue collar male employees from cohorts 1906-1921</td>
<td>1979 reform</td>
<td>60-66</td>
<td>IV</td>
<td>Reduced mortality</td>
</tr>
<tr>
<td>Bloemen et al. (2013)</td>
<td>Netherlands</td>
<td>Civil servants aged 53-60 during 1999-2005</td>
<td>2005 window</td>
<td>55 and above</td>
<td>IV-DiDiD with individual FE</td>
<td>Reduced mortality</td>
</tr>
<tr>
<td>Hernæs et al. (2013)</td>
<td>Norway</td>
<td>Full cohorts 1928-1938 in AFP firms</td>
<td>1990s reforms</td>
<td>62-67</td>
<td>IV and DiDiD</td>
<td>No effect</td>
</tr>
<tr>
<td>This thesis</td>
<td>Norway</td>
<td>Full cohorts 1902-1906</td>
<td>1973 reform</td>
<td>67-70</td>
<td>IV, DiD and individual FE</td>
<td>Increased mortality</td>
</tr>
</tbody>
</table>
10.4 Recommendations for future research

The results in this thesis cautiously support one hypothesis about the effect of retirement on mortality, namely that retirement increases mortality in the short term. But this should not be seen as a confirmation of the theories that predict a short-term increase in mortality following retirement. The theories presented in chapter 2 do not only produce predictions. They also postulate some mechanisms that produce the outcomes. The fact that this thesis’ results fit the predictions of some theories better than others is not in itself evidence that the causal mechanisms these theories postulate are true. This thesis does not test any mechanisms (except that of selection) and it thus uninformative about the causal processes at work.

One might be tempted, then, to suggest that a natural next step would be to test what mechanisms produce the results presented here. This could be done by for instance looking at changes in stress levels or health behavior following some exogenously induced retirement opportunity. But such a recommendation would be premature. Before embarking on the attempt to find an explanation of the effect, there is a need to establish what that effect is. As the previous section showed, there is currently no fact of the matter or established causal relationship in the research literature to explain.

It goes without saying, then, that more research is needed to establish the effect of retirement on mortality. One opportunity is to wait a few years and then exploit the Norwegian 2011 pension reform or some other recent pension reform that has taken place in another country. Repeated studies of different natural experiment may bring us closer to some truth about the effect and how it may vary with groups and contexts. An actual experiment whereby randomly selected people are given the opportunity to retire early would be even better. Such an experiment would be quite expensive, but less prone to the potential biases that always haunt quasi-experimental research designs.

Another interesting topic could be to investigate the potential spillover effects of partners’ retirement for couples. Such an analysis could actually be done on the data used in this thesis by matching individuals with their partners. But such an analysis has not been performed here, as it falls outside of the scope of this thesis.

In any case, this thesis underlines the importance of adequately dealing with selection bias in future research on this topic. Issues relating to selection, omitted variables and reverse causation have recently received increasing attention within sociology, and further
developments in the use of experimental and quasi-experimental data for the purposes of causal inference should be welcomed.
11 Conclusions
A number of theories give diverging predictions about the relationship between retirement and mortality. This thesis examined whether and how retirement is associated with mortality, and whether and how retirement affects mortality. The analyses were performed on cohorts born between 1902 and 1907, who for the most part retired in the 1970s.

The association between retirement and mortality was explored through event history analyses using discrete-time hazard models. These results show that being retired is associated with a continuously increasing probability of dying. Twenty years after retirement, the probability of dying was 17.0 percent higher among retired men relative to men at the same age who were not retired. The corresponding figure for women was 8.7 percent. Excluding disability pensioners substantially reduced the probability of dying at all durations up to 20 years after retirement. This indicates that health-related selection into retirement through disability pensions is an important driver of the association between retirement and mortality.

A causal link between retirement and mortality was identified by treating a pension reform that came into effect in 1973 as a natural experiment. Overnight, the minimum age for old age retirement eligibility was lowered by three years, giving some birth cohorts the opportunity to retire up to three years earlier than others. This variation in retirement eligibility was exploited in difference-in-differences and individual fixed effects estimations of the effect of retirement eligibility on mortality, and in instrumental variable estimations of the effect of retirement age on mortality. The results from these analyses show that retirement eligibility or earlier retirement increases mortality. Among men, the instrumental variable results indicate that one year earlier retirement increases the probability of dying before age 80 by 1.5 percentage points. This is a substantive effect. For women the effect is smaller – approximately 0.5 percentage points. Subgroup analyses uncovered some effect heterogeneity. The effect was apparently largest among men with lower education and among women with higher education. There was no evidence to support a “honeymoon effect” with regard to mortality following retirement.

The difference-in-differences, individual fixed effects and instrumental variables results are consistent, and the estimates appear to be robust. The effects appear to be driven primarily by a relatively short-term increase in mortality following retirement, although other interpretations of these findings are also plausible. Such a short-term effect is consistent with theories conceptualizing retirement as a stressful life event or continuity break, with short-
term changes in health behavior following retirement, or with the loss of some protective effect of employment. The results further indicate that the strong and continually increasing association between retirement and mortality is primarily driven by an omitted variable bias or some health-related selection process, such as a healthy worker effect.

