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The Impact of New Drug Launches on the Loss of Labor from Disease and Injury: Evidence from German Panel Data

by

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Abstract

This paper studies the evolution of early retirement due to disease and injury in the German labor force between 1988 and 2004. Using data from the German Federation of Public Pension Providers, the IMS Health Drug Launches database and the WHO Mortality Database, we show that new drug launches have substantially helped to reduce the loss of labor at the disease-level over time. We employ a variety of econometric methods to exploit the pseudo-panel structure of our dataset and find that in Western Germany alone each new chemical entity has on average saved around 200 working years in every year of the observation period. Controlling for individual determinants of health-related retirement, such as worker's age, sex and type of work, we also find evidence that the 2001 reform of pension laws has led to further reductions in the loss of labor from disease and injury.

Keywords: Medical technology, Early retirement, Rehabilitation services, Pension reform, Pseudo-panel data

JEL: I12, I18, J26

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I. Introduction

In this paper, we study the evolution of early retirement due to disease and injury in the German labor force from 1988 through 2004 and test the hypothesis that new drug launches have helped to reduce the loss of labor at the disease level over time. Amid rapid population aging, Germany has a vital interest in finding new ways to extend people's productive labor force participation up to, and beyond, the current official retirement age of 65 years. Herbertsson and Orszag (2003) estimate the total cost of early retirement at around 13 percent of Germany's potential GDP, more than 50 percent above the average for all OECD countries. Rehfeld (2006) reports health-related early retirement alone accounted for 20.4 billion Euros or 2.9 percent of Germany's total social budget in 2003, after peaking at close to 3.5 percent in the late 1990s; and public pension providers paid for 60.1 percent of the direct costs in 2003, with most of the rest distributed among accident insurance, welfare programs for civil servants and company-based private pension providers.

Aggregate data have recorded a general decline in the number of new health-related cases of early retirement since the mid-1990s, especially among male blue-collar workers in the West and both male and female blue-collar workers in the East of Germany. In 2003, a total of 1.76 million disease and injury pensions still accounted for 7.3 percent of all pensions paid by Germany's public pension providers. However, the aggregate decline has masked an increasing trend towards early retirement for specific medical diagnoses, above all for psychiatric problems and – less pronounced – for neoplasms. Since the early 1980s, the average age of health-related early retirement has even dropped from around 58 years to 49 years for West-German men and from 55 years to 51 years for West-German women, whereas it has stayed roughly constant below 50 years for both sexes in Eastern Germany since 1993.

The literature has mainly attributed the observed general decline to changes in the relevant legal environment, such as most recently the major reform of German pension laws that came into effect in 2001 (Rehfeld 2006; Viebrok 2003). This reform has made filing a medical application for early retirement less attractive financially by introducing age-adjusted replacement ratios instead of the immediate right to a full pension. However, a back-of-the-envelope calculation, based on the total number of lost employment years in 1994 and 2002 as reported in Statistisches Bundesamt (2000 and 2004), suggests there have been substantial improvements for major diseases long before the new pension laws. While the overall disease- and injury-related number of lost employment years, relative to the number of employees, declined by approximately 20 percent between 1994 and 2002, there was a much larger decline in the case of diseases of the nervous system and the senses (more than 60 percent), in the case of cardiovascular diseases (45 percent), and in the case of diseases of the digestive system (46 percent). On the other hand, the reported employment loss due to psychiatric and behavioral disorders increased by more than 20 percent. For detailed tables, see Statistisches Bundesamt (2006).

Our study focuses on the total amount of labor lost since 1988 – the number of lost working years relative to normal retirement at 65 – and thus takes into account that in terms of the economic burden, declining entry ages may compensate for a reduced number of early retirement cases. We estimate that in Western Germany alone each launch of a new chemical entity for the treatment of specific diseases has reduced the number of lost working years by around 200 working years per year, after controlling for the diagnosis according to the International Classification of Disease and Injury, ICD-9 and ICD-10, respectively, and for other factors such as age, sex, type of work, the utilization of rehabilitation services and the impact of the 2001 reform. Without the stock of new chemical entities launched in Western Germany since 1988, the total number of working years lost to disease and injury in 2004 would have been one percent higher. The total gain of working years from new chemical entities over the entire 1988 to 2004 period is likely to have been in the order of one tenth of the loss of labor in 2004. On the basis of average monthly disability pensions at 703 Euros for men, who accounted for approximately 70 percent of lost working years, and 589 Euros for women in 2003, as reported in Rehfeld (2006), the implied financial gain to the public pension system amounts to 140 million Euros annually. The total savings from all 86 new chemical entities in the 17 year sample period is likely to have been at least ten times higher.

Methodologically, our paper builds on Lichtenberg's (2005) health production function approach in which the age at the time of death from a given disease is regressed on the number of new drugs launched to treat the disease and on a vector of other explanatories, which may include measures of education, income, nutrition, the environment and lifestyle. We augment this framework by introducing a second equation in which the number of lost working years to a given disease is regressed on the number of relevant drugs launched and on a vector of other variables that influence the propensity to retire with a given diagnosis and at a given age. We compare the estimates from separate regressions with results from a systems approach that estimates both equations simultaneously.

Because our main focus is on the timing of early retirement, not mortality, it is useful to limit the scope of this study to one country so that changes in the relevant laws governing the eligibility for public pensions on health grounds are limited to the time dimension. A cross country study would be fraught with greater difficulty in identifying the impact of new medical technology when early retirement behavior is also affected – in unknown ways – by country-specific laws. In general, we are confident about solving the identification problem because most new drug launches are specific for individual diseases, whereas the other major explanation of changes in retirement behavior over time, observed changes in government regulation, tends to affect all potential retirees equally, regardless of their individual medical problem.

Our identification strategy rests on the assumption that all determinants of retirement timing other than mortality are exogenous. This assumption is strictly valid only in the absence of contemporaneous or lagged dependence of disease-specific retirement hazards on the retirement hazard observed for other diseases. Even in our pseudo-panel, in which we analyze the retirement hazard for population subgroups, not individuals, the assumption may be invalid for two main reasons: first, each event of early retirement from one disease may alter the composition of the population at risk of early retirement thereafter so that other diseases become a relatively more likely reason of early retirement and second, the causes underlying different diseases may be correlated. For example, when medical progress lowers the propensity to retire for cardiovascular disease, it is not surprising that age-adjusted propensities to retire for other diseases whose underlying causes are positively or negatively correlated with cardiovascular disease will change as well. Moreover, in the case of an incurable chronic disease that develops according to an exogenous probability distribution once in a person's life time, each event of early retirement will tend to lower the subsequent probability of early retirement from that cause in the retiree's cohort.

These identification issues arise from the competing risks nature of early retirement, which can be triggered by a wide range of potential health problems at the individual level. Competing risks represent both an econometric and a policy challenge. The policy challenge is to set efficient incentives for the developers of new medical technologies that reduce "early" risks and must in some way be compensated for the windfall profits they create for subsequent developers addressing "later" risks. In this paper, we begin to address the econometric challenge by developing a test for the presence of competing risks in our dataset.

Our paper thus adds to the recent literature that has examined the diffusion of medicines and disease-based patterns of under-treatment in Europe (Schöffski 2002), investigated empirical correlations of health status and morbidity with age and other socio-economic characteristics and analyzed the determinants of health services utilization in all 15 countries included in the European Community Household Panel (Layte et al. 2005), and estimated the impact of new medical technology on longevity and on the quality of life (Lichtenberg and Virabhak 2002).

The remainder of this paper is divided into six sections as follows. Section 2 presents the relevant theory. Section 3 describes the data. Section 4 presents the main empirical issues considered in the paper and motivates the choice of econometric methods that we use to identify the empirical impact of new drug launches on retirement timing with endogenous longevity. Section 5 presents the estimation results for the early retirement regression and for the regression explaining the age distribution of deaths. Section 6 discusses these findings in the context of related literature. Section 7 concludes.

II. Theory

The individual demand for early retirement is derived from a worker's demand for health. It is constrained by the trade-off between health and wealth: At what age is the expected marginal benefit from continued work, generating higher income, equal to the expected marginal benefit from retirement, helping to maintain the worker's health? As health declines, so does the relative private reward of continuing to work when pensions are available from a pay-as-you-go system. See the downward-sloping demand schedule, DD in Figure 1, whose location depends, inter alia, on the size of the replacement ratio of pensions to wages.

The supply curve of early retirement can be thought as a vertical line, SS in Figure 1, whose location is determined by the laws governing the eligibility for early retirement conditional on the applicant's state of health. For example, the major reform of Germany's disability pension system that became effective in 2001 abolished the traditional distinction between Berufsunfähigkeit, the inability to continue working in the chosen occupation, and Erwerbsunfähigkeit, the inability to work in any job. The new system distinguishes between workers still able to work between three and six hours a day, who may hence claim only a reduced pension, and those only able to work less than three hours a day. In addition, the new law adjusts for the age of claimants in line with the rules for early claims of old-age pensions by severely disabled persons.

It is important to note that pay-as-you-go pension funding implies a divergence between the private and social opportunity costs of early retirement when health declines. The social costs will exceed the private costs when the retiree is still in relatively good health, but the social costs will fall below the private costs when the retiree's health is very poor. The reason is that the private opportunity costs do not account for the possibility that labor productivity falls below the wage level, nor for the social insurance value of early retirement against further declines in health.

Figure 2 depicts the sequential decision making that precedes a worker's early retirement. It shows that both acute medical care and rehabilitation services may help to reduce the effective demand for early retirement. Successful acute care, in which disease-specific pharmaceutical input often plays a decisive role, produces a healthy worker who does not apply for early retirement. Rehabilitation services, by contrast, are often ordered by the pension insurer to restore a worker's health after an application for early retirement has been filed and medical eligibility has been determined; acute care has then been insufficient, yet rehabilitation services may still return a healthy worker who no longer demands to retire early. In a similar vein, the pension insurer may order retraining, termed vocational rehabilitation in the insurers' jargon, for jobs that are compatible with the worker's health impairment.

Acute care and rehabilitation services are more likely to be substitutes for mild diseases and more likely to be complements for serious diseases. In the case of mild diseases, either acute care or rehabilitation may be sufficient to prevent early retirement, whereas many serious diseases require both and can still lead to early retirement. Like drugs, rehabilitation services are often disease-specific and the extent to which the utilization of rehabilitation services and drugs should be considered substitutes or complements in the prevention of early retirement may vary across diseases. In the case of chronic diseases, for example, rehabilitation may be accompanied by continued medication.

An important difference between the allocation of drugs and rehabilitation services is that drugs are generally available to all sick workers on equal terms, whereas a pension insurer may find it profitable to target scarce rehabilitation resources at those cases where the expected savings in terms of discounted pension payments are greatest. These cases tend to be young workers, where the prospect of many future working years can justify a given rehabilitation expense even if the probability of successful rehabilitation is relatively slim. Germany's public pension insurers operate their own proprietary network of rehabilitation clinics, the only part of the country's health system that fully integrates financing and provision of care.

To motivate our empirical analysis and derive testable hypotheses, let us consider a version of Philipson and Becker's (1998) model of endogenous longevity in the presence of mortality-contingent claims. A key insight of that paper is that greater longevity may stimulate private savings whenever the pension annuity is smaller than required for intertemporal consumption smoothing. However, to focus on the *demand for early retirement*, we shall – initially – assume exogenous longevity with a remaining life of T years. In this case, it is the earlier timing of retirement which may stimulate a demand for greater private savings, given the prospect of more years in retirement. But the shorter time that early retirees spend working means that the accumulated amount of savings available at the time of retirement may actually be lower. Consumption smoothing then implies that the annual consumption level, too, will be lower – even in the extreme case, assumed below, that the size of the pension annuity, r, is independent of the time of retirement.

For a given retirement age, the indirect utility

$$V(W,T) \equiv \max_{c(t)} \int_{t=0}^{T} U(c(t))dt \qquad \text{s.t.} \quad \int_{t=0}^{T} c(t)dt \leq W$$

is obtained by perfect consumption smoothing, given wealth W, so that consumption levels are equal in every year: $c(t)=W/T$ and $V(W,T)=TU(W/T)$.

$W = \int_{t=o}^{R} y(t)dt + \int_{R}^{T} r(t)dt$ where $y(t) = f(H(t))$ represents the functional relationship between health H, which tends to decline with age and may be subject to additional irregular disease shocks, and the marginal product of labor, which is assumed to equal wage income,

with $\frac{\partial y}{\partial H} > 0, \frac{\partial y^2}{\partial H^2} < 0$.

For simplicity, we assume that $r(t)$ is exogenous and constant for all t. If the time of retirement, R, is a variable whose value can be chosen freely, utility will be maximized by some R such that $y(R) \geq r$. If $y(t) > r \; \forall \, t$, the utility maximizing R will be equal to T and there will be no retirement. However, we assume that health declines monotonically with time, and so does y and hence there is likely to be a time $\tilde{t}$ where $y(\tilde{t}) = r$. The sooner H declines, the earlier will be the optimal time of retirement R for a given level of the pension annuity r. This is the basic model of the demand for retirement on health grounds. Perfect consumption smoothing requires positive savings in all periods where $y(t) > r$. In essence, early retirement represents a mechanism to prevent further declines in personal health.[2]

Medical care, assumed to be available in fixed quantities per period, M, can be viewed as an alternative mechanism so that the relative price of medical care and early retirement will determine workers' demand for both. To account for this role of medical care, we assume that $y(t) = f(H(t, M)) - pM(t)$ where $H = h(t, M) = \max(T - t, qM)$. It follows that people will not utilize medical care as long as $T - t \geq qM$, but they will consider medical care only if $qM > T - t$ while $\frac{\partial y}{\partial H} \geq pM$. Intuitively, there will be some decline in health before people consider medical care as a way to halt their declining productivity. For a given effectiveness or quality of medical care, q, the demand for medical care will depend on the price p, and vice versa. We assume that *new* medical technology reduces p and/or increases q. However, if q is too low and/or p is too high to make the purchase of medical care worthwhile, people

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[2] We do not address the possibility that a low expected health status during retirement may make full consumption smoothing undesirable when the marginal utility of consumption declines with health.

will want to respond to a decline in their health by going straight into early retirement. In the presence of health insurance, p is the consumer co-payment or co-insurance.

In this model, medical care serves as a substitute for early retirement. In reality, by contrast, *acute medical care* may – ex post – often appear to be a complement in the retiree's health production function since early retirement will rarely be granted without a prior attempt to treat and cure the applicant's disease. Public pension insurers will usually base the early retirement decision on a physical examination of the applicant's health and the prospects of restoring the applicant's ability to work. Our model does not address these details. But our empirical analysis will consider *rehabilitation services* as a potential substitute for early retirement, as these services are ordered by the pension insurer in an attempt to avoid incurring the early retirement liability towards the insured.

Endogenous mortality. Both early retirement and medical care, by halting the work-related decline in health, may lead to a longer life when mortality is assumed to be endogenous. This may have implications for the timing of early retirement, primarily through the increased demand for savings to smooth consumption over more years. A longer life expectancy may hence reduce the private incentives for early retirement and boost the demand for high-quality medical treatment so as to return to work quickly and accumulate as large a stock of savings as possible for the eventual retirement. This will raise the price of early retirement relative to medical care. Early retirement will hence happen later than in the case of exogenous mortality, but the demand for medical care to treat a given disease will be greater.

At the disease-level, the number of lost working years and lost life years are likely to be correlated over time – and this correlation could be either positive, since for a given state of medical technology early retirement may enhance the chances of recovering from disease or injury of living longer, or negative, since improvements in medical technology may both reduce the need for early retirement and extend the remaining life expectancy for those receiving the best treatment. The presumption of a positive correlation follows from the health production point of view, emphasizing the – usually lagged – impact of early retirement on mortality, relative to the hazard of death in the absence of early retirement. By contrast, a negative correlation can be inferred from an investment point of view, emphasizing that a longer life expectancy may reduce the private incentives for early retirement at a given level of medical technology and boost the demand for high-quality medical treatment so as to return to work quickly and accumulate as large a stock of savings as possible for the eventual retirement. This negative correlation is likely to be contemporaneous if current mortality is the best predictor of future mortality from a given disease.

Competing risks. A final consideration concerns the empirical implications of the competing risk nature of retirement causes. Following van den Berg (2005), competing risks can be

modeled as multiple durations that start at the same time for a given subject, but are only observed until the first duration is completed; it is then revealed which of the multiple durations is completed first. Both mortality and early retirement with more than one cause represent competing risks that are, however, fundamentally unidentified in empirical models (Honoré and Lleras-Muney 2006). We therefore do not seek to quantify disease-specific hazards, but aim to provide a novel test for the relevance of competing risks for the timing of health-related retirement in our pseudo-panel.

Testable implications: Although our rudimentary model has not explored the interaction of public and private savings, personal health status and investments in health in all its details, we see the following testable implications emerging from the analysis:

- Early retirement for a given disease will occur less frequently and later, the more drug launches have been accumulated.

- Early retirement for all diseases will occur later, the lower the replacement ratio of pensions to wages and the more restrictive the medical eligibility criteria – implying that the legal reform coming into effect 2001 reduces the rate of early retirement.

- A decline in numbers, or an increase in the average age, of early retirement for a given disease will be accompanied by an increase of early retirement from other causes that typically occur at a later age, after controlling for the impact of new drug launches.

- A decline in the mortality from a given disease will have an additional impact on the timing of early retirement from that disease, assuming that current mortality is the best measure of expected longevity.

- Holding medical care constant, a decline in early retirement for a given disease will subsequently lead to a rise in mortality from that disease.

- Under the hypothesis of a substitutional relationship between early retirement and the consumption of rehabilitation services in the health production function, retirees who have received rehabilitation services for the main diagnosis justifying their retirement will tend to be older than those who retire without having received these rehabilitation services. To control for age as an independent determinant of the propensity to receive rehabilitation services we use observations on the rehabilitation services that retirees have received for other than their respective main diagnoses.

III. Data

Timing of early retirement. The dataset from the Federation of Public Pension Providers in Germany (VDR) provides detailed information on the stocks and flows in Germany's statutory pension insurance, in which more than 80 percent of the economically active population is enrolled. In 2003, for example, 31.1 million of Germany's 38.6 million economically active persons paid monthly contributions to establish pension claims. While civil servants are excluded as they are insured in the government's own tax-funded civil servant health care system, which is not covered by VDR, the statutory pension insurance is mandatory for every worker except the self-employed and those with very low earnings.

In 2003, the total number of mandatory insureds was 27 million or 87 percent of all insureds in Germany's statutory pension insurance. The VDR gives data on new beneficiaries of disability pensions by disease, which are further differentiated by age, sex, region, type of work and a variety of other criteria. These data are grouped and available for six age intervals: 0–39, 40–44, 45–49, 50–54, 55–59, and older than 59 years. In addition, the average age of new disability pensioners is given. Diseases are defined according to the International Classification of Disease and Injury, version ICD9 up to 1999 and ICD10 from 2000 on. The rules we applied to match ICD9 and ICD10 to ensure consistency of the time series is described in the data appendix.

Figure 3 illustrates the age-specificity for three major disease categories that led to early retirement in 1996 and 2003 – cancer, circulatory and psychiatric diseases. While between 30 and 50 percent of early retirement for cancer and circulatory diseases was recorded for workers in the second oldest age group and much smaller shares in the younger age groups, early retirement was much more evenly distributed across age groups in the case of psychiatric diseases. Figure 4 basically confirms this observation using Kaplan-Meier estimates of survival rates, the probability of not having retired for a given disease at a certain age, for men and women as workers until their retirement on medical grounds. As a caveat, we point out that people might exit the pool of insured workers in other ways than retirement, such as death, and that measuring the efficiency in the prevention of early retirement would ideally require Kaplan-Meier estimates of survival conditional on being sick with a given disease; but we do not observe the number of diseased workers.

Given these limitations, the first six panels compare the survival rates for cancer, circulatory, psychiatric diseases and the aggregate of all diseases for Eastern and Western Germany in 2003. For each age group, the highest survival rates are found for circulatory diseases, except for males in age intervals 55–59 and 60–64 where the highest survival rates are found for cancer and psychiatric diseases. Psychiatric problems seem to have taken a particularly heavy toll among women in the West. The last six panels of Figure 4 compare these survival rates in the years 1996 and 2003. Survival rates are generally higher in 2003 than in 1996, especially

for circulatory diseases, but appear to be slightly lower for cancer among East German males. Comparing Eastern and Western Germany, relatively small differences in cancer survival rates stand in contrast too much larger interregional differences for circulatory and psychiatric diseases. But whether these differences are due to differences in access to medical technology or due to temporal interdependence implied by competing risks cannot be decided unless the temporal dependence of changes in diseases-specific hazards is properly taken into account, as Honoré and Lleras-Muney (2006) demonstrate in the context of mortality from cancer and cardiovascular disease.

Age distribution of deaths. By using data on the age distribution of deaths, available for the 1988 to 2004 period from the World Health Organization's (WHO) Mortality Database, we obtain a separate measure to control for medical technology's effectiveness at the disease level as well as an empirical handle to account for the endogeneity of retirement behavior with regard to remaining life expectancy. The distribution of deaths across age groups is therefore a potentially important variable in our empirical strategy to assess the impact of new drug launches on retirement behavior.

Figure 5 shows Kaplan-Meier "physical" survival estimates for male and female mortality due to cancer, circulatory and psychiatric diseases in 1996 and 2003. These interval estimates are based on a similar set of six age groups, defined over five-year intervals, beginning with those below 40 years of age, but ending with an open interval of all those aged 60 or older instead of the closed interval of 60—64 year old retirement candidates. The varying graphical end points in the highest age interval reflect the differences in remaining life expectancy from the three diseases. In the first four panels, the highest risk of death is associated with cancer although in the case of men, this risk is only slightly higher than the hazard associated with circulatory diseases. Psychiatric diseases, by contrast, appear to have the highest survival rates. Moreover, in contrast to our Kaplan-Meier estimates for retiring, the psychiatric mortality hazard, virtually unchanged over time, appears to be much less of a problem of younger workers. As shown in the lower three panels of Figure 5, cancer and circulatory disease survival rates are generally higher among females than among males, notwithstanding the evident progress between 1996 and 2003. Again, a caveat about the yet to be fully understood implications of the competing risks nature of mortality causes applies.

Drug launches. In the German health care system, access to new drugs is generally not rationed on the basis of patients' health insurance status. To all enrollees of statutory sickness funds, more than 90 percent of the population, reimbursement of new drugs becomes available at the same time – after it has been approved by the so-called "federal joint committee," in which both the doctors' associations and the sickness funds are represented. For specific new drugs, quicker reimbursement may sometimes by available to those with full private health insurance, approximately 8 percent of the population. Yet, the difference in

health insurance status does not result in significant differences in prescription drug access: Even enrollees of the statutory system can choose to purchase a new drug out-of-pocket that the "federal joint committee" has yet to approve for general reimbursement. While the VDR database does not actually say whether those retiring as disability pensioners have private or statutory health insurance, we assume the vast majority is in the statutory system, since only a relatively small fraction of workers with private health insurance will be enrolled in public pension insurance. Among the two major groups with a right to opt out of statutory health insurance, the self-employed will tend to choose both private health insurance and non-participation in the public pension system, whereas early retirement by civil servants, who often choose some form of private health insurance to complement the health care subsidies from their government employer, will not even be recorded in the VDR database.

Against this background, we treat the time of a drug's launch on the German market as the time at which it is available to all workers at risk of health-related early retirement in our dataset. The IMS Health Drug Launches database has tracked market launches of new chemical entities (NCEs) and non-new chemical entities (non-NCEs) worldwide since 1982. When this database labels a product ingredient as a new chemical entity, these launches are guaranteed to be initial launches as explained in more detail by Lichtenberg (2005). It is only for products launched before 1982 that we cannot know exactly whether these contained new chemical ingredients at the time that were subsequently included in drugs launched after 1982. Following Lichtenberg, we do not count any ingredients that the database does not explicitly designate "new chemical entities" as initial launches since these might contain ingredients first launched prior to 1982. We therefore count these as non-new chemical entities, which include initial launches and re-launches. Many of these may of course be new in the sense that they possess improved pharmacological properties, such as lower side effects, but we ignore these differences among the "non-new" drug launches.

We measure the relevant stock of pharmaceutical technology for our analysis by calculating the cumulative numbers of new and non-new drug launches during three subperiods of our analysis time: first, the period before the onset of the retirement risk in our analysis, 1982—87, second, a period in which we only consider West Germany, 1988—1994, and third, the period in which we consider both parts of reunified Germany, 1995—2004. For the first subperiod, IMS records zero new chemical entity launches and 8 non-new chemical entity launches in category labeled "Germany" as well as 31 new chemical entity launches and 2133 non-new chemical entity launches in a category labeled "West Germany." For the second subperiod, IMS records 48 new chemical entity launches and 8607 non-new chemical entity launches in the "Germany" category as well as 7 new chemical entity launches and 2797 non-new chemical entity launches in the category "West Germany." In the third subperiod, IMS records 44 new chemical entity launches and 6940 non-new chemical entity

launches for all of Germany. Figures 6 and 7 show the distribution of these drugs across the major therapeutic classes in our study, as described in Table 1 and in the appendix.

Over all therapeutic classes, we count a total 86 new chemical entity launches and 13545 non-new chemical entities between 1982 and 2004. We consider 16 therapeutic classes, such as central nervous system and parasitology drugs, with a few of the drugs applicable to more than one class. We do not attempt to assign the drugs to more detailed therapeutic subclasses because that would greatly increase the number of drugs that should be assigned to several of these subclasses. For example, anti-infective systemic drugs may be used to treat HIV and tuberculosis. The cumulative number of newly launched chemical entities in therapeutic area i is given by $Cum\_NCE_{iT} = \sum_{t=1982}^{\tilde{T}} NCE_{it}$, where t = 1982,…, 2004 and $\tilde{T}$ indicates the current year within the sample period. Likewise, the cumulative number of non-new chemical entities in therapeutic area i is given by $Cum\_non\text{–}NCE_{iT} = \sum_{t=1982}^{\tilde{T}} non-NCE_{it}$.

The linkage of drug launches to diseases. Although the classification of drug launches in the IMS Health Drug Launches database and of diseases in the International Classification of Disease and Injury (ICD) follow different principles, especially at the detailed level, their hierarchical design facilitates the identification of linkages at higher levels of aggregation. At the highest level, 16 therapeutic classes correspond quite closely to 19 main disease categories in ICD9 allowing us to match 13 of these drug classes with the appropriate disease categories. For example, drug class A comprises the drugs used in the treatment of diseases bearing ICD9 codes 520–579 and drug code G comprises the drugs used in the treatment of diseases bearing ICD9 codes 580–629 and 630–676. Table 1 provides an overview of our matching between IMS drug classes and ICD9 codes and gives the total cumulative numbers of new chemical entities and non-new chemical entities for each drug class. For further details of the matching, see the appendix.

Utilization of rehabilitation services. The great efforts Germany's public pension insurers make to prevent costly retirement by providing targeted rehabilitation services are extensively documented in the VDR database. For example, the database reveals the number of cases at the disease level where early retirement was preceded by the utilization of rehabilitation services addressing the same diagnosis that justified the retirement, and whether the rehabilitation services were provided in the last year, the last two years, or the last five years before the early retirement decision. As Figure 8 shows, the number of female retirees *without* prior rehabilitation has declined more than the number of female retirees *with* prior rehabilitation, while the corresponding ratio has stayed roughly constant for male retirees since 1995. Our study exploits the more detailed information on rehabilitation input to investigate whether rehabilitation services have been effective in preventing or postponing early retirement at the disease level.

In this context, we must take into account that pension insurers ordering rehabilitation services for an applicant may not solely be motivated by medical need, regardless of age. In addition, an important motif may be the financial savings from returning a healthy worker. Concentrating a large part of scarce rehabilitation resources on young workers, where successful rehabilitation generates the largest financial savings to the pension insurer, may hence be a rational strategy. Back-of-the-envelope calculations suggest, the expected financial savings from a successful rehabilitation of a worker aged 40 may justify a five times greater rehabilitation effort, relative to the probability of success, than for a worker aged 60. Put differently, for the same annual pension payments, a rational pension insurer would try rehabilitation on a 40 year-old worker even if the probability of success were only 20 percent of that of a 60 year-old worker.

To maximize the returns from rehabilitation investments overall, the disease-specific opportunities for a successful rehabilitation will also play a role. As Figure 9 shows, the concentration of rehabilitation services in different age groups of retirees does indeed vary across diseases. In the case of cancer, the top panel reveals that 50 percent of early retirees for cancer aged 60 or older have received prior rehabilitation for cancer, but less than 20 percent of those retiring between 55 and 59, and much lower percentages of those retiring at younger ages. Moreover, the concentration among the old has increased from 1996 to 2003. In the case of circulatory diseases, the middle panel reveals that there is a strong concentration of rehabilitation services among 55 to 59 year olds although it has declined from 38 to 25 percent between 1996 and 2003.

Figure 10 provides further evidence on the disease-specific distribution of rehabilitation services across age groups. It displays the ratio of the number of rehabilitation services for six selected diseases to the number of early retirees in each of six age groups. Again, the very strong concentration of rehabilitation for cancer among the oldest workers, shown in the first panel, is striking. Indeed, special rules for cancer patients require Germany's public pension insurers to consider even pensioners beyond the official maximum age of retirement as eligible for free rehabilitation services. But the picture is very different for the other five disease categories – circulatory, psychiatric, respiratory and digestive as well as diseases of the nervous system. In these cases, the ratio of rehabilitation services to the number of early retirees is much larger among the young, although the greatest relative increases in rehabilitation effort between 1996 and 2003 are recorded for the oldest workers.

In order to distinguish between the implications of differential age-related targeting of rehabilitation services from those of differential rehabilitation effectiveness at the disease level, our empirical analysis uses three different measures of rehabilitation input: first, the number of early retirees with prior rehabilitation for the retirement-inducing disease; second, the ratio of the number of early retirees with prior rehabilitation for the retirement-inducing

disease to the number of early retirees with prior rehabilitation for *other* diseases; and third, a dummy variable for retirees without any prior rehabilitation. The first of these variables is clearly inappropriate as a measure of effectiveness, since by definition the rehabilitation effort on workers who then retire has been unsuccessful. A negative impact of this variable on the loss of working years could mean that overall the rehabilitation effort has been successful or that it has been targeted mainly at older workers. Deciding which of these possibilities is correct would require observation of all successful cases of rehabilitation.

However, the VDR database does not provide the number of workers where disease-specific rehabilitation has successfully prevented early retirement. We therefore propose using the second input variable to obtain an indirect measure of rehabilitation effectiveness. Its numerator includes only unsuccessful cases of rehabilitation whereas its denominator includes cases of rehabilitation that have presumably been largely successful for other diseases than the retirement-inducing disease, provided the diagnosis was right in the first place. Assuming that rehabilitation targeted at a specific disease has been successful in preventing some, if not all cases of potential early retirement for that disease, we should observe that on average, cases of unsuccessful rehabilitation happen at a higher age than episodes of successful rehabilitation for other diseases in the same group of workers. In essence, we are using the recipients of rehabilitation for *other* diseases as an instrument for the unobserved cases of successful rehabilitation for retirement-inducing disease and conclude that the targeted rehabilitation effort has been effective, on average, if the ratio defined by our second rehabilitation input variable has a significant negative impact on the loss of working years. In this case, prior rehabilitation for the retirement-inducing disease must have helped to postpone retirement relative to prior rehabilitation that the same group of workers received for *other* diseases.

Finally, a positive impact of the third rehabilitation input variable, the dummy for those without any rehabilitation prior to their health-related retirement, on the loss of working years would reinforce the conclusion that rehabilitation is generally effective in preventing or postponing early retirement on medical grounds.

IV. Empirical Methods

Econometric framework. Our data represents a *pseudo panel* and allows us to apply a variety of panel data methods. The presumed endogeneity of early retirement with respect to remaining life expectancy at the disease-level can be captured by a two-equation model – including an early retirement and a separate mortality equation. In addition, dynamic panel methods may be required because each case of deferral of retirement this year, for example because the public pension insurer wants to try rehabilitation first, may affect the probability of early retirement next year and this would introduce exactly the sort of autocorrelation structure that dynamic panel methods are designed to address.

The early retirement equation. We hypothesize that the number of lost working years from disease i in year t depends on the cumulative number of new chemical entities launched and the cumulative number of non-new chemical entities launched to treat disease i by time t, and other factors:

$$(1) \qquad \tilde{R}_{it} = \beta(Cum\_NCE_{it}) + \gamma(Cum\_non-NCE_{it}) + \delta X_{it} + \eta Y_{jit} + \mu Z_{jit} + \varepsilon_{it}$$

where $\tilde{R}_{it}$ is the number of lost working years from disease i in year t, Cum_NCE_{it} is the stock of new chemical entities launched to treat disease i by year t, $Cum\_non\text{-}NCE_{it}$ is the stock of non-new chemical entities launched to treat disease i by year t, and X_{it} is a vector of factors that may affect the number of early retirees and the timing of retirement. We define

$$(2) \qquad X_{it} = \alpha'_i + \lambda'_{ji} + \omega'_i + \xi'_i + \theta'_t + \nu'_{it}$$

with α'_i = a fixed disease effect which is invariant across years in disease i, λ'_{ji} = a fixed effect for disease i in educational background, blue-collar workers (j=1), white-collar workers (j=2) and miners (j=0), respectively, ω'_i = a fixed effect for disease i in men (1) and women (0), respectively, ξ'_i = a fixed effect for disease i in Eastern (1) and Western Germany (0), respectively, θ'_t = a fixed year effect which is invariant across diseases in year t, and Y_{jit} = the number of early retirees in disease i who received rehabilitation services for disease i (j=1) and the number of early retirees in disease i who received rehabilitation services for other diseases (j=2), and Z_{jit} = a vector of mortality from disease i (j=1) and from other causes (j=2) at various age intervals.

Substituting (2) into (1) yields

$$(3) \qquad \tilde{R}_{it} = \beta(Cum\_NCE_{it}) + \gamma(Cum\_non-NCE_{it}) + \eta Y_{jit} + \mu Z_{jit} + \alpha_i + \lambda_{ji} + \omega_i + \xi_i + \theta_i + u_{it}$$

where $\alpha_i = \delta\alpha'_i$, $\lambda_{ji} = \delta\lambda'_{ji}$, $\omega_i = \delta\omega'_i$, $\xi_i = \delta\xi'_i$, $\theta_t = \delta\theta'_t$, and the standard error term $u_{it} = \delta\nu'_{it} + \varepsilon_{it}$.

The mortality equation. We employ a second equation which is similar to Lichtenberg's regression to examine the impact of new drug launches on the loss of life. However, we do estimate not only the impact of new drugs launches but also the influence of early retirement and other factors on the loss of life.

Specifically we estimate the following mortality equation:

(4) $$L_{it} = \beta(Cum\_NCE_{it}) + \gamma(Cum\_non-NCE_{it}) + \delta X_{it} + \mu Z_{jit} + \varepsilon_{it}$$

where L_{it} is the lost life years relative to 100 from disease i in year t, Cum_NCE_{it} and $Cum\_non\text{-}NCE_{it}$ are the stocks of new chemical entities launched and non-new chemical entities to treat disease i by year t, respectively, and X_{it} is a vector of factors that may affect the number of deaths and the timing of death

(5) $$X_{it} = \alpha_i' + \omega_i' + \theta_t' + \nu_{it}'$$

with α_i' = a fixed disease effect which is invariant across years in disease i, ω_i' = a fixed effect for disease i in men (1) and women (0), respectively, θ_t' = a fixed year effect which is invariant across diseases in year t, and Z_{jit} = a vector of factors including disease-specific early retirement from disease i (j=1) and from other disease (j=2) at various age intervals.

Substituting (5) into (4) yields

(6) $$L_{it} = \beta(Cum\_NCE_{it}) + \gamma(Cum\_non-NCE_{it}) + \mu Z_{jit} + \alpha_i + \omega_i + \theta_t + u_{it}$$

where $\alpha_i = \delta\alpha_i'$, $\omega_i = \delta\omega_i'$, $\theta_t = \delta\theta_t'$, and the standard error term $u_{it} = \delta\nu_{it}' + \varepsilon_{it}$.

Estimation methods. We begin by estimating the early retirement and the mortality equations separately, but do not use the simplest static estimation method for panel data known as the fixed effects model. Although this method may in principle provide consistent estimates in the presence of disease-specific effects, the presence of fixed effects could introduce correlations between lagged dependent variables and the residuals and might therefore lead to biased results in a dynamic panel model. In a static model, moreover, the fixed effects method does not allow us to identify the impact of time-invariant variables included among our regressors of interest.

Instead of the fixed effects model, we employ the random-effects generalized least squares method (henceforth random effects GLS) to estimate our static model. In many situations, the random effects GLS is an efficient estimator because it exploits both within and between group variations to obtain a weighted average of fixed and between effects. With this method, we can even estimate the impact of time-invariant variables such as disease, sex, educational background, and the region on lost working years in the early retirement equation.

However, our aim is to estimate not only the early retirement equation for lost working years but also the mortality equation for lost life years, and to account for the endogeneity of lost life years with respect to lost working years, and vice versa. This is not feasible with the random effects GLS. Assuming the lost working year variable is not strictly exogenous in the equation for lost life years, and vice versa, correlations between the explanatory variables and the error terms will occur. The random effects GLS estimator will then yield biased and inconsistent results. To overcome these problems, we require a panel data simultaneous equation model that takes the relationship between the equation for lost working years and the equation for lost life years. We therefore propose to employ a two-stage least squares (2SLS) model, namely the error component two-stage least squares (EC2SLS) model, which deals with the endogeneity problem by means of instrumental variables to produce consistent estimators.

We prefer EC2SLS in the static model because the presence of error components renders simpler 2SLS estimators asymptotically inefficient, whereas EC2SLS is derived as an asymptotically efficient and consistent estimator under fairly general assumptions. We perform the estimation using Baltagi's (1981) EC2SLS estimator, which improves upon the earlier Belestra and Varadharajan-Krishnakumar 2SLS estimator by using a broader set of transformations of the instruments spanning those used by the latter estimator. Because the instrumental variables must be correlated with the suspect explanatory variable, but uncorrelated with the error term, we use the variables on the right-hand side in the mortality equation as instrumental variables for the endogenous regressor of lost life years in the early retirement equation.

However, while EC2SLS leads to consistent estimates if the underlying assumptions are satisfied, it is not necessarily the most efficient estimator in our context because it does not make use of all available moment conditions, nor does it take into account the differenced structure of the residual disturbances (Ahn and Schmidt, 1995). It may even be biased when lagged dependent variables are included as regressors in both the early retirement and mortality equations. We therefore also employ a dynamic panel model, in which we account for the dynamic dependence structure that could otherwise lead to biased estimates.

To estimate the dynamic panel model, we use the generalized method of moments (GMM) proposed by Arellano and Bond (1991). In the early retirement equation, GMM corrects for the endogeneity of the lost-life-years variable, and for the potential bias introduced by the lagged lost-working-years variable in the presence of fixed effects. Likewise, in the mortality equation, GMM corrects for the endogeneity of the lost working year variable, and for the bias introduced by the lagged lost-life-years variable in the presence of fixed effects. The use of instrumental variables allows parameters to be estimated consistently in the model, which includes endogenous right-hand side variables such as the lost life year variable in the early

retirement equation. Moreover, the GMM estimator will no longer be biased by any omitted variables that are constant over time. We estimate both the one-step GMM estimator and the two-step GMM estimator for the dynamic panel to verify that the *signs* of coefficients are consistent. However, we do not report the two-step GMM results, because Arellano and Bond recommend using one-step results for inference on the *size* of the coefficients. Arellano and Bond emphasize the need to test for the hypothesis of no serial correlation in the error term of the first-differenced equation. Especially the test for second-order autocorrelation is important because second-order autocorrelation would imply inconsistent estimates. We also report the Sargan test for over-identifying restrictions to determine the validity of the instrumental variables used.

While GMM has many important advantages, it may suffer from a few drawbacks of its own. For example, biases can occur when instrumental variables are weak. In particular, when the number of time series observations is small, the GMM method is often poorly behaved; under the relevant moment conditions, lagged levels of the endogenous regressors may be weak instruments for the subsequent first-differences. Because we want to avoid basing our inference on estimation results that are specific to one method only, we use all three methods in parallel, the random effects GLS and EC2SLS methods in the static model and GMM in the dynamic model. We evaluate specification tests to determine the merits of each method for our purposes.

Testing for competing risks. Another potentially relevant source of inter-temporal interdependence is the competing risks nature of diseases as causes of early retirement. New drugs that help to prevent early retirement from one disease may give other diseases for which effective drugs have not yet been introduced the opportunity to become a more significant factor in early retirement, although typically at a higher average age of the retirees. Testing for the presence of competing risks is complicated by the fact that we do not track individuals over time, but only observe hazard rates of early retirement for groups of workers at various levels of aggregation. Even if competing risks are relevant at the individual level, they may play little or no role at higher levels of group-wise aggregation.

To design a test at the level of aggregation used in our empirical analysis of retirement determinants, we re-arrange the data and create three non-overlapping five-year time intervals during which we can observe cohorts of workers at risk as they grow old: 1988—1992; 1993—1997; and 1998—2002. While we cannot be sure that all workers under 40 years of age will be in the next age group, 40—44 years of age, five years later, this is guaranteed to be the case for all workers at risk of early retirement in any of the four age groups between 40 and 59 – namely 40—44 years, 45—49 years, 50—54 years, and 55—59 years of age – since each of these spans five years and is followed by another five-year interval in which the same cohort of workers will still be at risk of early retirement. Only workers in the 60—64 age

group will not be at risk of early retirement five years later, since they will have passed the maximum retirement age and will hence already be retired.

There are only three cohorts of workers that are at risk of early retirement in all three time intervals. The first of these cohorts, aged 40 to 44 in the first time interval, will be in the 45—49 age group in the second time interval and in the 50—54 age group in the third time interval. The second cohort, aged 45 to 49 in the first time interval, will be in the 50—54 age group in the second time interval and in the 55—59 age group in the third time interval. The third cohort, aged 50 to 54 in the first time interval, will be in the 55—59 age group in the second time interval and in the 60—64 age group in the third time interval.

We define as the fourth and fifth cohorts those that are observed over two time intervals. The fourth cohort comprises workers who are in the 55—59 age group in the first time interval and in the 60—64 age group in the second time interval. The fifth cohort comprises workers who are in the 40—44 age group in the second time interval and in the 45—49 age group in the third time interval. Finally, we define as the sixth cohort workers aged 40 to 44 in the third time interval and as the seventh cohort workers aged 60 to 64 in the first time interval; these two cohorts are hence only observed once in the re-arranged dataset.

Figures 11 and 12 display the changing retirement hazards in time intervals two and three, differentiated by the five-year age groups and disease classes, for male and female workers, respectively. These hazards rates are plotted on the vertical axis whereas the horizontal axis shows the original hazard rate in the first time interval. This captures the idea that “later” hazard rates depend on “earlier” hazard rates. It is immediately apparent that virtually all dots in both graphs lie below the imaginary diagonal for equal hazard rates, confirming the rather general hypothesis that there have been significant reductions in the retirement hazard over time.

Furthermore, the Figures 11 and 12 give us detailed insight into changing hazard rates at the level of diseases for specific age groups and cohorts of workers at risk of early retirement. Each dot in the graph is accompanied by a six-digit number starting in the first digit with number 2 or 3 to indicate the time interval of the hazard rate on the vertical axis, followed by two digits to indicate the lower bound of the age group and by three further digits to indicate the disease class. For example, the highest hazard rate on the vertical axis, at around .021, is observed in time interval 2 for male workers aged 60 to 64 suffering from musculoskeletal diseases (class 710—739). But in the first time interval, plotted on the horizontal axis, the corresponding hazard rate for musculoskeletal diseases among male workers aged 60 to 64 is even higher, at .025. In the third time interval, finally, the corresponding hazard rate has dropped to .01, more than 50 percent lower than in the preceding time interval.

The graphs also allow us to follow changing hazard rates *within* cohorts as they age. For example, the same cohort that experienced a hazard rate of early retirement of .01 due to musculoskeletal diseases when aged 60 to 64 had a hazard rate of close to .015 five years earlier when these workers were aged 55 to 59 years. This age group in turn had an original hazard rate, in the first time interval, of around .018 and a hazard rate of .009 in the third time interval. The graphical display of cohort-specific hazard rates thus suggests that changes observed within cohorts as they age can be decomposed into a time effect, which may be due to medical progress and/or legal changes, and a cohort-specific aging effect, which may be due to the age-specificity of diseases and the competing risks nature of diseases as retirement hazards.

In Figures 11 and 12, only a small group of major diseases, above all musculoskeletal, circulatory and – in the case of women – psychiatric diseases, represent relatively large hazards of early retirement and these diseases' hazard rates are at their peak in the age group 55 to 59, but appear to decline with time when the age group is held constant. Most diseases are associated with much lower hazard rates of early retirement, although together they may create a significant part of the overall disease-induced retirement hazard in younger age groups.

Figures 13 and 14 provide a closer look at the smaller hazards by focusing on diseases associated with retirement hazard rates below .005 for male and female workers, respectively. Both of these graphs is made up of eight panels so that we obtain separate displays of the hazard rates in the second time interval, 1993 to 1997, shown in the four panels on the left and of the hazard rates in the third time interval, 1998 to 2002, shown in the four panels on the right. The rows distinguish four age groups, starting with workers aged 45 to 49 and followed by those aged 50 to 54, 55 to 59 and 60 to 64, respectively. To avoid confusion, we label only a few dots in each of these panels, pertaining to the retirement hazards for neoplasm (disease class 140—239), psychiatric problems (290—359), diseases of the respiratory system (460—519), and injuries and poisoning (800—999), besides musculoskeletal (710—739) and circulatory (390—459) diseases. Only the first class code defining the range of disease classes belonging to the respective disease category is shown in the graphs.