The effects that are identified in this thesis are local and perhaps contingent on the national context and the time period. This does not mean that a generalization of these findings across contexts and time is impossible. But such a generalization should be done with care and requires some theoretical justification. Nevertheless, retirement appears to have increased mortality in 1970s’ Norway.
12 References


Durkheim, E. (1978) [1897]. Selvmordet. Oslo: Gyldendal Norsk Forlag


Mood, C. (2010). Logistic regression: Why we cannot do what we think we can do, and what we can do about it. *European sociological review, 26*(1), 67-82


*All references in this thesis are reported.*

*The number of words in this thesis is 36 642.*
Appendix: approval from the privacy ombudsman

Prosjektvurdering - Tilrådning fra personvernombudet i SSB

Behandlingsansvarlig: Statistisk sentralbyrå (SSB)

Prosjekt: Age of retirement and mortality in Norway
Endret: Forskningsavdelingen, Gruppe for offentlig økonomi og befolkningsmodeller (510)
Prosjektleder: Astrid Syse, forsker (510)
Deltakere: Kjetil Telle, Gruppeleder/Forsker 0 (510)
  Adrian Farne Rogne, prosjektmedarbeider (510)
  Kenneth Wiik, forsker (590)
  Andreas Fagereng, forsker (530)
  Anders Grøn Kjelrad, forsker (530)

FORMÅL
Formålet med studien er å undersøke sammenhengen mellom arbeid, pensjonering og dødselig - og i neste omgang sykelighet. Prosjektet er tredelt.

Del 1 skal gjennomføres utelukkende ved hjelp av interne registerdata fra SSB, hvor målet er å sammenligne dødelighet blant individer med ulikt pensjoneringstidspunkt og -alder, og undersøke i hvor stor grad familiedemenners karaktertrekk påvirker dødeligheten og sammenhengen mellom pensjonering og dødselig. Videre skal studien undersøke betydningen av utdanning, yrke og inntekt - herunder pensjonsrettig - for ulike pensjonsvalg og dødelighet.

(1 del 2 og 3 skal det inkluderes data fra Dødsfallsregisteret og IPLOS. Denne tilrådningen gjelder kun del 1)

UTVALG
Alle personer bosatt i Norge 1970-2018 (ca 9 mill) og alle aktive foretak/virksomheter i perioden

DATAMATERIALET

INNSAMLING/OPPBEVARING
Prosjektleder eller autorisert prosjektleder får tilgang til de nødvendige registerdata fra de aktuelle fagseksjoner og forestår selv kobling i samarbeid med LDA-510. Data oppbevares på dedikert prosjektmøde (HELSPEN) på SSBs forskningsserver.

VARIGHET
31.12.2019
VURDERING AV ANDRE INSTANSER

Del 1 av studien gjennomføres innenfor rammen av statistikkloven.

KOMMENTARER/VILKÅR

Denne tilrådning gir ledet kun del 1 av prosjektet.
Personvernombudet finner at behandlingen kan finne sted med hjemmel i personopplysningsloven § 8 og § 9 litra b og h, jf. statistikkloven § 1-1 og § 3-1 litra e.

Personvernombudet mener behandle av personopplysninger i dette prosjektet tilfredsstiller de krav som oppstiller i personvernlovgivningen. En eventuell ulempe for de involverte vil være marginal sett i forhold til den samfunnsnytte prosjektet representerer.


Personvernombudet har ingen innvendig til at den planlagte behandling av personopplysninger kan igangsettes, fortsatt at:

1. Behandling av personopplysninger i studien skjer i samsvar med og innenfor det formål som er oppgitt i meldingen.
2. Studien gjennomføres med identifiserte data.
   http://www.bygninget.ssb.no/Artikler/1257515360.78 Så snart datakildene er koblet og analysert er etablert, parkerer koblingsnøkkelen på et eget område hvor kun prosjektleder (ev. LDA) har tilgang. Alle etterfølgende bruk av koblingsnøkkel ut over oppdatering av nye årsager av data, skal på forhand forelegges og godkjennes av personvernombudet.
3. Prosjektleder i studien kan få tilgang til analysedata etter dokumentert saklig behov. Prosjektleder skal styre tilgangen til de ulike datakildene, dvs. at den enkelte forsker skal kun ha tilgang til de deler av analysematerialet som er nødvendig for utførelse av sin del av prosjektet. Det skal foretas en særskilt og dokumentert behovsprøvning av prosjektlederne tilgang til data fra FD-trygge LDA. Det kan lagres på prosjektledere av område (SHELSPEN) på en av SSBs forskningsvarene.
   4. Data kan lagres på prosjektleder av område (SHELSPEN) på en av SSBs forskningsvarene.

Personvernombudet finner at prosjektet kan unntas fra sin informasjonsplikt overfor utvalget, da prosjektet har hjemmel i lov og grunnet vanskeligheten det å gi informasjon ville medføre, jf. personopplysningsloven § 20 annet ledd.

Det gjøres oppmerksom på at personvernombudet kan henvendt det juridiske behandlingsgrunnlaget og de personvernmessige forhold ved prosjektet. Tilgang til data må avklares med den enkelte ansvarliges fagsjekjon i SSB.
Universitetet i Oslo
Institutt for sosioologi og samfunnsgeografi
Oslo, Norge

Oslo, 21.10.2016
Marte Rønning,
seksjonsleder ved Gruppe for offentlig økonomi og befolkningsmodeller
Forskningsavdelingen, Statistisk sentralbyrå

Bekreftelse på at prosjektet er utvidet til også å inkludere personer som døde før 1970

Jeg bekrefter herved at personvernområdet ved SSB har gitt skriftlig godkjenning på at datagrunnlaget i prosjektet «Age of retirement and mortality in Norway» utvides med årgangene 1967-1969, slik at utvalget består av alle personer bosatt i Norge fom 1967 (ca 9 mill) og alle aktive foretak/virksomheter i perioden.

Med vennlig hilsen

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