By comparing the differences in retirement hazard rates across age groups within one and the same time interval, we can see that only some diseases are associated with substantial changes over workers' life cycle. Other diseases, especially some of those associated with relatively small retirement hazards, appear to remain a constant hazard as workers age. These observations thus support the idea of classifying diseases according to the age-specificity of their retirement hazard. Such classification, however, has to refer to one baseline time window in order to be consistent. In the first time window, for example, the male hazard rate for cancer rises from .0005 in the 40—44 age group to .0011 in the 45—49 age group to

.0021 in the 50—54 age group to .0045 in the 55—59 age group before falling slightly to .0042 in the 60—64 age group. For the same age groups, the female hazard rate for cancer rises from .0007 to .0013 to .0022 to .0042 and finally to .0050. Cancer is thus revealed to be a disease of the old. Remember that the first time window is defined by 1988-92 period; and in Figures 13 and 14 the corresponding hazard rates for age groups 45-49 and higher are shown on the horizontal axis. The hazard rates for the 40-44 age group are not shown in these graphs.

However, the same pattern is revealed for almost all diseases: hazard rates of early retirement rise with age, holding the time window constant. Even in the case of congenital anomalies, we find the hazard rate doubles every five years of a worker's increase in age, holding the time window constant. Quantifying the age-specificity of the retirement hazard of diseases is thus quite difficult. To obtain a simple measure, one could use the average age of early retirement for a given disease, the median age or the difference between these two, and we do in fact find significant differences in all of these measures. However, they ignore the overall size of the hazard, which actually varies by several orders of magnitude across diseases as Figures 11 to 14 show. Even under the competing risks hypothesis, rare diseases cannot be expected to have the same impact on the retirement hazard of other diseases as common diseases with a large retirement hazard might have.

In order to provide preliminary tests for the relevance of the competing risks in our dataset, we therefore proceed in two steps. We first report a series of non-parametric tests for significant differences in hazard rates across age groups, cohorts and changes in the hazard rates over time (Table 13). We then report a number of multiple regressions to test for the persistence of disease-specific hazard rates, controlling for sex, time, age group and cohort-specific effects as well as the impact of past hazard rates of the four diseases that pose the largest health risks in terms of early retirement (Table 14).

In Table 13, panel A, we first report results of the Wilcoxon signed-rank test and the sign test of whether changes in the age group-specific hazard rates observed between consecutive five-year time windows are significantly different from zero. We find they are, except for the changes in hazard rates in the 40—44 and 45—49 age groups. However, as the last two columns of panel A show, the two changes themselves that we observe for each age group across the three time windows in our dataset are generally not significantly different in the case of male workers, but in most age groups they are in the case of female workers. Panel B reports tests of whether changes observed in age group-specific hazard rates over time are significantly different across age groups; in almost all bilateral comparisons we find they are.

We omit to report the tests we performed to evaluate bilateral comparisons between the hazard rates within cohorts, which are found to be significantly different in all possible

comparisons except for female workers in cohort 5, when comparing the 1993—97 time window with the 1998—02 time window. Panel C of Table 13 goes on to report the results of the Wilcoxon signed rank test and the sign test of whether changes in the cohort-specific hazard rates observed between consecutive five-year time windows are significantly different from zero. We only report these tests for the three cohorts observed in all three time windows and find significant changes except for male workers in cohort 3, when comparing the 1993—97 time window with the 1998—02 time window. The last columns in Panel C report tests of whether the changes in hazard rates observed in cohorts over time are significantly different between cohorts, which is confirmed for bilateral comparisons except for the Wilcoxon signed-rank test in the case of male workers in cohort 1 and 3, when comparing the 1993—97 time window with the 1998—02 time window.

Table 14 reports the results of multivariate ordinary least squares regression analyses to test whether the four diseases representing the largest early retirement hazards have explanatory power for the hazard rates observed in subsequent time windows for other diseases in the same cohort of workers. Under the hypothesis of competing risks, we expect that changes in the major hazards have a significant impact on subsequently observed hazards from minor diseases. The first column reports our baseline model in which the agegroup-specific hazard rates for all but the major four diseases are regressed on the cohort-specific preceding hazard rates for these major diseases – musculoskeletal, circulatory, cancer and psychiatric diseases – as well as on the agegroup-specific original hazard rate for the disease in the dependent variable, a dummy for men and a dummy for the second or third time-window in which the dependent is observed. We find this simple regression explains about 80 percent of the observed variation in hazard rates for all but the four major diseases. Except for the influence of the original hazard rate, the magnitude of the coefficients is quite small; the dummy for men and the cohort-specific hazard rates for cancer and psychiatric diseases are not even significant.

The second column reports a revised model in which the insignificant disease regressors are dropped. Both the negative coefficient for the cohort-specific hazard rate of musculoskeletal diseases and the positive coefficient for the cohort-specific hazard rate of circulator disease are now larger, but their size is still dwarfed by the impact of the agegroup-specific original hazard rate for the disease in the dependent variable. A unit increase in the original hazard rate is now estimated to result in a more than two thirds of a unit increase in the explained hazard rates. The model in column 2 also appears to have a slightly better adjusted R^2, but we cannot compare this with the baseline model because the number of observations is also increased, as we now include the hazard rates for cancer and psychiatric diseases in the dependent variable. Columns 3 and 4 report variations of the second model in which we first include a set of four cohort dummies, which all turn out to be insignificant, and then three agegroup dummies, which turn out to be significant. However, including the agegroup

dummies renders both the time window dummy and the cohort-specific hazard rates for musculoskeletal diseases insignificant. With the limited number of observations we have it is not surprising that identification reaches its limits.

Summing up, we conclude there is no real evidence to support the hypothesis that competing risks are a significant factor at the level of aggregation across diseases and age groups that characterizes our study. We reach this conclusion not because we do not see a great deal of variation in the disease-specific retirement hazards across time, age groups and cohorts in our dataset. We document substantial evidence of such variation. However, the only major disease category that appears to have a robust impact on subsequent retirement hazards for other diseases is circulatory diseases, which have seen a steady decline as a retirement hazard for both men and women since the early 1980s. Based on our test results, we go on to investigate the empirical impact of new drug launches on retirement hazards without further consideration of the competing risks hypothesis.

V. Estimation Results

We begin in Tables 3 to 8 by reporting estimates of the early retirement equation for Western Germany over two periods of time – the entire sample period from 1988 to 2004 and the subperiod from 1992 to 2004. We run separate regressions for the subperiod because more detailed data on the allocation of rehabilitation services at the disease level is available in this case. For the entire period, only the aggregate number of early retirees with prior rehabilitation for all age groups is available at the disease level. For the subperiod, we have more detailed information, such as separate information on the number of early retirees with prior rehabilitation for the retirement-inducing disease and for other diseases than the retirement-inducing disease. The retirement-inducing disease might also be called "rehabilitation-resistant" since the dataset only includes actual cases of early retirement so that all rehabilitation effort spent on the disease on which the claim is based must have failed in preventing the loss of that particular worker. By contrast, prior rehabilitation efforts for other diseases might have been successful, unless they were based on a false medical diagnosis; in this case they would have been merely superfluous.

Tables 9 to 11 report the same types of regressions for total Germany, comprising both Eastern and Western Germany, but due to limited data availability in the East, only for the subperiod from 1995 to 2004. In Table 12, we also report results for the mortality equation estimated with the generalized method of moments (GMM) known as Arellano-Bond dynamic panel data estimation. in order to highlight the impact of new drug launches on the loss of life in total Germany from 1995 to 2004. To estimate the early retirement equation, we employ all three estimation methods discussed in the preceding section: first, random-effects generalized least squares regressions (Random-effects GLS); second, the error component two-stage least square estimation (EC2SLS); and third, the Arellano-bond dynamic panel data estimation (GMM).

Table 3 reports random-effects GLS early retirement regressions for Western Germany from 1988 to 2004. In model 1, we include among the explanatory variables two variables that respectively measure the number of lost life years for the retirement-inducing and for other diseases to account for mortality in the age group for which the dependent observation belongs, whereas in models 2 and 3, we include the *cumulative* number of lost life years for the retirement-specific and for other diseases; while all four measures are specific to each age group, the measures used in models 2 and 3 are likely to be considerably larger and more relevant. As explained in Table 2, the cumulative number of lost life years is determined by adding the number of lost life years in the age group to which a retiree belongs and in all higher age groups, thus comprising all age groups in which that worker might be affected by mortality from the retirement-inducing or from other diseases. In this way, our regressions control for changes in remaining life expectancy at the disease level, in line with the derivation of the demand for early retirement from the trade-off between life-cycle wealth and

health. Model 3 also differs in that we include the number of early retirees *without* prior rehabilitation as an explanatory to capture the impact of rehabilitation effort, instead of the number of early retirees *with* prior rehabilitation, as in models 1 and 2.

All regressors turn out to be statistically significant. The cumulative number of new chemical entity launches has a negative coefficient of considerable size that varies between -162 in model 3 and -220 in model 2: The greater the available stock of NCEs to treat a given disease, the smaller the number of lost working years attributed to that disease. By contrast, the stock of non-NCEs has a significant positive coefficient, but the impact of each drug in this category appears to very small. We further find that both the simple mortality measure used in model 1 and the cumulative number of lost life years used in models 2 and 3 have coefficients close to zero, but we note that the level of significance is much larger for the latter than for the former. For the impact of rehabilitation, we estimate a positive coefficient for both the number of early retirees *with* rehabilitation and the alternative variable "*without* prior rehabilitation" – a puzzle to be investigated further. All three models are consistent in estimating a much larger loss of working years for blue-collar, male and younger workers, and a much smaller loss for miners. Finally, the legal reform coming into effect at the beginning of 2001 is estimated to have reduced the loss of labor by 266 to 632 working years in every subsequent year.

Table 4 reports the results of EC2SLS regressions. Again, model 1 includes among the explanatory variables simply the number of lost life years for the retirement-inducing and for other diseases, whereas models 2 and 3 include the *cumulative* number of lost life years for the retirement-specific and for other diseases. Moreover, model 3 again uses the number of early retirees *without* prior rehabilitation as an explanatory, instead of the number of early retirees *with* prior rehabilitation that is used in models 1 and 2. All three models yield results that are virtually the same in terms of coefficient signs and very similar in terms of coefficient magnitudes when compared with the corresponding regressions in Table 3.

Table 5 reports one-step results of our dynamic panel data model estimated with GMM. As in the two preceding tables, we use the simple measure of lost life years in model 1 and the more appropriate measure of cumulative lost life years in models 2 and 3 and we use the number of early retirees *without* prior rehabilitation as an explanatory in model 3, instead of the number of early retirees *with* prior rehabilitation that is used in models 1 and 2. In addition, we use the one-year lagged number of lost working years as an endogenous regressor and see that its coefficient is between .9 and 1.0 and highly significant in all three models. These regressions thus reveal substantial persistence in health-related retirement behavior at the disease level, which is not captured by the other variables. In fact, both in terms of coefficient signs and magnitudes, the dynamic panel model largely confirms our static estimates for the variables already included in the preceding tables. Only the dummy variable for the legal reforms

coming into effect at the beginning of 2001 is now estimated to have a positive impact on the number of lost working years. If true, this would mean that the reforms had the unintended and perverse effect of increasing the burden of early retirement. However, this particular result is likely to be biased because by using first differences of all variables, the GMM fails to take into account that the reforms are not withdrawn at the end of 2001, but remain in force in all subsequent years. Note that the other dummy variables – for male, blue-collar workers and miners – have been dropped altogether because they are time-invariant and first differences of these are always zero.

Tables 6 to 8 report the results of our early retirement regressions for Western Germany from 1992 to 2004, the period for which we have more detailed information – differentiated by age group at the disease level – on the number of early retirees with prior rehabilitation for the main retirement-inducing diseases and on those with prior rehabilitation for other diseases. To capture the impact of rehabilitation efforts on early retirement in line with the testable implications of our theoretical considerations, we include three different rehabilitation variables in each of these regressions: *first*, the number of early retirees *with* prior rehabilitation for the main retirement-inducing disease, *second*, the *ratio* of the number of early retirees with prior rehabilitation for the main retirement-inducing disease to the number of early retirees with prior rehabilitation for *other* diseases, and *third*, a dummy variable for early retirees *without* any rehabilitation input. We also use the static regression models in the next two tables to illustrate the effect of including dummy variables to control for disease-specific fixed effects.

Table 6 reports results of random effects GLS regressions. Model 1 does not include disease dummies, whereas model 2 includes the full set of disease dummies for all 19 diseases, minus one to avoid perfect multicollinearity, and model 3 includes the seven disease dummies that are estimated to be statistically significant in model 2. For the main variables of interest, the estimated coefficients are similar to the ones reported in preceding tables. The coefficients for the disease dummies used in models 2 and 3 are not reported, but in line with Figures 11 to 14, the estimates suggest a significant positive impact on the loss of labor from psychiatric diseases, diseases of the nervous system and musculoskeletal diseases of the system. However, it is unclear how the smaller size of the coefficient for the stock of new chemical entities should be interpreted; since many of these drugs are disease-specific, inclusion of the disease dummies raises the issue of identification. We therefore do not recommend interpreting the coefficient for the drug variables when all or a subset of disease dummies are included.

Our main interest in the regressions reported in Tables 6 to 8 is to better understand the impact of rehabilitation services, ordered by the pension insurer, on the loss of labor from disease and injury. We first note that the dummy variable for no rehabilitation is insignificant

in all three models. As in the preceding tables, the impact of prior rehabilitation for the main retirement-inducing disease is estimated to be significant and positive. However, all three models in Table 6 show a significant negative impact of the *ratio* of the number of early retirees with prior rehabilitation for the main retirement-inducing disease to the number of early retirees with prior rehabilitation for *other* diseases. Thus, increasing the rehabilitation effort targeted at the main retirement-inducing disease *relative* to the rehabilitation effort for other diseases appears to result in at least some prevention of retirement for the targeted disease. The negative coefficient for the ratio suggests that early retirees with prior rehabilitation for the main retirement-inducing diagnosis tend to retire later than early retirees with prior rehabilitation for other diseases. This in turn tends to reduce the number of lost working years.

Table 7, reporting the results of EC2SLS to take into account the potential endogeneity of the explanatory variable lost life years, largely confirms the findings of Table 6. However, in the second model of Table 7, we now estimate a significant and large negative coefficient for the dummy variable for no rehabilitation when a set of disease-specific dummies, not reported in the table, for the nine most important diseases – such as psychiatric, musculoskeletal, cancer, circulatory, respiratory, and digestive diseases – is also included. The positive coefficient for no rehabilitation underlines the conclusion from our interpretation of Table 6 that targeted rehabilitation is effective in preventing or delaying health-related early retirement.

Table 8 reports the results of two dynamic panel models estimated with GMM. Only model 2 yields consistent estimates, however, since for model 1 the null hypothesis of no second-order autocorrelation is rejected. This specification test is not rejected for model 2, which only differs from model 1 by including three additional lags of the dependent variable as regressors. Two of these are significant, but small in magnitude. However, the first lag of the number of lost working years is highly significant and close to one, as in Table 5. Most other variables of interest also have the expected sign. Even the impact of Germany's pension law reforms coming into effect at the beginning of 2001 is now estimated as strongly negative. However, the ratio of the number of early retirees with prior rehabilitation for the main retirement-inducing disease to the number of early retirees with prior rehabilitation for other diseases is no longer significant. Also the dummy variable for early retirees without prior rehabilitation is insignificant in Table 8.

Tables 9 to 11 report the results of early retirement regressions for total Germany from 1995 to 2004. For this period, we have aggregate information over all age groups on the number of early retirees with prior rehabilitation for the main retirement-inducing disease and for other diseases. In addition, we estimate the age group-specific numbers of early retirees with prior rehabilitation for the main retirement-inducing diseases and other diseases by exploiting separate information which we have on the total number of rehabilitation services allocated to

each age groups. Because this information is not provided at the disaggregate disease-level, we make the simplifying assumption that the early retirees receiving rehabilitation for any given disease are distributed across the age groups proportionally to the distribution that we observe for the total numbers of rehabilitation services in each age group. We thus let the assumed age distribution of all rehabilitation for a given disease be determined by the percentage age distribution of rehabilitation services observed for all diseases together. We apply the same rule to determine the age distribution of the number of early retirees with prior rehabilitation for other diseases.

Table 9 and 10 report the results of random-effects GLS and EC2SLS estimations, respectively. In both tables, models 1 and 3 differ from models 2 and 4 in the way we measure rehabilitation input. Whereas models 1 and 3 include the aggregate number of early retirees with prior rehabilitation for each retirement-inducing disease and for other diseases for all age groups, models 2 and 4 include the age group-specific numbers based on the rules of estimation just described. In models 3 and 4, we also include the ratio of the number of retirees with prior rehabilitation for the main retirement-inducing diagnosis to the number of early retirees with prior rehabilitation for other diagnoses and the dummy variable for early retirees without prior rehabilitation. The signs and magnitudes of the estimated coefficients turn out largely similar to our previous estimates. Only the coefficient for the stock of new chemical entities is now much smaller, namely below 100 or half as large as in previous regressions. By contrast, the dummy variable for no rehabilitation is again estimated as highly significant and positive, confirming the effectiveness of rehabilitation in preventing or delaying health-related early retirement. Moreover, we find a significant negative coefficient for the dummy for residence in Eastern Germany. The reform of pension laws coming into effect at the beginning of 2001 still appears to have a substantial negative impact on the number of lost working years, but not nearly as large as in some of the preceding tables.

Table 11 reports one-step results of our dynamic panel data model estimated with GMM. Again the four models vary in the variables included to measure rehabilitation input. Models 1 and 3 include the aggregate number of early retirees with prior rehabilitation for each retirement-inducing disease and for other diseases, models 2 and 4 include the age group-specific numbers instead. Only models 3 and 4 include the ratio of the number of early retirees with prior rehabilitation for the main retirement-inducing disease to early retirees with prior rehabilitation for other diseases. These two models also replace the number of early retirees with prior rehabilitation for other diseases the retirement-inducing disease by the dummy variable for early retirees without prior rehabilitation. But the estimated coefficient is not significant. By contrast, the estimated coefficient for the stock of new chemical entities is highly significant and close to 200, as in previous regressions for Western Germany only. We note that only the inclusion of three additional lags of the dependent variable leads to an acceptable specification test for no second-order autocorrelation that is required for consistent

estimates. We therefore recommend to interpret only the coefficients in models 3 and 4, not in models 1 and 2 of Table 11. We also note that the estimated coefficients for the various rehabilitation measures, such as the significant positive coefficient for the ratio of the number of early retirees with prior rehabilitation for the main retirement-inducing disease to the number of those with prior rehabilitation for other diseases, are not in line with expectations and represent a puzzle. It may be that these estimates are biased because inclusion of additional lags of our dependent variable as regressors is reducing the number of time series observations and because first differencing eliminates the dummy variable for Eastern Germany in these regressions for the 1995 to 2004 subperiod.

To provide some insight into the mortality dynamics at the disease level, Table 12 reports one-step and two-step results for the mortality equation, using Arellano-Bond dynamic panel data model and GMM and lost life years in total Germany from 1995 to 2004 as the dependent variable. As explained in our methods section, two-step results can be used to verify that coefficients' signs are consistent, but only one-step results should be used for inference on the magnitude of the coefficients. The results show that the lagged lost life year variable has a significant and positive impact on the lost life year variable, whereas the stock of new chemical entities has the expected large negative effect, which is highly significant in the two-step regression. Although the one-step result does not pass the specification test for second-order autocorrelation, the effect appears to be of similar magnitude as the effect of new chemical entities that we find for early retirement in Western Germany. Non-new chemical entities also have contributed to lower mortality at the disease level. By contrast, the coefficient for lost working years is insignificant in the mortality regression. Considering the theoretical trade-off between health and wealth in the demand for early retirement, it is not necessarily surprising that the legal reform effective since the beginning of 2001 are estimated to have a significant positive impact on mortality. But that such an effect should be detectable so soon after the event calls for closer empirical scrutiny, which is beyond the present paper.

VI. Discussion

In this paper, we have used alternative econometric approaches to study the impact of new drug launches on the loss of labor from disease and injury over time. By including a second equation for the impact of new drug launches on mortality and by including changes in mortality in the set of explanatory variables of the equation for the loss of labor, we have allowed the choice of early retirement and the savings motive for old age to be interdependent, as implied by economic theory. Although the mortality measures are statistically significant in the early retirement equation and the loss of working years is statistically insignificant in the mortality equation, the size of the estimated coefficients is close to zero.

With regard to our main hypothesis, we find that regardless of econometric method, the negative impact of new chemical entities on the number of lost working years at the disease level is robust and sizable. In regressions for Western Germany alone, each new drug incorporating new chemical entities saves around 200 working years in every year that it is on the market. The corresponding coefficient in regressions for total Germany is also highly significant and has the same sign, yet only about half the absolute size.

The finding of a lower impact of new chemical entities in total Germany, compared with Western Germany alone, may reflect delayed diffusion of new drugs in Eastern Germany, which in turn might be caused by a more conservative prescribing behavior and by a lower density of medical practitioners in that region, with implications for the frequency of patient contacts, the timing of treatment initiation in case of illness and subsequent compliance. An additional factor might have been the mass restructuring of former East German industries in the early 1990s in which many less healthy workers joined the ranks of the long-term unemployed and thus ceased to be candidates for early retirement on medical grounds. Remember that our dataset for total Germany begins only in 1995, when much of the industrial restructuring had already been completed. It was beyond the scope of this study to further investigate the relative importance of these explanations for the lower impact that new chemical entities appear to have had on retirement behavior in Eastern Germany. However, we believe that regressions using the longer time series of observations we have for Western Germany more likely reveal the true impact of new chemical entities on the loss of labor from disease and injury over time.

In both total and Western Germany, the impact of *non*-new chemical entities is less clear; although statistically significant in our regressions, the estimated coefficient is too small to attribute much economic significance to the stock of non-new chemical entities in our explanation of the hazard of early retirement at the disease level.

In addition to medical technology, our study considers the utilization of health services, in the form of rehabilitation services ordered by Germany's public pension providers to prevent early retirement and the associated loss of labor. We find evidence that targeted rehabilitation has been effective at the disease level in the sense that it either prevents or delays early retirement, relative to the average age at which workers retire from a given disease without the benefit of the rehabilitation effort. This conclusion is also supported by the observation that the absence of rehabilitation effort tends to increase the loss of labor at the disease level.

However, these conclusions are tentative for three reasons: first, we only observe retiring workers, where by definition, rehabilitation targeted at the retirement-inducing disease has ultimately been unsuccessful from the public pension provider's point of view. Second, our data does not reveal the extent of rehabilitation effort for individual workers, but only at the level of groups of workers retiring for a given disease. And third, an explicit theoretical model that explains the allocation of scarce rehabilitation resources across diseases and across age groups is not available. It is unlikely that medical opportunity alone determines this allocation; instead, a pension provider may find it profitable to target rehabilitation primarily at workers where large savings in disability pension payments may be obtained even if the medical prospects for successful rehabilitation are relatively poor.

More research would be desirable to better understand the impact of rehabilitation services on early retirement. Such research should, inter alia, seek to understand the interdependence of medical rehabilitation and retraining, referred to as vocational rehabilitation by Germany's public pension providers. It would be desirable to derive testable implications from an explicit model of optimization that explains the determinants of resource allocation in the effort to restore workers health and return them to suitable jobs. To provide additional insights, future empirical research should be based on more detailed knowledge at the disease level about the specific medical opportunities for successful rehabilitation. Such knowledge would also help to understand the nature of the relationship between rehabilitation and medical technology: for example, to what extent is new medical technology, such as new drug launches, a substitute or a complement to rehabilitation services in preventing early retirement?

Other recent studies have chosen to focus on individual diseases that highlight and avoid some of the limitations of large cross-section or panel data studies, such as ours. For example, Conti et al. (2006) investigate the impact of depression on the labor force participation of elderly workers in the US and find evidence of direct and indirect effects on workers' propensity for early retirement. Ignoring the indirect effects would lead to an underestimation of the impact of depression and a potential overestimation of the impact of other diseases or life events, such as widowhood, with which depression often interacts. This finding is related to the problem of competing risks that we found difficult to identify at the level of aggregation across disease categories that characterizes our study. Knowledge about specific

interaction effects between diseases and between different medical treatment technologies would surely help to identify complementarities and competing risks in cross-section and panel studies.

In a second recent study of disability retirement due to a particular disease, Cutler et al. (2006) explore the role of improved medical treatment for cardiovascular disease in explaining the decline in disability that has been observed for more than two decades in the US. They conclude that specific pharmaceutical product innovations explain more than 50 percent of the observed reductions in disability and deaths over time, controlling for the utilization of other types of medical care inputs, such as hospitalizations.

Within the framework of cross-section and panel studies, a promising strategy to better distinguish the role of medical technology from other determinants of health and labor force participation is to broaden the scope of these types of study to include more than one country. Institutional differences in countries' health systems, pension laws and labor markets as well as differences in access to medical technology provide natural experiments that are waiting to be evaluated. An example for this type of study is provided by Kalwij and Vermeulen (2005) who study the impact of multi-dimensional measures of health on the elderly's labor force participation in a cross-section of 11 European countries based on the Survey of Health, Aging and Retirement in Europe (SHARE). They find substantial differences in the importance of different attributes of health across countries. But these authors do not consider the role of medical technology.

International panel studies may also provide an opportunity to better understand the problem of competing risks in determinants of early retirement and death due to disease and injury. It is difficult to identify competing risks models in the absence of counterfactual observations; see Honoré and Lleras-Muney (2006) for the methodological state-of-the-art in the estimation of competing risks when their observability is interdependent due to endogenous censoring in a given set of subjects at risk. Different institutional and epidemiological conditions as well as differential access to medical technology across countries might help to identify competing risks in appropriate multinational panel studies in the future. This would not only be desirable because we could then avoid overestimating the impact of the "earlier" medical technology innovations and underestimating the impact of "later" innovations. It might also help to identify the type of medical technologies where the competing risks problems is likely to create significant disincentives and coordination problems for investors.[3]

However, to fully understand the role of medical technology, we will also need new contributions from economic theory – address in particular the following sets of issues: *First*,

[3] See Dow et al. (1999) for an analysis of investment incentives in the presence of competing risks.

the relationship between early retirement and endogenous longevity, which matters because medical innovation may have direct and indirect effects on the propensity for early retirement. The *direct* effect stems from better medical treatment that helps to maintain or even improve people's health, and labor productivity, whereas the *indirect* effect results from changes in the private incentives for early retirement. On the one hand, a longer life expectancy and the prospect of a higher quality of life may increase the demand for private savings and may hence increase people's labor force participation. On the other hand, people may want to enjoy some of their improved health by increasing their consumption of leisure.

Secondly, economic theory should address the relationship between the demand for early retirement and the demand for medical care and should provide a testable health production function that encompasses potential complementarities, such as the relationship between acute care and early retirement, and potentially substitutional relationships, such as that between early retirement and the utilization of rehabilitation services. On this basis, the role of innovations in medical technology in changing the relevant trade-offs and constraints in the production and maintenance of health over the life cycle could be investigated in much greater detail.

VII. Concluding Remarks

In this paper, we have explored the evolution of early retirement due to disease and injury in the German labor force between 1988 and 2004, combining for the first time three relevant datasets: the disability retirement records from the German Federation of Public Pension Providers, the IMS Health Drug Launches database and the WHO Mortality Database. Using a variety of econometric methods to exploit the pseudo-panel structure of the combined dataset, we find that in Western Germany alone each new chemical entity has on average saved around 200 working years in every year of the observation period. Controlling for individual determinants of health-related retirement, such as worker's age, sex and type of work, we also find evidence that the 2001 reform of pension laws has led to further reductions in the loss of labor from disease and injury.

In addition, we use non-parametric tests and regression analysis to investigate the relevance of the competing risks hypothesis in the empirical determinants of early retirement at the disease level. We argue that the presence of competing risks in individual retirement behavior is unlikely to affect the conclusions suggested by our empirical estimates of the impact of new drug launches on retirement timing in Germany. We therefore conclude that our findings provide a first estimate of an important part of the social gains from reductions in early retirement that were brought about by new pharmaceutical products in Germany between 1988 and 2004. The total welfare gains are likely to be substantially larger as they must include an aggregate measure of workers' willingness-to-pay for potential health benefits and the gain in lifetime personal income that voluntary deferral of retirement affords. Nonetheless, we believe our findings could help to assess the extent to which Germany, and other OECD countries, might want to raise the official maximum retirement age and change eligibility criteria for health-related early retirement in response, or anticipation, to future progress in medical technology.

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Appendices

Data sources

We obtained the required dataset from the Federation of Public Pension Providers in Germany (VDR), titled "VDRSY Version 1.01 – Der Statistik-Tabellen-Viewer des VDR," and additional data on variables which serve as exogenous controls in our study, such as a detailed chronological list of all the relevant legal changes affecting German retirement behavior during the sample period. We also obtained the IMS Health Drug Launches Database, now named the IMS Life Cycle New Product Focus, and the WHO Mortality Database.

The drug launches documented in the IMS Health Drug Launches database are classified by therapeutic category in line with the the Anatomical Classification of Pharmaceutical Products that has been developed and maintained by the European Pharmaceutical Marketing Research Association (EphMRA) and is the intellectual property of this association. The classification is very detailed, with more than 500 therapeutic classifications (A1A1-V3X9) at the most detailed level, e.g. mouth antiseptics and anti-infectives (A1A2). At the highest level, there are 16 categories (A-V), e.g. alimentary tract and metabolism (A). We use 13631 records of individual product introductions and 86 new chemical entities for 1982-2004 in Germany. Each record in the IMS Health Drug Launches database consists of the information on the date of product launch, the active ingredients of the product, the therapeutic class of the product and the corporation.

Because Germany was separated into East and West Germany before 1990, the IMS data provide information on drug launches for two categories, named West Germany and Germany. Data on drug launches in the West Germany category are available from 1982 to 1992 and in the Germany category from 1985 to 2004. Thus, between 1982 and 1984 data on drug launches are recorded in the West Germany category only, between 1985 and 1992 in both the West Germany and Germany categories, and then, from 1993 to 2004, in the Germany category only. To get the number of new drug launches in total Germany from 1982 to 2004, we add the number of new drug launches in the two categories. But as they are reported in both categories during 8 years from 1985 to 1992, we carefully examine the data as to make sure that new drug launches are not counted twice if they appear in both the West Germany and Germany categories.

The International Classification of Diseases (ICD) is under the responsibility of the World Health Organization (WHO) and is used to classify diseases and other health problems. In German disability retirement, the 10th Revision of the International Classification of Diseases (ICD-10) came into use as from 2000 and replaced the 9th Revision of the International Classification of Diseases (ICD-9). This change makes it necessary to match between ICD-9 and ICD-10 for the period under consideration (1988-2004). The ICD-9 contains 19 so-called

chapters, whereas ICD-10 contains 21 chapters. These chapters constitute disease categories on a highly aggregate level. We performed the matching between the two classifications on the basis of 19 ICD-10 chapters. Matching on a more disaggregate level cannot be easily performed since there are 1182 three-character categories in ICD-9, while there are 2036 three-character categories in ICD-10. Very often the disease categories cannot be translated on a one-by-one basis. However, matching on the level of the 19 main disease categories can be performed with a relatively high accuracy.

Certain infectious and parasitic diseases correspond with ICD-10 codes A00-B99 and were matched with ICD-9 codes 001-139, neoplasms correspond with ICD-10 codes C00-D48 and were matched with ICD-9 codes 140-239, diseases of the blood and blood-forming organs and certain disorders involving the immune mechanism correspond with ICD-10 codes D50-D90 and were matched with ICD-9 codes 280-289, endocrine, nutritional and metabolic diseases correspond with ICD-10 codes E00-E90 and were matched with ICD-9 codes 240-279, mental and behavioral disorders correspond with F00-F99 and were matched with ICD-9 codes 290-319, diseases of the nervous system correspond with ICD-10 codes G00-G99 and were matched with ICD-9 codes 320-359, diseases of the eye and adnexa correspond with ICD-10 codes H00-H59 and were matched with ICD-9 codes 360-379, diseases of the ear and mastoid process correspond with ICD-10 codes H60-H95 and were matched with ICD-9 codes 380-389, diseases of the circulatory system correspond with ICD-10 codes I00--I99 and were matched with ICD-9 codes 390-459, diseases of the respiratory system correspond with ICD-10 codes J00-J99 and were matched with ICD-9 codes 460-519, diseases of the digestive system correspond with ICD-10 codes K00-K93 and were matched with ICD-9 codes 520-579, diseases of the skin and subcutaneous tissue correspond with ICD-10 codes L00-L99 and were matched with ICD-9 codes 680-709, diseases of the musculoskeletal system and connective tissue correspond with ICD-10 codes M00-M99 and were matched with ICD-9 codes 710-739, diseases of the genitourinary system correspond with ICD-10 codes N00-N99 and were matched with ICD-9 codes 580-629, pregnancy, childbirth and the puerperium correspond with ICD-10 codes O00-O99 and were matched with ICD-9 codes 630-676, certain conditions originating in the perinatal period correspond with ICD-10 codes P00-P96 and were matched with ICD-9 codes 760-779, congenital malformations, deformations and chromosomal abnormalities Q00-Q99 and were matched with ICD-9 codes 740-759, symptoms, signs and abnormal clinical and laboratory findings, not elsewhere classified correspond with ICD-10 codes R00-R99 and were matched with ICD-9 codes 780-799, and injury, poisoning and certain other consequences of external causes correspond with ICD-10 codes S00-T98 and were matched with ICD-9 codes 800-999.

List of Symbols

c	Consumption
H	Health
i	Index of diseases
j	Index of rehabilitation services received for disease i (J=1) and for other diseases (j=2), respectively
k	Lag for drug launches
M	Medical care
NCE	New chemical entities
Non-NCE	New drugs that do not incorporate new chemical entities
p	Price
q	Effectiveness or quality of medical care
r	Pension annuity
R	Time of retirement
t	Index of time
$\tilde{t}$	Time at which worker is indifferent between continued work and retirement
T	Remaining life in years
U	Utility
V	Indirect utility function
W	Wealth
X	Vector of fixed effects influencing retirement timing
y	Functional relationship between health and labor productivity
Y	Percentage of early retirees who received rehabilitation services
Z	Vector of mortality from disease I and other causes at various age intervals

Tables

Table 1: Drugs and Diseases

IMS drug class(es)		ICD 9 codes	ICD 9 disease class(es)	Cumulative number of NCE from 1982 to 2004	Cumulative number of Non-NCE from 1982 to 2004
A	Alimentary Tract and Metabolism	520-579	Diseases of the digestive system; endocrine, nutritional and metabolic diseases	11	2526
B	Blood and Blood Forming Organs	280-289	Diseases of the blood and blood-forming organs and certain disorders involving the immune mechanism	7	249
C	Cardiovascular System	390-459	Diseases of the cardiovasculary system	20	2220
D	Dermatologicals	680-709	Diseases of the skin and subcutaneous tissue	3	880
G	Genitourinary System and Sex Hormones	580-629, 630-676	Diseases of the genitourinary system	5	769
H	Systematic Hormonal Preparations (excluding sex hormones)	240-279	Hormonal disorders	1	135
J	General Anti-Infectives, Systemic, Parasitology	001-139	Certain infectious and parasitic diseases	7	819
K	Hospital Solutions			0	11
L	Cytostatica, Antineoplastic and Immunomodulating Agents	140-239	Neoplasm	9	287
M	Musculoskeletal System	710-739	Diseases of the musculoskeletal system and connective tissue	6	874
N	Central Nervous System (CNS)	290-319, 320-359	Mental and behavioral disorders, diseases of the nervous system	7	1879
P	Parasitology			1	20
R	Respiratory System	460-519	Diseases of the respiratory system	4	1456
S	Sensory Organs	360-379, 380-389	Diseases of the eye and adnexa; diseases of the ear and mastoid process	1	306
T	Diagnostic Agents	740-759, 760-779, 780-799, 800-999	Injuries, poisoning and abnormalities	2	156
V	Various		Various	2	958
			Total	86	13545

Table 2: Variable Descriptions

Variable	Description	Data Availability	Mean	Std. Dev.
A. Endogenous variables				
Lost working years (LWY)	The number of lost working years in each age group by disease, sex, region, and Year	Eastern and Western Germany from 1995 to 2004	2393.47	7327.35
Lost working years in West (LWYW)	The number of lost working years in each age group by disease, sex, region and year in Western Germany.	Western Germany from 1988 to 1992 and from 1995 to 2004; total Germany in 1993 and 1994	3323.39	9174.13
Lost life years (LLY)	The number of lost life years for the same diagnosis by age group, disease, sex and year.	Total Germany from 1988 to 2004	94480.1	396244.4
B. Variables appearing in both early retirement and mortality equations ($\tilde{R}_{it}$ and L_{it})				
New chemical entities	The cumulative number of new chemical entities for the main therapeutic areas over years since 1982.	Germany from 1985 to 2004 and Western Germany from 1982 to 1992	3.359	3.85
Non-new chemical entities	The cumulative number of non-new chemical entities for the main therapeutic areas over years since 1982.	Germany from 1985 to 2004 and Western Germany from 1982 to 1992	487.306	527.105
Post reform2001	Dummy variable for post reform2001 is equal to 1 if year is in 2001and afterwards, and equal to 0 otherwise.		0.235	0.424
Age group	Age group is equal to 39, 44, 49, 54, 59, 64 corresponding to the end of each age interval: under 40, 40-44, 45-49, 50-54, 55-59, 60-64, respectively.		51.5	8.539
Dummy for Male	Dummy variable for male is equal to 1 if the early retiree is male, and 0 for female.		0.5	0.5
C. Additional variables appearing in the early retirement equation ($\tilde{R}_{it}$)				
Lost life years for other diagnoses (LLY other diagnoses)	The number of lost life years for other diagnoses by age group, disease, sex and year.	Total Germany from 1988 to 2004	1700642	2518492
Cumulative number of lost life years (Cumulative LLY)	The cumulative number of lost life years for the retirement-inducing disease – by age group, disease, sex and year – is the sum of lost life years in the same and in higher age groups, for a given retirement-inducing disease.	Total Germany from 1988 to 2004	482641.8	1026006
Cumulative number of lost life years for other diagnoses (Cumulative LLY other diagnoses)	The cumulative number of lost life years for other diagnoses – by age group, disease, sex and year – is the sum of lost life years in the same and in higher age groups, for all other diseases apart from the given retirement-inducing disease.	Total Germany from 1988 to 2004	8687518	1555883

Table 2 continued: Variable Descriptions

Rehabilitation (Reha1)	The number of early retirees with prior rehabilitation for the same diagnosis for all age groups by disease, sex, region and year.	Eastern and Western Germany from 1995 to 2004	155.512	749.602
Rehabilitation for other diagnoses (Reha2)	The number of early retirees with prior rehabilitation due to other diagnoses for all age groups by disease, sex, region, and year.	Eastern and Western Germany from 1995 to 2004	17.615	146.157
Rehabilitation by age groups	The number of early retirees with prior rehabilitation for the same disease in each age group by disease, sex, region and year.	Eastern and Western Germany from 1995 to 2004	26.2	146.916
Rehabilitation for other diagnoses by age groups	The number of early retirees with prior rehabilitation for other diseases in each age group by disease, sex, region and year.	Eastern and Western Germany from 1995 to 2004	2.63	24.677
Reha1/Reha2	The ratio of the number of early retirees with prior rehabilitation for the retirement-inducing disease to those with prior rehabilitation for other diseases.	Eastern and Western Germany from 1995 to 2004	22.64	20.98
Prior rehabilitation (in West)	The number of early retirees with prior rehabilitation for all age groups by disease, sex and year in Western Germany.	Western Germany from 1988 to 2004	368.77	1422.19
No rehabilitation1	The number of early retirees without prior rehabilitation services for all age groups by disease, sex, region and year.	Eastern and Western Germany from 1995 to 2004	254.696	1064
No rehabilitation2	The number of early retirees without prior rehabilitation services for all age groups by disease, sex, region and year in Western Germany.	Western Germany from 1988 to 2004	434.11	1476.51
Blue-collar workers	Dummy variable for blue-collar workers is equal to 1 if the early retiree is a blue-collar worker, and otherwise 0		0.333	0.4714
Miners	Dummy variable for miners is equal to 1 if the early retiree is a miner, and otherwise 0		0.333	0.4714
East German residence	Dummy variable for East German residence is equal to 1 if the early retiree is in Eastern Germany, and otherwise 0		0.5	0.5
No reha dummy	Dummy variable is equal to 1 if reha1=0 and reha2=0, otherwise 0.			

Note on data availability: *In the year 2000, the disease-specific retirement data was subject to the change in the disease classification from ICD9 to ICD10, so that some early retirees have not been classified into one of the 17 aggregate disease classes in the "Age table" in the source dataset provided by the Federation of Public Pension Providers (VDR). Hence, in each disease class the sum of early retirees across age groups from the "Age table" is smaller than the sum of those with and without prior rehabilitation from the "Rehabilitation table" in 2000. To overcome this discrepancy, we distribute the total number of early retirees from the "Rehabilitation table" in accordance with the percentage distribution of early retirees in the "Age table" across age groups. – We do not have separate information on the numbers of early retirees in Eastern and Western Germany in 1993 and 1994. To estimate these numbers, we distribute the number of early retirees in total Germany in 1993 and 1994 according to the percentage distribution of early retirees in Eastern and Western Germany in 1995.*

Table 3: Results of Random Effects GLS Regression for Lost Working Years in Western Germany from 1988 to 2004

Coefficients	1*	2	3+
New chemical entities	**-177.76**	**-219.60**	**-162.33**
Z	-8.67	-10.56	-7.79
P>|z|	0.000	0.000	0.000
Non-new chemical entities	**1.15**	**1.24**	**1.20**
Z	10.21	10.92	10.53
P>|z|	0.000	0.000	0.000
Cumulative lost life years (LLY)	**0.00**	**0.00**	**0.00**
Z	7.72	15.73	13.33
P>|z|	0.000	0.000	0.000
Cumulative LLY other diagnoses	**-0.00**	**-0.00**	**-0.00**
Z	-2.32	-11.18	-11.74
P>|z|	0.020	0.000	0.000
Prior rehabilitations	**2.54**	**2.42**	**2.61**
Z	68.05	64.75	64.34
P>|z|	0.000	0.000	0.000
Post reform 2001	**-266.09**	**-523.87**	**-631.70**
Z	-5.09	-9.44	-11.39
P>|z|	0.000	0.000	0.000
Blue-collar workers	**2401.38**	**2437.30**	**1677.79**
Z	5.08	5.10	3.54
P>|z|	0.000	0.000	0.000
Miners	**-1915.51**	**-1957.49**	**-2095.60**
Z	-4.05	-4.10	-4.42
P>|z|	0.000	0.000	0.000
Age group	**-48.21**	**-93.36**	**-98.04**
Z	-1.87	-3.99	-4.22
P>|z|	0.062	0.000	0.000
Dummy for male	**1020.62**	**1236.77**	**1027.48**
Z	2.64	3.17	2.65
P>|z|	0.008	0.002	0.008
Cons	**4330.97**	**9861.32**	**10476.80**
Z	3.25	7.00	7.48
P>|z|	0.001	0.000	0.000
Sigma_u	7084.26	7168.56	7116.72
Sigma_e	2621.59	2597.58	13682602.58
Rho	0.88	0.88	0.88
No. of observations	23256	23256	23256
No. of groups	1368	1368	1368
Obs. per group: min	17	17	17
Average	17.0	17.0	17.0
Max	17	17	17
Wald chi^2 (10)	5269.71	5795.20	5740.50
Prob > chi^2	0.000	0.000	0.000
R^2: within	0.1776	0.1929	0.1898
Between	0.2792	0.2904	0.3077
Overall	0.2697	0.2812	0.2966

* **Lost life years (LLY)** is used instead of **Cumulative LLY** and **LLY other diagnoses** is used instead of **Cumulative LLY other diagnoses.**

+ **No rehabilitation 2** is used instead of **Prior rehabilitations.**

Table 4: Results of Error Component Two-stage Least-square Regressions (EC2SLS) for Lost Working years in Western Germany from 1988 to 2004, treating Lost Life Years as an Endogenous Regressor

Coefficients	1*	2	3[+]
New chemical entities	**-167.89**	**-229.75**	**-170.78**
Z	-3.82	-11.00	-8.16
P>|z|	0.000	0.000	0.000
Non-new chemical entities	**1.29**	**1.12**	**1.08**
Z	11.29	9.65	9.30
P>|z|	0.000	0.000	0.000
Cumulative lost life years (LLY)	**0.01**	**0.00**	**0.00**
Z	1.51	6.29	4.85
P>|z|	0.132	0.000	0.000
Cumulative LLY other diagnoses	**0.00**	**-0.00**	**-0.00**
Z	1.98	-12.06	-12.58
P>|z|	0.048	0.000	0.000
Prior rehabilitations	**2.47**	**2.46**	**2.65**
Z	47.54	64.89	64.25
P>|z|	0.000	0.000	0.000
Post reform 2001	**-211.00**	**-542.17**	**-650.85**
z	-3.38	-9.73	-11.69
P>|z|	0.001	0.000	0.000
Blue-collar workers	**2422.09**	**2427.08**	**1652.72**
z	0.95	5.14	3.51
P>|z|	0.342	0.000	0.000
Miners	**-1939.72**	**-1945.55**	**-2083.50**
z	-0.76	-4.12	-4.42
P>|z|	0.447	0.000	0.000
Age group	**-169.44**	**-105.24**	**-109.59**
z	-1.25	-4.53	-4.72
P>|z|	0.213	0.000	0.000
Cons	**9866.71**	**12077.70**	**12538.42**
z	1.42	8.48	8.81
P>|z|	0.157	0.000	0.000
sigma_u	43838.26	7177.57	7118.22
sigma_e	2909.24	2638.57	2623.88
rho	1.00	0.88	0.88
No. of observations	23256	23256	23256
No. of groups	1368	1368	1368
Obs. per group: min	17	17	17
average	17.0	17.0	17.0
max	17	17	17
Wald chi^2 (9)	4943.91	5569.70	5572.27
Prob > chi^2	0.000	0.000	0.000
R^2: within	0.1737	0.1907	0.1879
between	0.1707	0.2955	0.3119
overall	0.1696	0.2856	0.3002

* **Lost life years (LLY)** is used instead of **Cumulative LLY** and **LLY other diagnoses** is used instead of **Cumulative LLY other diagnoses.**

[+] **No rehabilitation 2** is used instead of **Prior rehabilitations.**

Table 5: Results of the Arellano-Bond Dynamic Panel Data Regressions for Lost Working Years in Western Germany from 1988 to 2004

Coefficients	1*	2	3+
Results	*One-step*	*One-step*	*One-step*
Lagged lost working years	**0.97**	**0.98**	**0.91**
z	207.85	208.70	233.69
P>|z|	0.000	0.000	0.000
New chemical entities	**-253.67**	**-284.02**	**-199.97**
z	-15.95	-17.00	-12.55
P>|z|	0.000	0.000	0.000
Non-new chemical entities	**-0.16**	**-0.53**	**-0.61**
z	-1.63	-5.19	-6.33
P>|z|	0.103	0.000	0.000
Lost life years (LLY)	**-0.00**	**-0.00**	**-0.00**
z	-1.00	-9.04	-19.44
P>|z|	0.316	0.000	0.000
LLY other diagnoses	**0.00**	**0.00**	**0.00**
z	6.66	7.33	8.86
P>|z|	0.000	0.000	0.000
Prior rehabilitation	**0.05**	**0.10**	**1.47**
z	1.63	3.11	49.78
P>|z|	0.104	0.002	0.000
Post reform 2001	**243.35**	**259.27**	**250.95**
z	7.33	7.79	7.96
P>|z|	0.000	0.000	0.000
Cons	**37.80**	**68.01**	**67.71**
z	6.35	9.30	9.77
P>|z|	0.000	0.000	0.000
No. of observations	20520	20520	20520
No. of groups	1368	1368	1368
Obs. per group: min	15	15	15
average	15	15	15
max	15	15	15
Wald chi^2 (7)	76110.46	75393.07	75393.07
Sargan test of over-identifying restrictions:			
chi^2 (119)	13758.18	13502.63	12642.36
Prob > chi^2	0.000	0.000	0.000
Arellano-Bond test that average autocovariance in residuals of order 1 is 0:			
H0: no autocorrelation			
z	-49.09	-49.05	-47.44
Pr > z	0.000	0.000	0.000
Arellano-Bond test that average autocovariance in residuals of order 2 is 0:			
H0: no autocorrelation			
z	-5.18	-5.11	-6.01
Pr > z	0.000	0.000	0.000

* **Lost life years (LLY)** is used instead of **Cumulative LLY** and **LLY other diagnoses** is used instead of **Cumulative LLY other diagnoses.**

+ **No rehabilitation 2** is used instead of **Prior rehabilitations.**

Table 6: Results of Random Effects GLS Regressions for Lost Working Years in West Germany from 1992 to 2004

Coefficients	1	2[a]	3[b]
New chemical entities	**-211.26**	**-169.42**	**-165.41**
z	-8.10	-6.15	-6.08
P>|z|	0.000	0.000	0.000
Non-new chemical entities	**1.03**	**0.99**	**0.97**
z	6.23	5.67	5.60
P>|z|	0.000	0.000	0.000
Cumulative lost life years (LLY)	**0.00**	**0.00**	**0.00**
z	15.11	15.83	15.96
P>|z|	0.000	0.000	0.000
Cumulative LLY other diagnoses	**-0.00**	**-0.00**	**-0.00**
z	-4.94	-5.48	-5.46
P>|z|	0.000	0.000	0.000
Rehabilitation (Reha1)	**2.47**	**2.35**	**2.35**
z	48.99	46.71	46.71
P>|z|	0.000	0.000	0.000
Reha1/Reha2	**-7.21**	**-7.62**	**-7.61**
z	-3.83	-4.06	-4.06
P>|z|	0.000	0.000	0.000
No reha dummy	**144.93**	**217.36**	**212.01**
z	1.00	1.53	1.49
P>|z|	0.316	0.127	0.136
Post reform 2001	**-418.99**	**-466.79**	**-467.48**
z	-7.00	-7.73	-7.75
P>|z|	0.000	0.000	0.000
Blue-collar workers	**2558.1**	**2573.12**	**2573.22**
z	5.29	5.88	5.89
P>|z|	0.000	0.000	0.000
Miners	**-2174.69**	**-2228.60**	**-2227.99**
z	-4.49	-5.09	-5.10
P>|z|	0.000	0.000	0.000
Age group	**-84.72**	**-79.64**	**-79.43**
z	-3.53	-3.63	-3.62
P>|z|	0.000	0.000	0.000
Dummy for male	**1140.34**	**1126.70**	**1125.17**
z	2.87	3.13	3.13
P>|z|	0.004	0.002	0.002
Cons	**7838.29**	**6749.68**	**6630.66**
z	5.13	4.19	4.64
P>|z|	0.000	0.000	0.000
sigma_u	7227.46	6550.61	6544.75
sigma_e	2605.30	2605.30	2605.30
rho	0.89	0.86	0.86
No. of observations	17784	17784	17784
No. of groups	1368	1368	1368
Obs. per group: min	13	13	13
average	13.0	13.0	13.0
max	13	13	13
Wald chi^2	3346.46 (12)	3952.25 (30)	3953.36 (19)
Prob > chi^2	0.000	0.000	0.000
R^2: within	0.1466	0.1488	0.1488
between	0.2708	0.4411	0.4401
overall	0.2602	0.4161	0.4152

[a] Dummy variables for each of 19 diseases, minus one.
[b] Dummy variables for seven major diseases.

Table 7: Results of Error Component Two-stage Least-square Regressions (EC2SLS) for Lost Working years in Western Germany from 1992 to 2004, treating Lost Life Years as an Endogenous Regressor

Coefficients	1	2[c]
New chemical entities	**-189.65**	**-117.88**
z	-7.21	-2.63
P>|z|	0.000	0.009
Non-new chemical entities	**0.79**	**1.65**
z	4.63	3.27
P>|z|	0.000	0.001
Cumulative lost life years (LLY)	**0.00**	**0.01**
z	6.98	2.63
P>|z|	0.000	0.008
Cumulative LLY other diagnoses	**-0.00**	**-0.00**
z	-5.62	-1.00
P>|z|	0.000	0.317
Rehabilitation (Reha1)	**2.52**	**2.28**
z	49.70	28.19
P>|z|	0.000	0.000
Reha1/Reha2	**-7.43**	**-7.27**
z	-3.91	-3.78
P>|z|	0.000	0.000
No reha dummy	**172.79**	**258.41**
z	1.20	1.80
P>|z|	0.231	0.072
Post reform 2001	**-456.97**	**-492.81**
z	-7.53	-7.65
P>|z|	0.000	0.000
Blue-collar workers	**2548.90**	**2586.88**
z	5.70	6.31
P>|z|	0.000	0.000
Miners	**-2161.13**	**-2258.55**
z	-4.83	-5.50
P>|z|	0.000	0.000
Age group	**-95.53**	**-36.76**
z	-4.25	-1.01
P>|z|	0.000	0.312
Cons	**9838.93**	**2683.223**
z	6.65	0.77
P>|z|	0.000	0.440
sigma_u	7219.73	7302.02
sigma_e	2842.56	2665.84
rho	0.87	0.88
No. of observations	17784	17784
No. of groups	1368	1368
Obs. per group: min	13	13
average	13.0	13.0
max	13	13
Wald chi^2	3198.16 (11)	3750.04 (20)
Prob > chi^2	0.000	0.000
R^2: within	0.1432	0.1400
between	0.2854	0.4325
overall	0.2729	0.4072

[c] Dummy variables for 9 major diseases.

Table 8: Results of the Arellano-Bond Dynamic Panel Data Regressions for Lost Working Years in Western Germany from 1992 to 2004

Coefficients	1	2
Results	*One-step*	*One-step*
Lagged lost working years	**0.90**	**0.96**
z	159.24	92.53
P>|z|	0.000	0.000
Lagged lost working years (-2)		**-0.10**
z		-8.01
P>|z|		0.000
Lagged lost working years (-3)		**-0.01**
z		-0.62
P>|z|		0.536
Lagged lost working years (-4)		**0.06**
z		6.69
P>|z|		0.000
New chemical entities	**-351.67**	**-171.24**
z	-18.57	-7.95
P>|z|	0.000	0.000
Non-new chemical entities	**0.24**	**0.67**
z	1.90	4.81
P>|z|	0.058	0.000
Cumulative lost life years (LLY)	**0.00**	**0.00**
z	11.32	8.18
P>|z|	0.000	0.000
Cumulative LLY other diagnoses	**0.00**	**0.00**
z	6.30	8.38
P>|z|	0.000	0.000
Rehabilitation (Reha1)	**0.68**	**0.16**
z	14.04	2.95
P>|z|	0.000	0.003
Reha1/Reha2	**-2.65**	**1.24**
z	-2.72	0.96
P>|z|	0.006	0.337
No reha dummy	**-58.34**	**-26.30**
z	-0.59	-0.25
P>|z|	0.555	0.803
Post reform 2001	**248.36**	**-183.84**
z	6.54	-4.61
P>|z|	0.000	0.000
Cons	**71.24**	**123.64**
z	6.73	9.44
P>|z|	0.000	0.000
No. of observations	15048	10944
No. of groups	1368	1368
Obs. per group: min	11	8
average	11	8
max	11	8
Wald chi^2	61842.24 (9)	39877.83 (12)
Sargan test of over-identifying restrictions:		
chi^2	8518.21 (65)	4164.02 (56)
Prob > chi^2	0.000	0.000
Arellano-Bond test that average autocovariance in residuals of order 1 is 0: H0: no autocorrelation		
z	-36.87	-61.81
Pr > z	0.000	0.000
Arellano-Bond test that average autocovariance in residuals of order 2 is 0: H0: no autocorrelation		
z	-10.91	0.85
Pr > z	0.000	0.396

Table 9: Results of Random Effects GLS Regressions for Lost Working Years in Total Germany from 1995 to 2004

Coefficients	1	2[†]	3[#]	4[†#]
New chemical entities	**-75.62**	**-86.88**	**-80.64**	**-93.15**
z	-5.08	-6.10	-5.39	-6.49
P>|z|	0.000	0.000	0.000	0.000
Non-new chemical entities	**-0.03**	**0.04**	**-0.01**	**0.06**
z	-0.28	0.39	-0.09	0.61
P>|z|	0.779	0.695	0.932	0.542
Cumulative lost life years (LLY)	**0.00**	**0.00**	**0.00**	**0.00**
z	17.99	17.68	17.81	17.55
P>|z|	0.000	0.000	0.000	0.000
Cumulative LLY other diagnoses	**0.00**	**0.00**	**0.00**	**0.00**
z	6.41	6.38	6.91	6.80
P>|z|	0.000	0.000	0.000	0.000
Rehabilitation (Reha1)	**2.03**	**14.81**	**2.70**	**18.54**
z	35.09	56.56	63.28	94.13
P>|z|	0.000	0.000	0.000	0.000
Rehabilitation for other	**3.03**	**17.99**	**198.38**	**273.45**
diagnoses (Reha 2) or	16.63	21.02	2.25	2.98
No reha dummy P>|z|	0.000	0.000	0.024	0.003
Reha1/Reha2			**-4.13**	**-4.59**
z			-3.79	-4.48
P>|z|			0.000	0.000
Post reform 2001	**-86.86**	**-82.94**	**-79.37**	**-81.24**
z	-2.79	-2.81	-2.50	-2.69
P>|z|	0.005	0.005	0.012	0.007
Blue-collar workers	**1656.00**	**1658.33**	**1694.14**	**1710.71**
z	6.47	6.74	6.69	7.01
P>|z|	0.000	0.000	0.000	0.000
Miners	**-1420.79**	**-1313.40**	**-1337.48**	**-1239.00**
z	-5.55	-5.31	-5.27	-5.05
P>|z|	0.000	0.000	0.000	0.000
East German residence	**-1652.25**	**-1556.49**	**-1588.92**	**-1501.99**
z	-7.90	-7.72	-7.67	-7.51
P>|z|	0.000	0.000	0.000	0.000
Age group	**-31.27**	**-35.86**	**-29.27**	**-34.42**
z	-2.43	-2.89	-2.29	-2.79
P>|z|	0.015	0.004	0.022	0.005
Dummy for male	**247.39**	**250.94**	**233.36**	**245.09**
z	1.17	1.23	1.11	1.21
P>|z|	0.242	0.218	0.265	0.226
Cons	**1590.76**	**1808.95**	**1117.46**	**1364.81**
z	1.85	2.19	1.30	1.65
P>|z|	0.064	0.028	0.193	0.099
sigma_u	5408.50	5172.28	5355.05	5128.02
sigma_e	1802.10	1688.52	1813.42	1704.32
rho	0.90	0.90	0.90	0.90
No. of observations	27350	26492	27350	26492
No. of groups	2736	2688	2736	2688
Obs. per group: min	8	1	8	1
average	10.0	9.9	10.0	9.9
max	10	10	10	10
Wald chi^2	5943.26 (12)	11300.41 (12)	5651.07 (13)	10736.92 (13)
Prob > chi^2	0.000	0.000	0.000	0.000
R^2: within	0.1656	0.2920	0.1552	0.2786
between	0.2771	0.3564	0.2884	0.3631
overall	0.2686	0.3510	0.2783	0.3562

[†] **Rehabilitation by age groups** is used instead of **Rehabilitation (Reha1)** and **Rehabilitation for other diagnoses by age groups** is used instead of **Rehabilitation for other diagnoses (Reha 2).**

[#] **No reha dummy** is used instead of **Rehabilitation for other diagnoses (Reha 2).**

Table 10: Results of Error Component Two-stage Least-square Regressions (EC2SLS) for Lost Working Years in Total Germany from 1995 to 2004, treating Lost Life Years as an Endogenous Regressor

Coefficients	1	2†	3	4†
New chemical entities	**-64.40**	**-77.97**	**-69.30**	**-84.24**
z	-4.28	-5.41	-4.58	-5.80
P>|z|	0.000	0.000	0.000	0.000
Non-new chemical entities	**-0.10**	**-0.02**	**-0.09**	**-0.00**
z	-0.96	-0.22	-0.83	-0.03
P>|z|	0.337	0.824	0.404	0.972
Cumulative lost life years (LLY)	**0.00**	**0.00**	**0.00**	**0.00**
z	11.38	11.52	11.24	11.47
P>|z|	0.000	0.000	0.000	0.000
Cumulative LLY other diagnoses	**0.00**	**0.00**	**0.00**	**0.00**
z	5.99	6.05	6.44	6.45
P>|z|	0.000	0.000	0.000	0.000
Rehabilitation (Reha1)	**2.06**	**14.89**	**2.72**	**18.60**
z	35.57	56.78	63.60	94.23
P>|z|	0.000	0.000	0.000	0.000
Rehabilitation for other diagnoses (Reha2)	**3.00**	**17.95**		
z	16.44	20.91		
P>|z|	0.000	0.000		
Reha1/Reha2			**-4.12**	**-4.57**
z			-3.77	-4.46
P>|z|			0.000	0.000
No reha dummy			**203.82**	**279.90**
z			2.32	3.06
P>|z|			0.020	0.002
Post reform 2001	**-104.85**	**-96.45**	**-97.51**	**-94.66**
z	-3.33	-3.23	-3.05	-3.11
P>|z|	0.001	0.001	0.002	0.002
Blue-collar workers	**1653.94**	**1658.99**	**1692.15**	**1711.63**
z	6.68	6.91	6.83	7.14
P>|z|	0.000	0.000	0.000	0.000
Miners	**-1413.98**	**-1310.03**	**-1333.01**	**-1236.74**
z	-5.71	-5.44	-5.37	-5.13
P>|z|	0.000	0.000	0.000	0.000
East German residence	**-1645.61**	**-1564.32**	**-1583.89**	**-1511.39**
z	-8.14	-7.95	-7.82	-7.69
P>|z|	0.000	0.000	0.000	0.000
Age group	**-34.81**	**-39.08**	**-33.02**	**-37.71**
z	-2.78	-3.21	-2.63	-3.10
P>|z|	0.005	0.001	0.008	0.002
Cons	**2170.65**	**2320.98**	**1722.64**	**1883.54**
z	2.54	2.81	2.00	2.27
P>|z|	0.011	0.005	0.045	0.023
sigma_u	5406.44	5206.21	5353.54	5161.56
sigma_e	1871.49	1749.11	1860.41	1750.28
rho	0.89	0.90	0.89	0.90
No. of observations	27350	26492	27350	26492
No. of groups	2736	2688	2736	2688
Obs. per group: min	8	1	8	1
average	10.0	9.9	10	9.9
max	10	10	10	10
Wald chi^2	5774.88 (11)	11129.59 (11)	5480.27 (12)	10569.78 (12)
Prob > chi^2	0.000	0.000	0.000	0.000
R^2: within	0.1645	0.2913	0.1542	0.2780
between	0.2845	0.3606	0.2945	0.3663
overall	0.2751	0.3546	0.2837	0.3590

† **Rehabilitation by age groups** is used instead of **Rehabilitation (Reha1)** and **Rehabilitation for other diagnoses by age groups** is used instead of **Rehabilitation for other diagnoses (Reha 2).**

Table 11: Results of Arellano-Bond Dynamic Panel-data Regressions for Lost Working Years in Total Germany from 1995 to 2004

Coefficients	1	2†	3#	4†#
Results	*One-step*	*One-step*	*One-step*	*One-step*
Lagged lost working years	**0.84**	**0.79**	**0.67**	**0.66**
z	172.33	139.34	43.60	42.28
P>|z|	0.000	0.000	0.000	0.000
Lagged lost working years (-2)			**-0.04**	**-0.04**
P>|z|			0.001	0.000
Lagged lost working years (-3)			**-0.09**	**-0.09**
P>|z|			0.000	0.000
Lagged lost working years (-4)			**0.12**	**0.12**
P>|z|			0.000	0.000
Lagged lost working years (-5)			**0.04**	**0.04**
P>|z|			0.000	0.000
New chemical entities	**-97.42**	**-101.14**	**-168.81**	**-173.79**
z	-8.08	-8.26	-7.65	-7.73
P>|z|	0.000	0.000	0.000	0.000
Non-new chemical entities	**0.45**	**0.46**	**0.75**	**0.79**
z	5.66	5.82	6.90	7.16
P>|z|	0.000	0.000	0.000	0.000
Cumulative lost life years (LLY)	**0.00**	**0.00**	**0.00**	**0.00**
z	8.47	8.43	4.42	4.54
P>|z|	0.000	0.000	0.000	0.000
Cumulative LLY other diagnoses	**0.00**	**0.00**	**0.00**	**0.00**
z	13.11	12.97	6.28	6.28
P>|z|	0.000	0.000	0.000	0.000
Rehabilitation for same diagnosis	**0.31**	**3.08**	**0.05**	**1.54**
(Reha1) z	7.74	14.19	0.98	6.01
P>|z|	0.000	0.000	0.325	0.000
Rehabilitation for other diagnoses	**1.46**	**10.73**	**-27.45**	**-27.39**
(Reha2) z	13.02	139.34	-0.48	-0.43
P>|z|	0.000	0.000	0.632	0.667
Reha1/Reha2			**3.29**	**3.07**
z			3.67	3.40
P>|z|			0.000	0.001
Post reform 2001	**-99.62**	**-98.09**		
z	-4.51	-4.40		
P>|z|	0.000	0.000		
Cons	**90.07**	**87.72**	**28.92**	**30.45**
z	12.49	12.11	4.11	4.18
P>|z|	0.000	0.000	0.000	0.000
No. of observations	21874	21070	10928	10574
No. of groups	2736	2670	2734	2668
Obs. per group: min	5	2	2	1
average	8.0	7.9	4.0	4.0
max	8	8	4	4
Wald chi^2	62970.97 (8)	64645.72 (8)	9378.03 (12)	9356.88 (12)
Sargan test of over-identifying restrictions				
chi^2	4055.38 (35)	4053.76 (35)	2752.08	2810.95 (21)
Prob > chi^2	0.000	0.000	0.000	0.000
Arellano-Bond test that average autocovariance in residuals of order 1 is 0: H0: no autocorrelation				
z	-45.54	-42.81	-23.27	-22-58
Pr > z	0.000	0.000	0.000	0.000
Arellano-Bond test that average autocovariance in residuals of order 2 is 0: H0: no autocorrelation				
z	-7.25	-6.49	-1.33	-0.62
Pr > z	0.000	0.090	0.183	0.538

† **Rehabilitation by age groups** is used instead of **Rehabilitation (Reha1)** and **Rehabilitation for other diagnoses by age groups** is used instead of **Rehabilitation for other diagnoses (Reha 2).**

No reha dummy is used instead of **Rehabilitation for other diagnoses (Reha 2).**

Table 12: Results of the Arellano-Bond Dynamic Panel-data Estimations for Lost Life Years in Total Germany from 1995 to 2004

Coefficients	1	2
Results	*One-step*	*Two-step*
Lagged lost life years	**0.89**	**0.89**
z	81.14	750.14
P>|z|	0.000	0.000
New chemical entities	**-276.38**	**-480.48**
z	-0.44	-5.72
P>|z|	0.660	0.000
Non-new chemical entities	**-12.61**	**-7.04**
z	-3.28	-7.29
P>|z|	0.001	0.000
Lost working years	**0.03**	**-0.04**
z	0.36	-1.34
P>|z|	0.716	0.180
Post reform 2001	**2661.42**	**1198.90**
z	2.30	5.87
P>|z|	0.021	0.000
Cons	**168.45**	**117.07**
z	0.58	2.21
P>|z|	0.560	0.027
No. of observations	1824	1824
No. of groups	228	228
Obs. per group: min	8	8
average	8.0	8.0
max	8	8
Wald chi^2 (5)	7311.05	933516.94
Sargan test of over-identifying restrictions		
chi^2 (35)	1353.67	96.86
Prob > chi^2	0.000	0.000
Arellano-Bond test that average autocovariance in residuals of order 1 is 0:		
H0: no autocorrelation		
z	-13.39	-1.74
Pr > z	0.000	0.08
Arellano-Bond test that average autocovariance in residuals of order 2 is 0:		
H0: no autocorrelation		
z	-2.44	-0.84
Pr > z	0.02	0.40

Table 13: Non-parametric Tests for Differences in Retirement Hazard Rates across Age Groups, Cohorts and for Changes in Hazard Rates over Time

Panel A: ***Test whether the changes in agegroup-specific hazard rates are significantly different from zero***[a]

Changes	from 88–92 to 93–97		from 93–97 to 98–02	
	Male	Female	Male	Female
Wilcoxon signed-rank test (Sign test)				
40–44	0.5327 (0.2379)	0.1842 (0.3593)	0.0004 (0.0013)	0.0002 (0.0007)
45–49	0.2683 (0.4807)	0.5197 (0.6476)	0.0003 (0.0001)	0.0001 (0.0000)
50–54	0.0206 (0.0075)	0.0329 (0.0192)	0.0031 (0.0075)	0.0001 (0.0000)
55–59	0.0014 (0.0075)	0.0003 (0.0001)	0.0001 (0.0000)	0.0002 (0.0001)
60–64	0.0012 (0.0013)	0.0001 (0.0000)	0.0001 (0.0000)	0.0001 (0.0000)

Test for differences in changes of agegroup-specific hazard rates between 1988-1992 to 1993-1997 and 1993-1997 to 1998-2002[a]

	Male	Female
Wilcoxon signed-rank test (Sign test)		
40–44	0.1212 (0.2379)	0.0002 (0.0001)
45–49	0.0133 (0.0963)	0.0001 (0.0000)
50–54	0.8563 (0.8145)	0.0641 (0.3593)
55–59	0.5869 (0.4807)	0.4688 (0.3593)
60–64	0.2351 (0.8145)	0.0002 (0.0007)

Panel B: ***Test whether the changes in age group-specific hazard rates are unequal across age groups***[a]

Changes	from 88–92 to 93–97		from 93–97 to 98–02	
	Male	Female	Male	Female
Wilcoxon signed-rank test (Sign test)				
40–44 vs. 45–49	0.0008 (0.0075)	0.0641 (0.1671)	0.0094 (0.0075)	0.0586 (0.0636)
40–44 vs. 50–54	0.0009 (0.0013)	0.0025 (0.0007)	0.0948 (0.2379)	0.0329 (0.0192)
40–44 vs. 55–59	0.0002 (0.0001)	0.0001 (0.0000)	0.0002 (0.0001)	0.0002 (0.0001)
40–44 vs. 60–64	0.0002 (0.0001)	0.0001 (0.0000)	0.0001 (0.0000)	0.0002 (0.0001)
45–49 vs. 50–54	0.0024 (0.0013)	0.0011 (0.0007)	0.1775 (0.4807)	0.0486 (0.1671)
45–49 vs. 55–59	0.0002 (0.0001)	0.0001 (0.0000)	0.0001 (0.0000)	0.0002 (0.0001)
45–49 vs. 60–64	0.0004 (0.0001)	0.0001 (0.0000)	0.0001 (0.0000)	0.0001 (0.0000)
50–54 vs. 55–59	0.0003 (0.0001)	0.0001 (0.0000)	0.0001 (0.0000)	0.0002 (0.0001)
50–54 vs. 60–64	0.0005 (0.0001)	0.0001 (0.0000)	0.0001 (0.0000)	0.0001 (0.0000)
55–59 vs. 60–64	0.8248 (0.4807)	0.0002 (0.0001)	0.0001 (0.0000)	0.0364 (0.0636)

Panel C: ***Test whether the changes in cohort hazard rates are different from zero***[ba]

Changes	from 88–92 to 93–97		from 93–97 to 98–02	
	Male	Female	Male	Female
Wilcoxon signed-rank test (Sign test)				
Cohort 1	0.0001 (0.0000)	0.0001 (0.0000)	0.0002 (0.0001)	0.0003 (0.0007)
Cohort 2	0.0001 (0.0000)	0.0001 (0.0000)	0.0001 (0.0000)	0.0001 (0.0000)
Cohort 3	0.0001 (0.0000)	0.0001 (0.0000)	0.9839 (0.8145)	0.0534 (0.1671)

Test whether the changes in cohort hazard rates are unequal across cohorts[ba]

Changes	from 88–92 to 93–97		from 93–97 to 98–02	
	Male	Female	Male	Female
Wilcoxon signed-rank test (Sign test)				
Cohort 1 vs. cohort 2	0.0001 (0.0000)	0.0002 (0.0007)	0.0001 (0.0000)	0.0001 (0.0000)
Cohort 1 vs. cohort 3	0.0001 (0.0000)	0.0001 (0.0000)	0.2683 (0.0963)	0.0079 (0.0192)
Cohort 2 vs. cohort 3	0.0001 (0.0000)	0.0001 (0.0000)	0.0000 (0.0000)	0.0002 (0.0001)

a Only p-values are reported.
b Changes in cohort hazard rates minus within-agegroup changes in hazard rates.

Table 14: Regression-based Tests for the Competing Risks Hypothesis in Health-related Retirement Hazards at the Disease Level

The dependent variable comprises the retirement hazard rates in each age group between 45 and 64 for all diseases in the 1993–1997 and 1998 –2002 time windows, except for the major diseases whose hazards rates are included as explanatories.	1	2	3	4
Original hazard	**0.405**	**0.683**	**0.686**	**0.687**
t	26.81	35.02	34.25	34.52
P>|t|	0.000	0.000	0.000	0.000
Dummy for male	**-0.000**	**-0.000**	**-0.000**	**-0.000**
t	-0.55	-1.47	-1.00	-1.18
P>|t|	0.586	0.143	0.318	0.238
Time window	**-0.000**	**-0.000**	**-0.000**	**-0.000**
t	-4.66	-2.61	-1.19	-1.34
P>|t|	0.000	0.010	0.235	0.181
Musculoskeletal hazard	**-0.049**	**-0.089**	**-0.094**	**-0.027**
t	-4.11	-7.03	-5.33	-0.86
P>|t|	0.000	0.000	0.000	0.390
Circulatory hazard	**0.060**	**0.092**	**0.084**	**0.077**
t	4.49	5.20	4.26	3.92
P>|t|	0.000-	0.000	0.000	0.000
Cancer hazard	**-0.027**			
t	-0.56			
P>|t|	0.575			
Psychiatric hazard	**0.030**			
t	1.06			
P>|t|	0.289			
Cohort 2			**-0.000**	
t			-0.55	
P>|t|			0.586	
Cohort 3			**0.000**	
t			0.16	
P>|t|			0.874	
Cohort 4			**0.000**	
t			0.71	
P>|t|			0.477	
Cohort 5			**-0.000**	
t			-0.13	
P>|t|			0.899	
Agegroup 45–49				**0.001**
t				2.08
P>|t|				0.039
Agegroup 50–54				**0.001**
t				2.21
P>|t|				0.028
Agegroup 55–59				**0.001**
t				1.96
P>|t|				0.051
Cons	**0.000**	**0.001**	**0.001**	**-0.001**
t	5.86	3.70	2.07	-0.95
P>|t|	0.000	0.000	0.039	0.344
No. of observations	240	272	272	272
F	134.26 (7, 232)	253.84 (5, 266)	140.37 (9, 262)	160.87 (8, 263)
Prob > F	0.000	0.000	0.000	0.000
R^2	0..8020	0.8267	0.8282	0.8303
Adj R^2	0.7960	0.8235	0.8223	0.8252
Root MSE	0.00016	0.00043	0.00043	0.00043

Figures

Figure 1: Relative Private Reward of Continuing to Work in a Pay-as-you-go Pension System when Health Declines

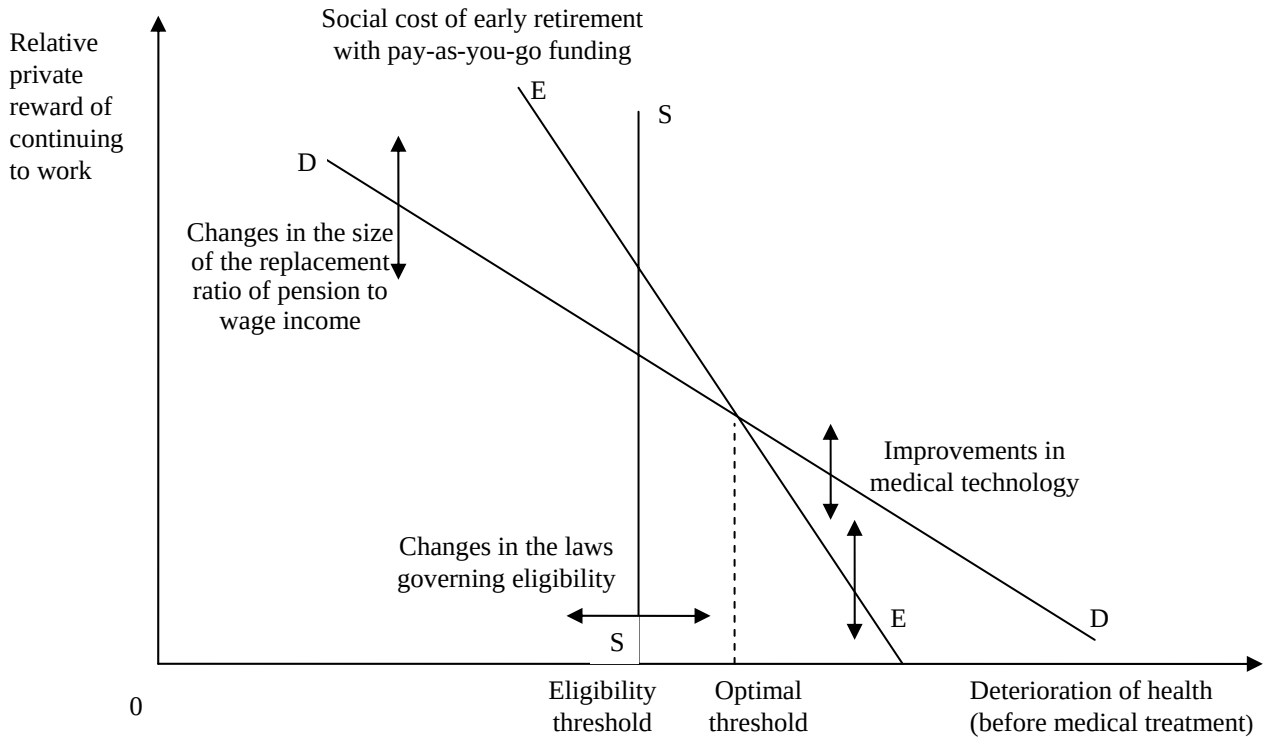


Figure 2: The Early Retirement's Decision

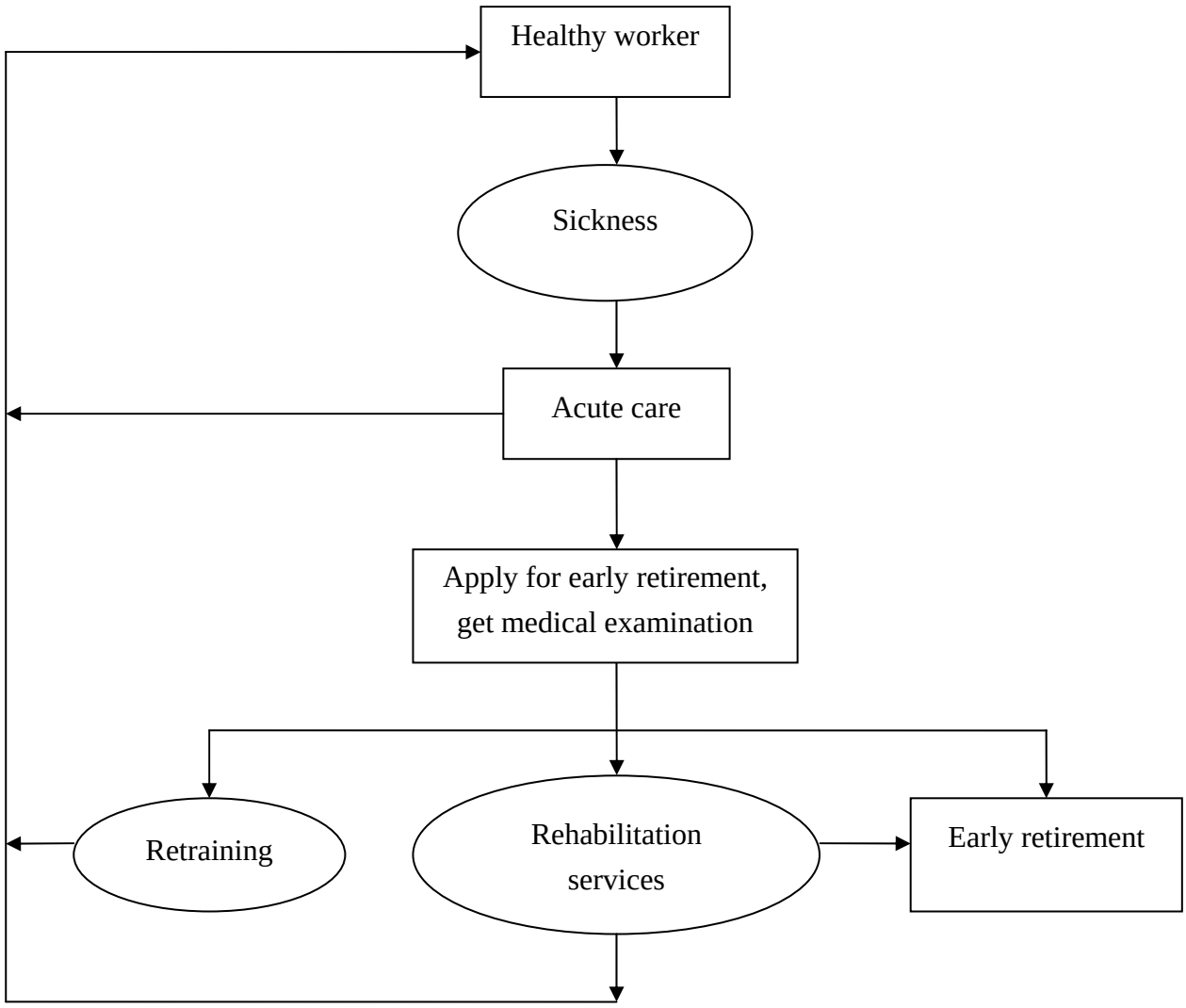
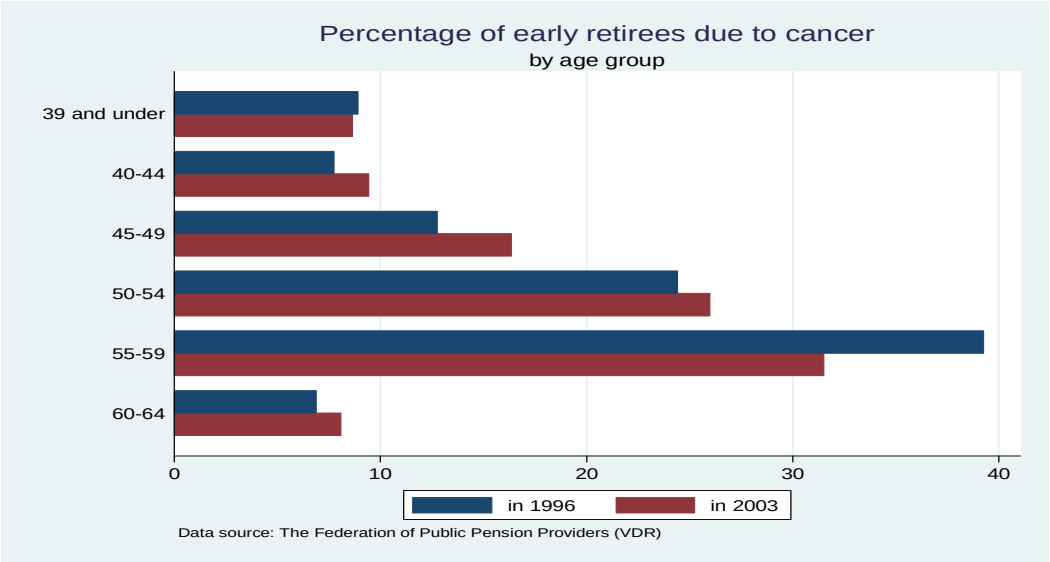
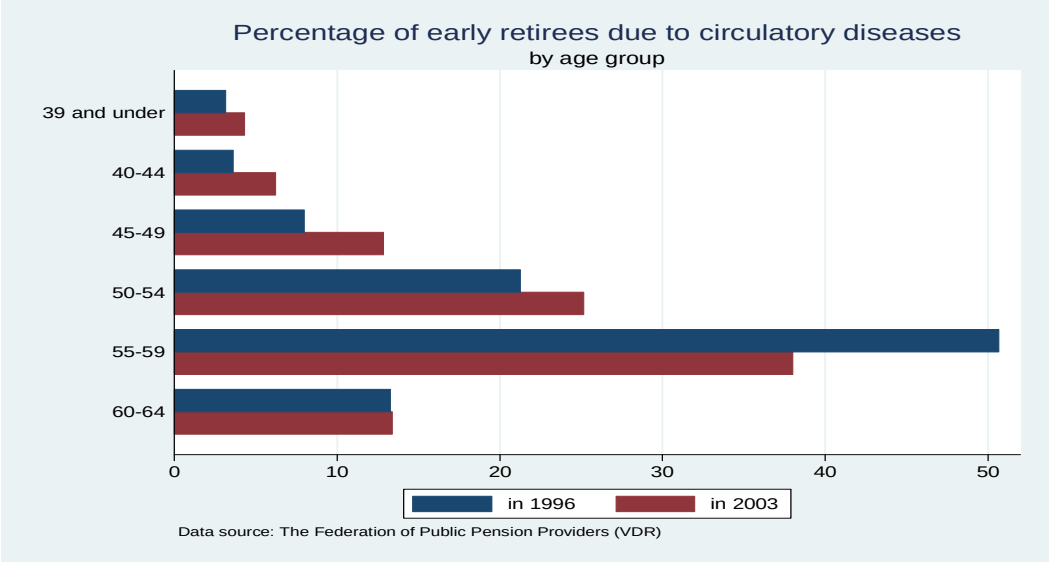


Figure 3: Percentage Distribution of Early Retirees for Selected Diseases across Agegroups in 1996 and 2003





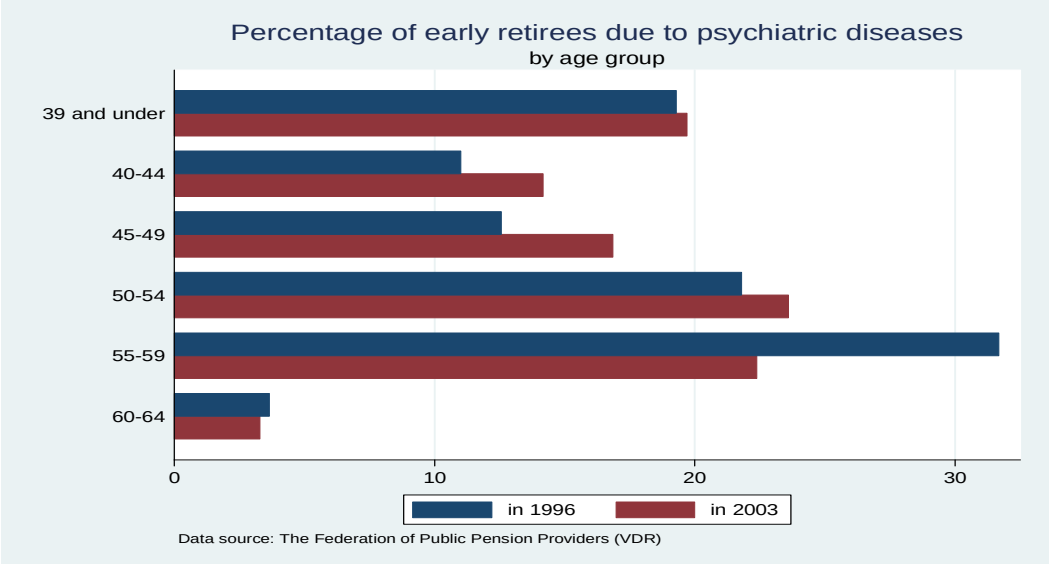
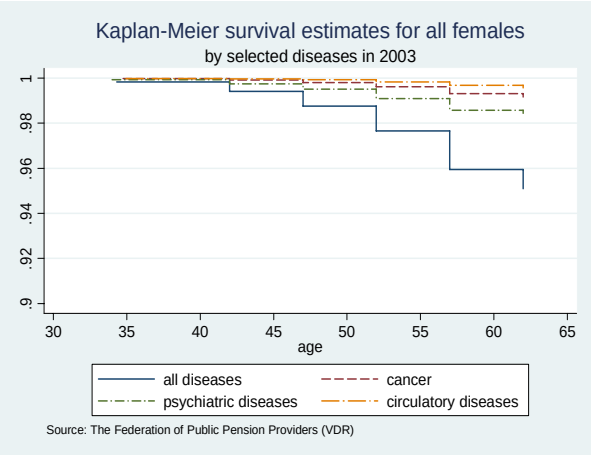
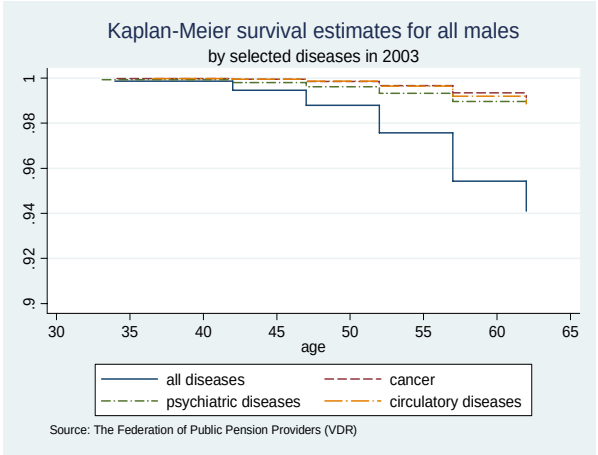
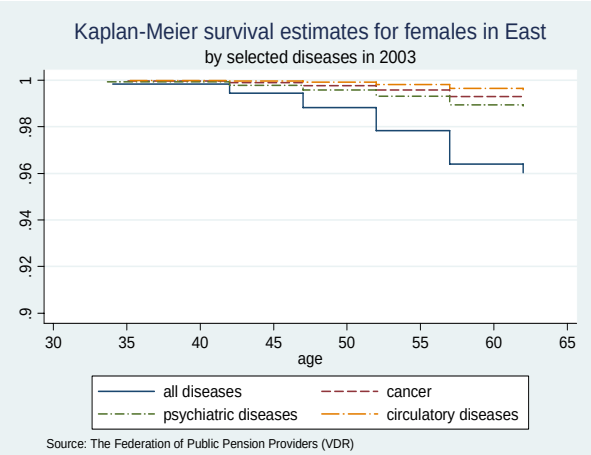
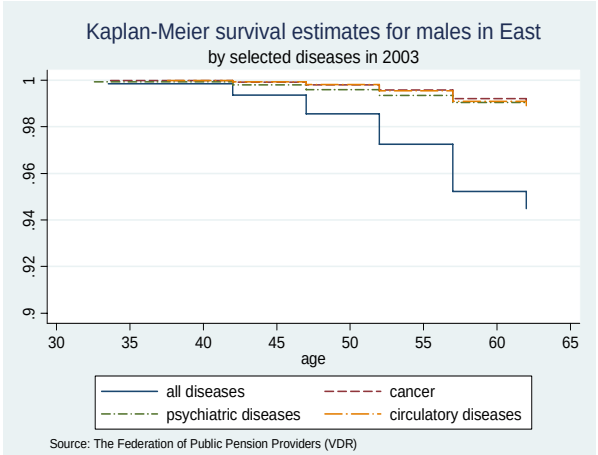


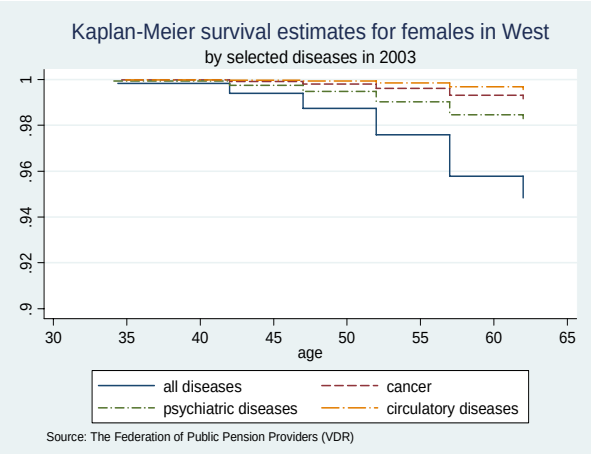
Figure 4: Kaplan-Meier Survival Estimates for Retiring











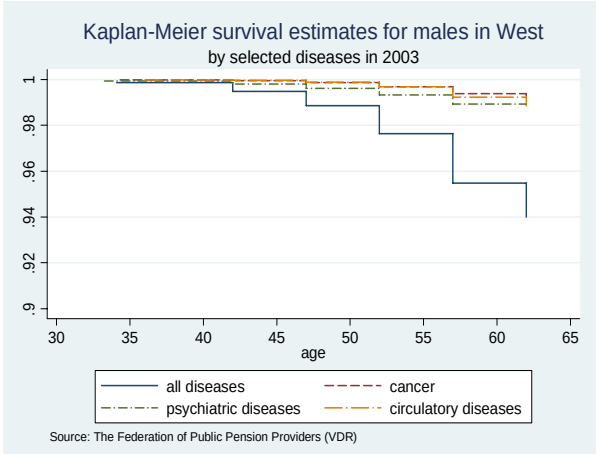
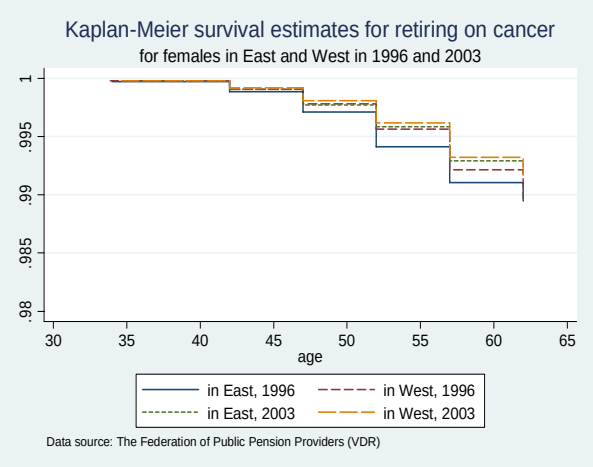
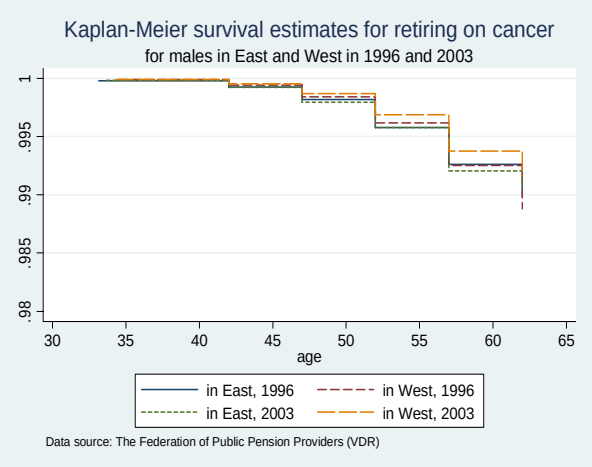
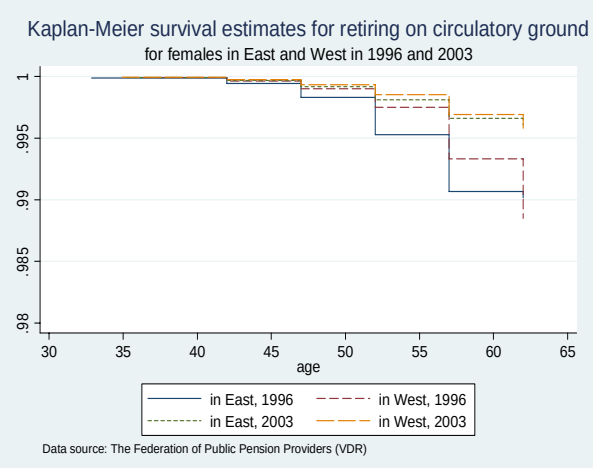
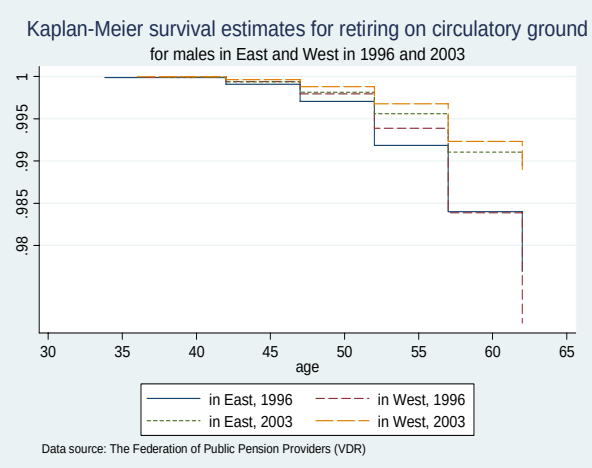


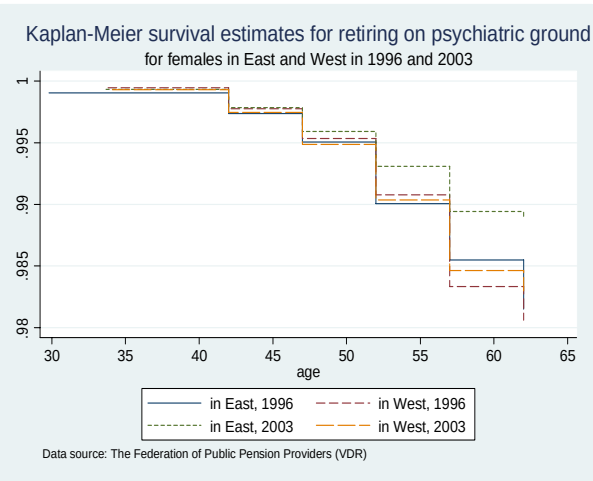
Figure 4 continued











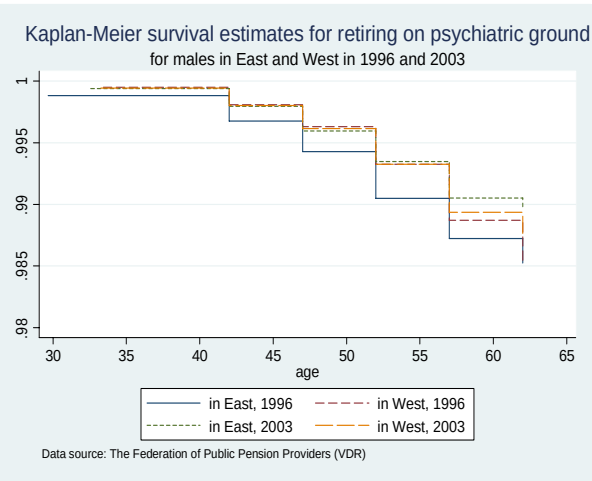
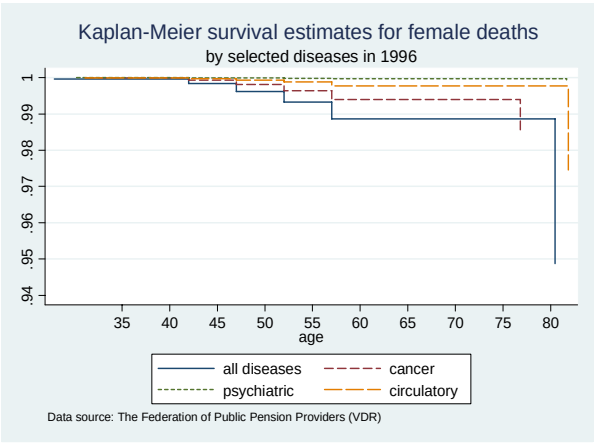
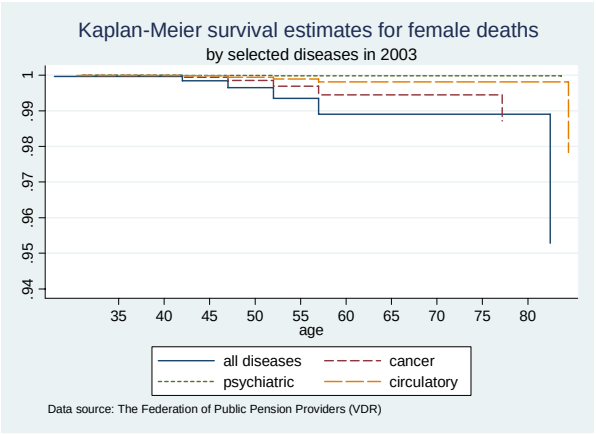
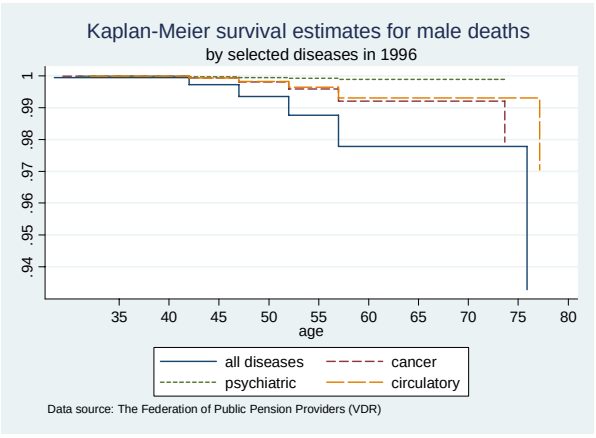
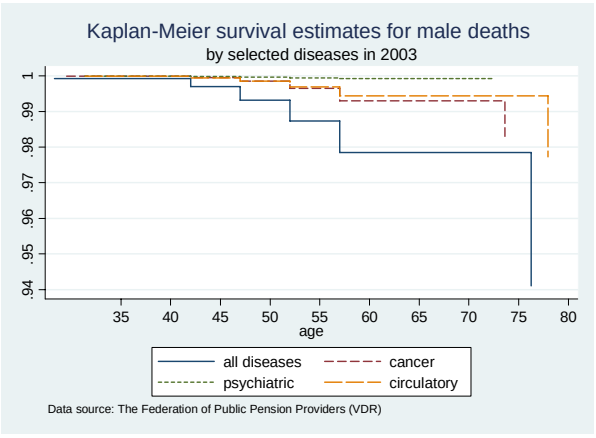


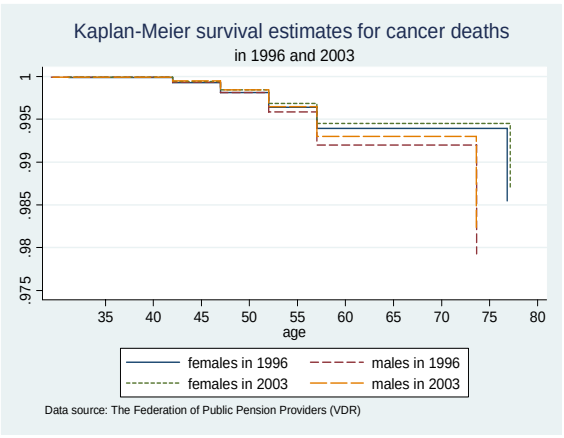
Figure 5: Kaplan-Meier Survival Estimates for Death

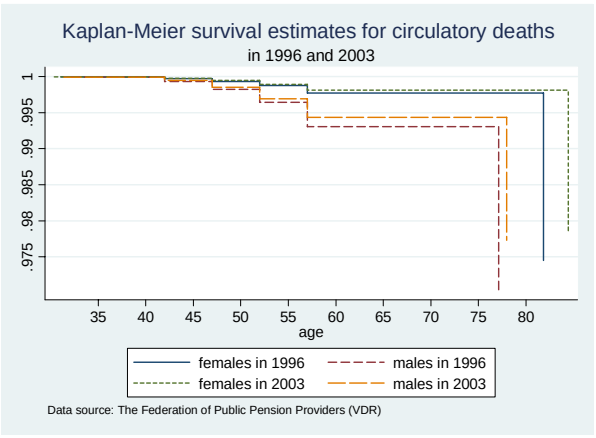












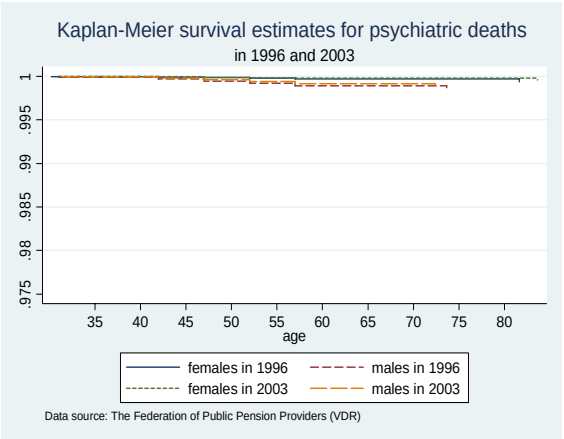


Figure 6a: Distribution of 44 New Chemical Entities Launched between 1995 and 2004, by Therapeutic Class

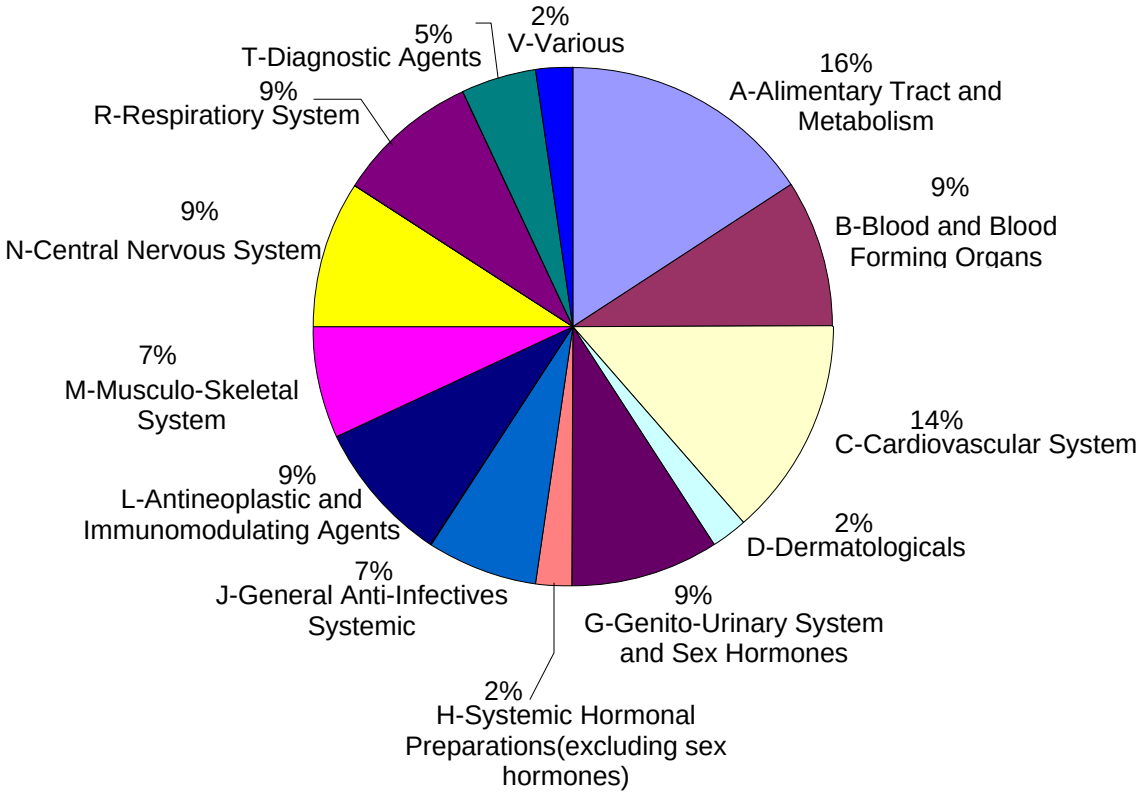
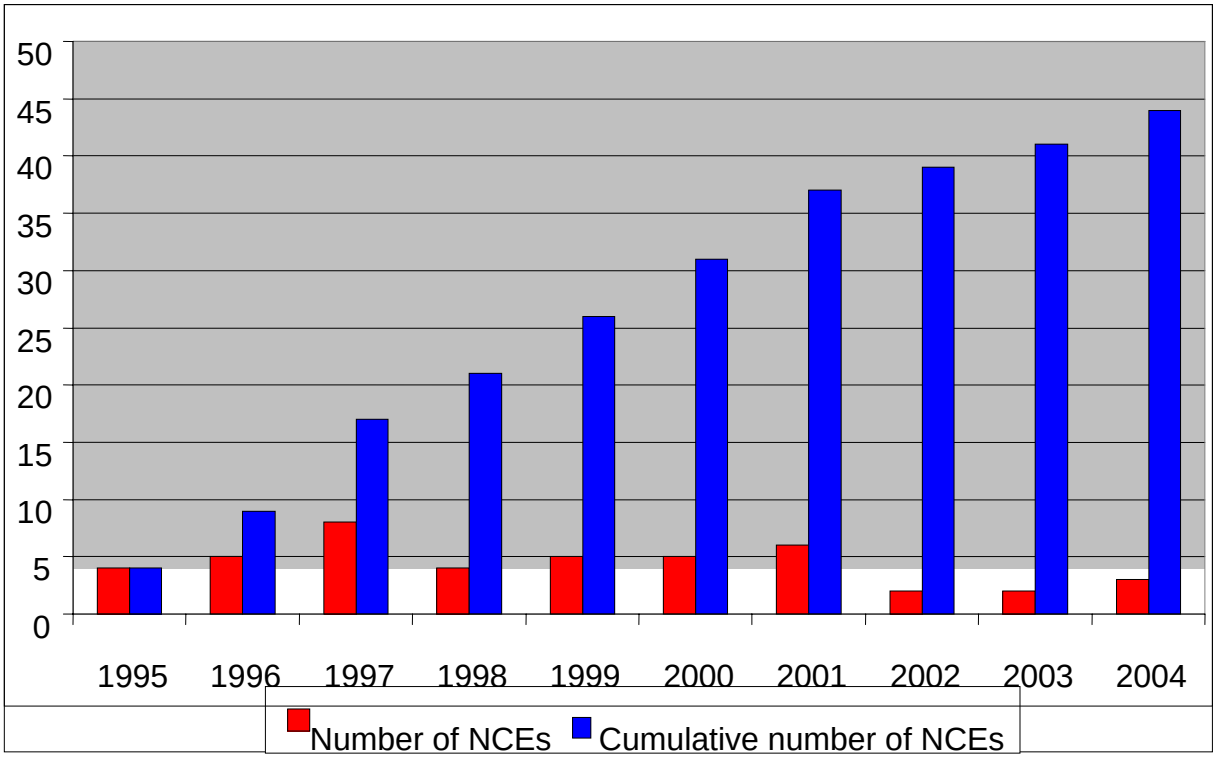


Figure 6b: Number and Cumulative Number of New Chemical Entities Launched, by Year



Source: The IMS Health Drug Launches

Figure 7a: Distribution of 6940 Non-new Chemical Entities Launched between 1995 and 2004, by Therapeutic Class

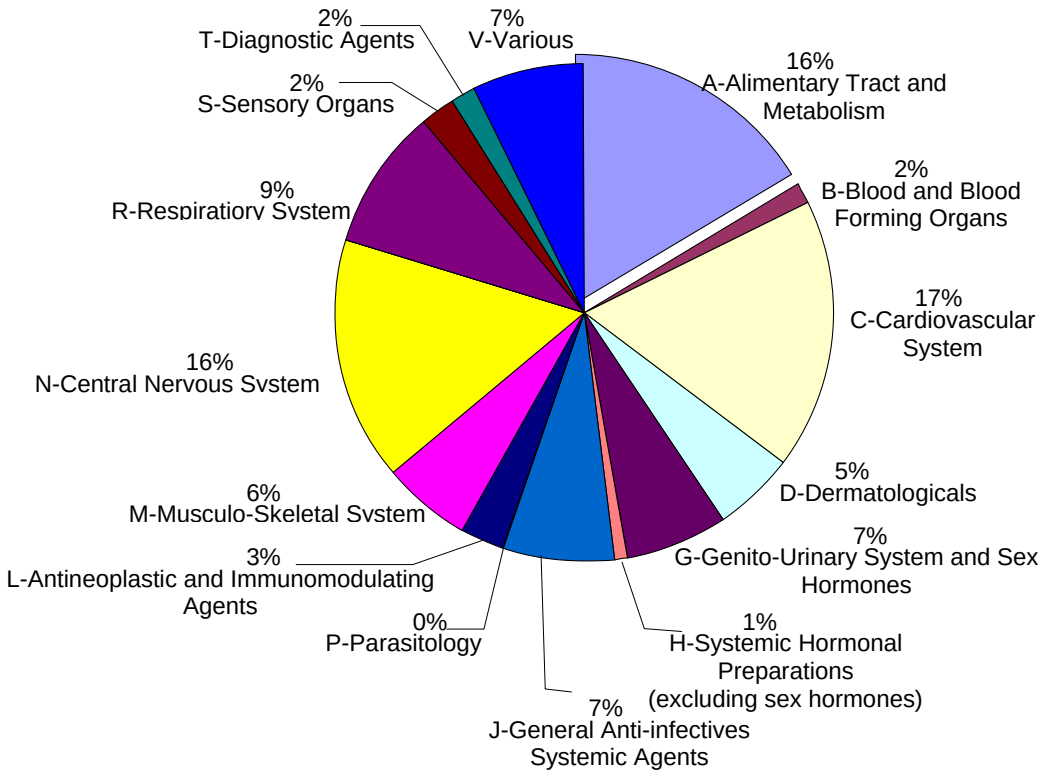
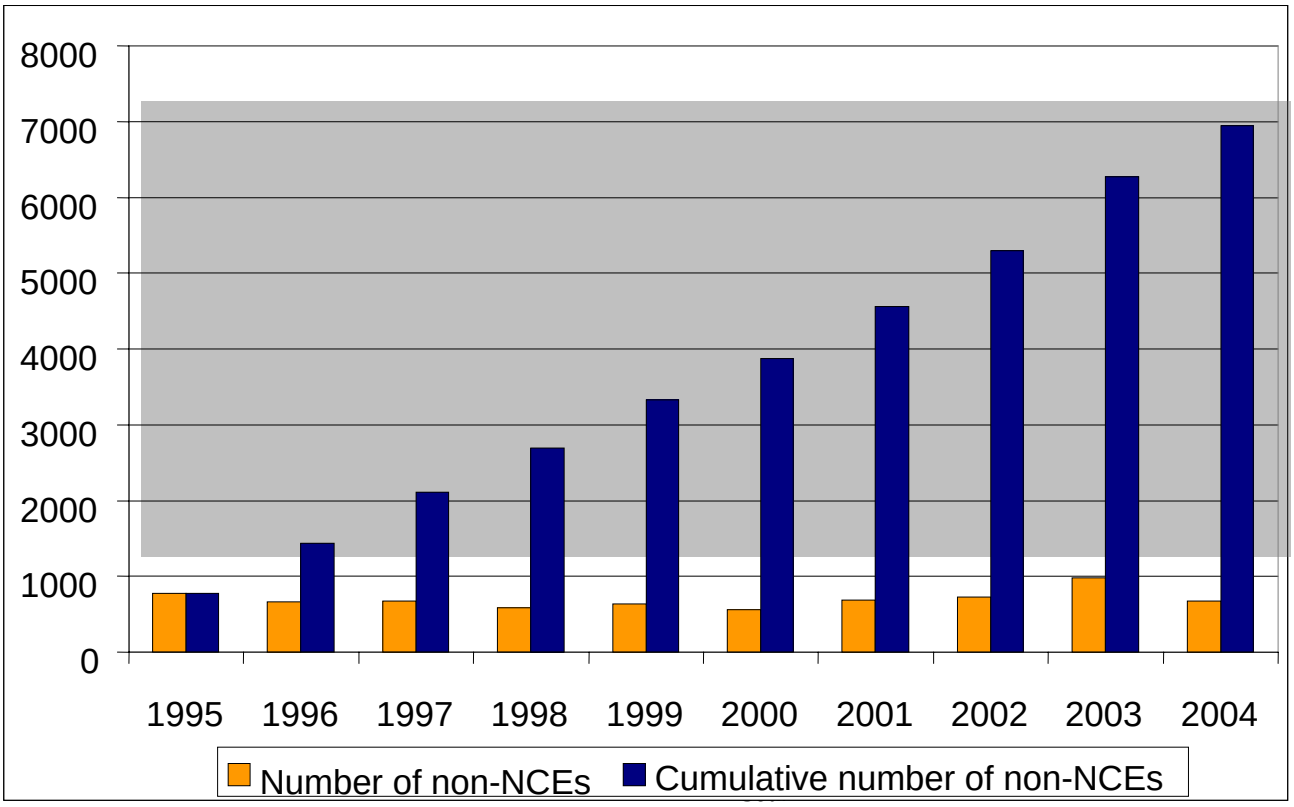
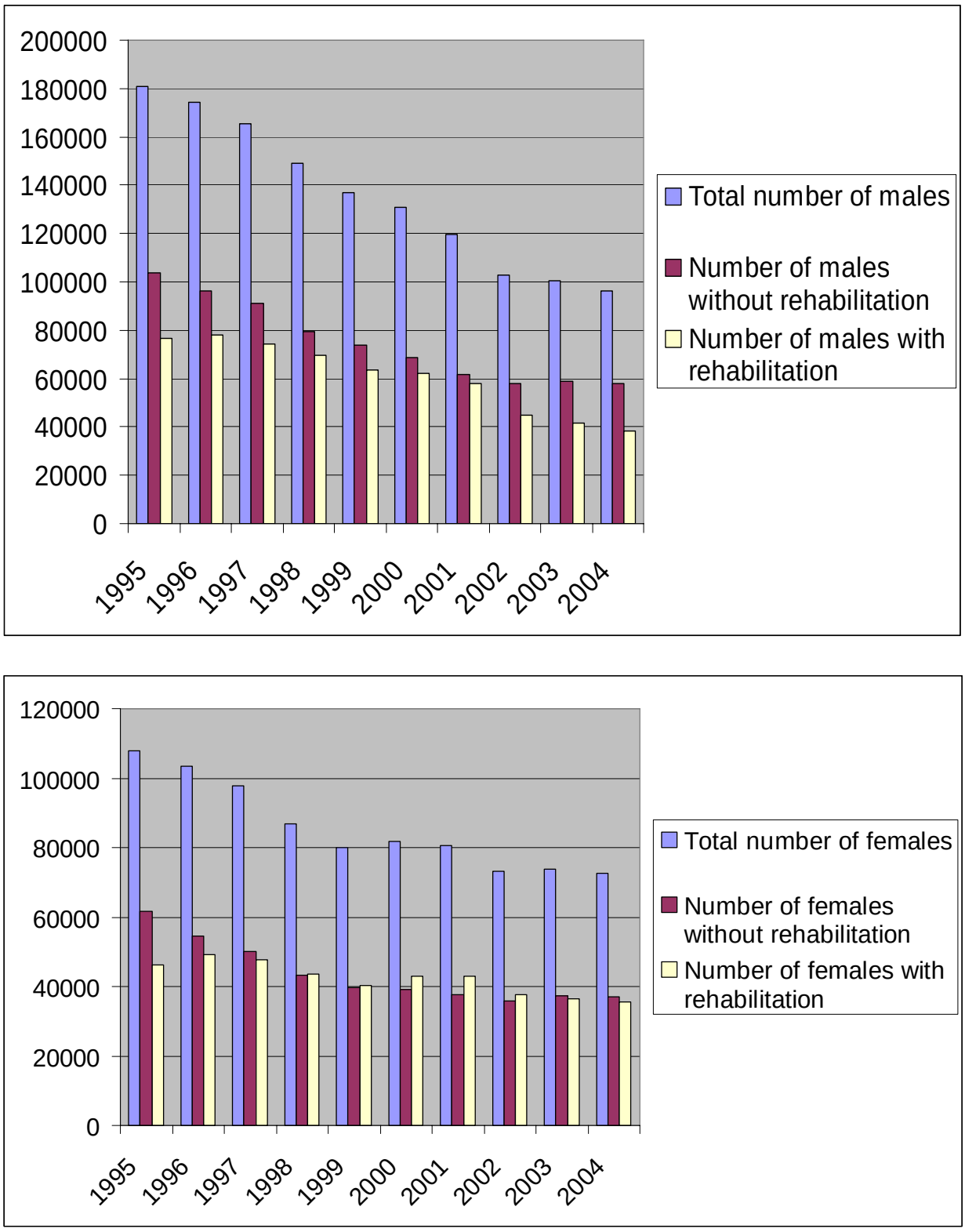


Figure 7b: Number and Cumulative Number of Non-new Chemical Entities Launched, by



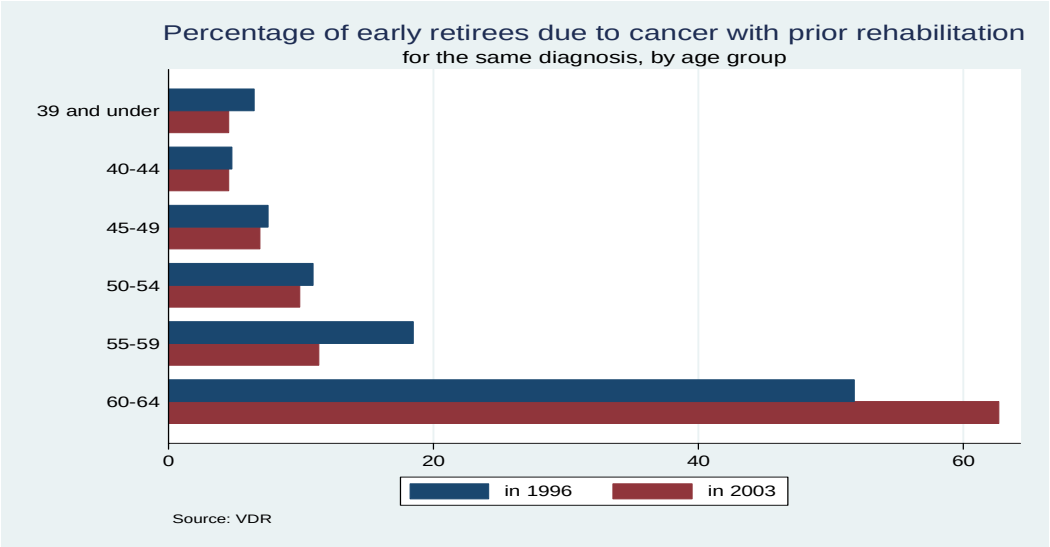
Source: The IMS Health Drug Launches

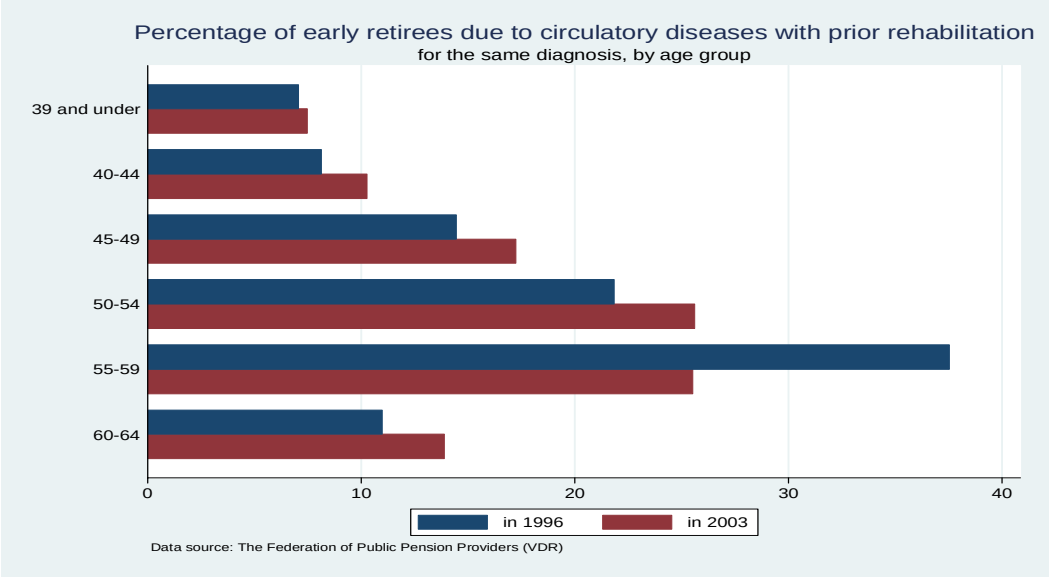
Figure 8: Number of Male Early Retirees (top panel) and Female Early Retirees (lower panel) With and Without Rehabilitation in Public Pension Insurance, by Year



Source: The Federation of Public Pension Providers in Germany (VDR)

Figure 9: Percentage Distribution of Early Retirees with Prior Rehabilitation for Selected Diseases across Agegroups in 1996 and 2003





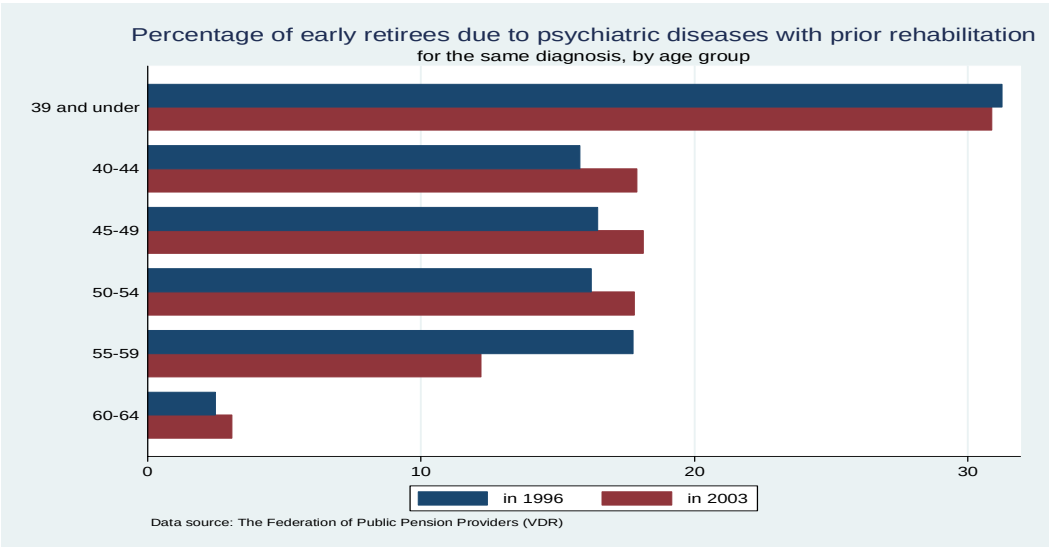


Figure 10: Ratio of Rehabilitation Services to Early Retirees for Selected Diseases in 1996 and 2003, in percent

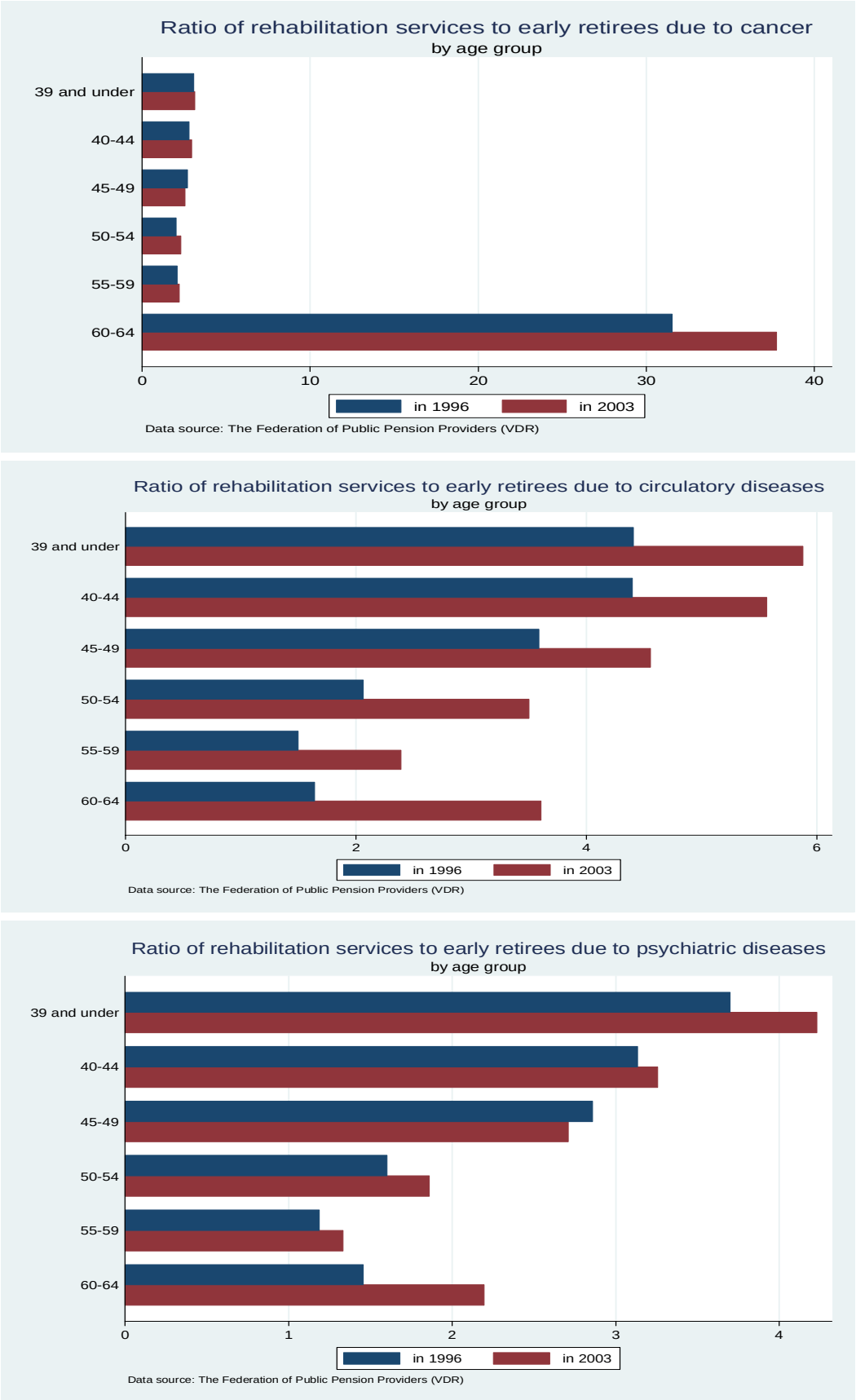
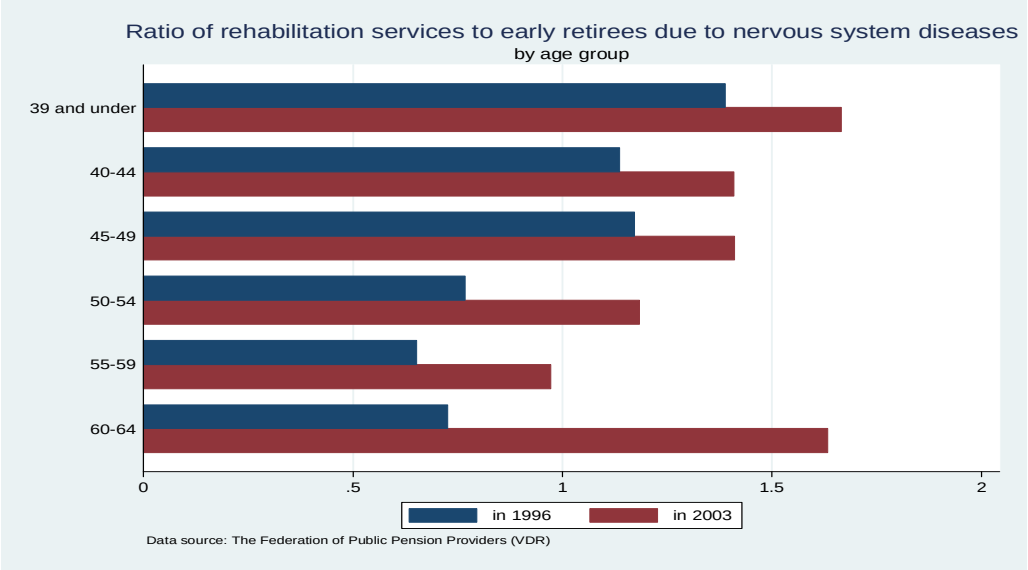
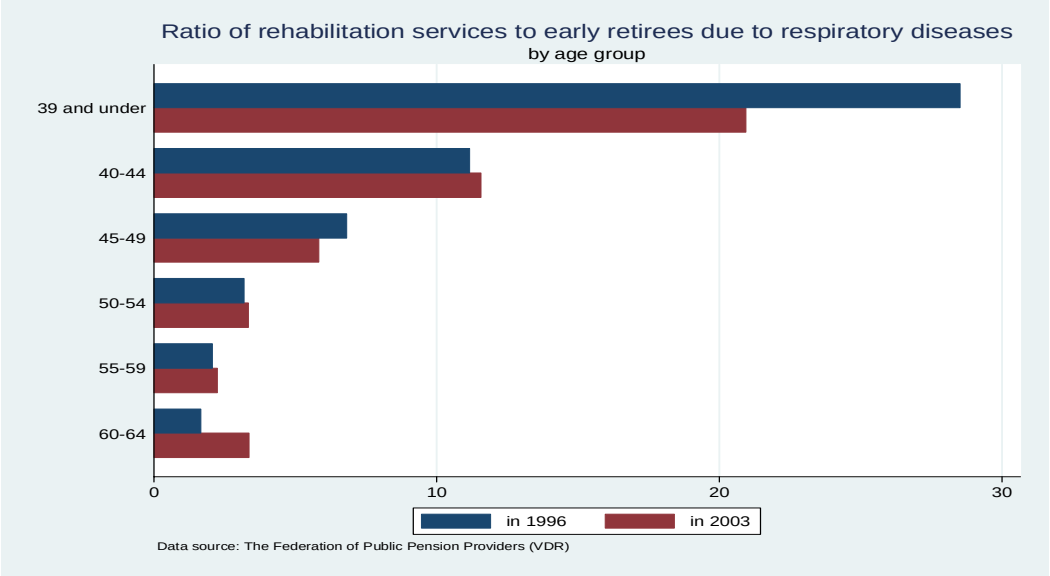


Figure 10 continued





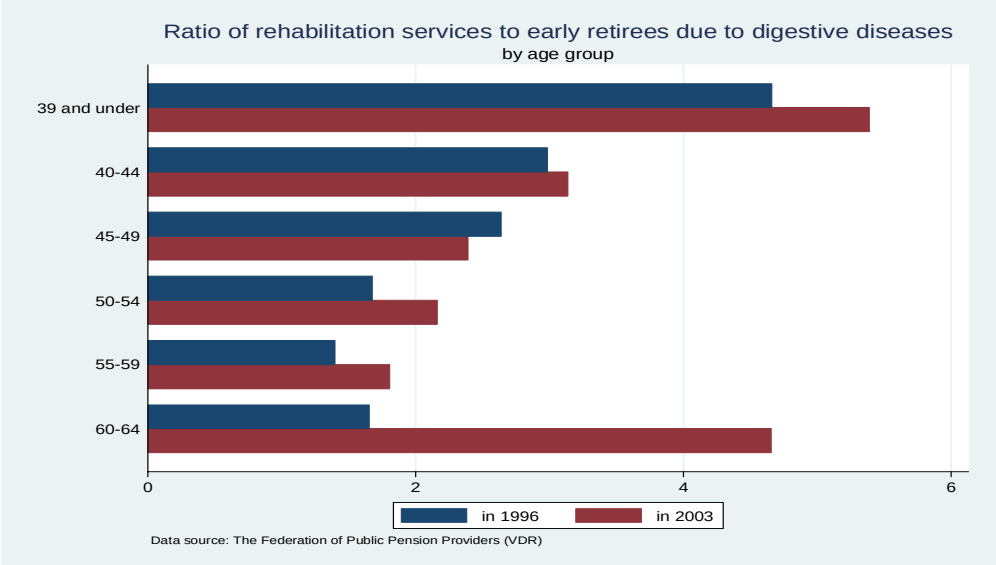


Figure 11: The Decline of Retirement Hazards for Men, by Age and Disease

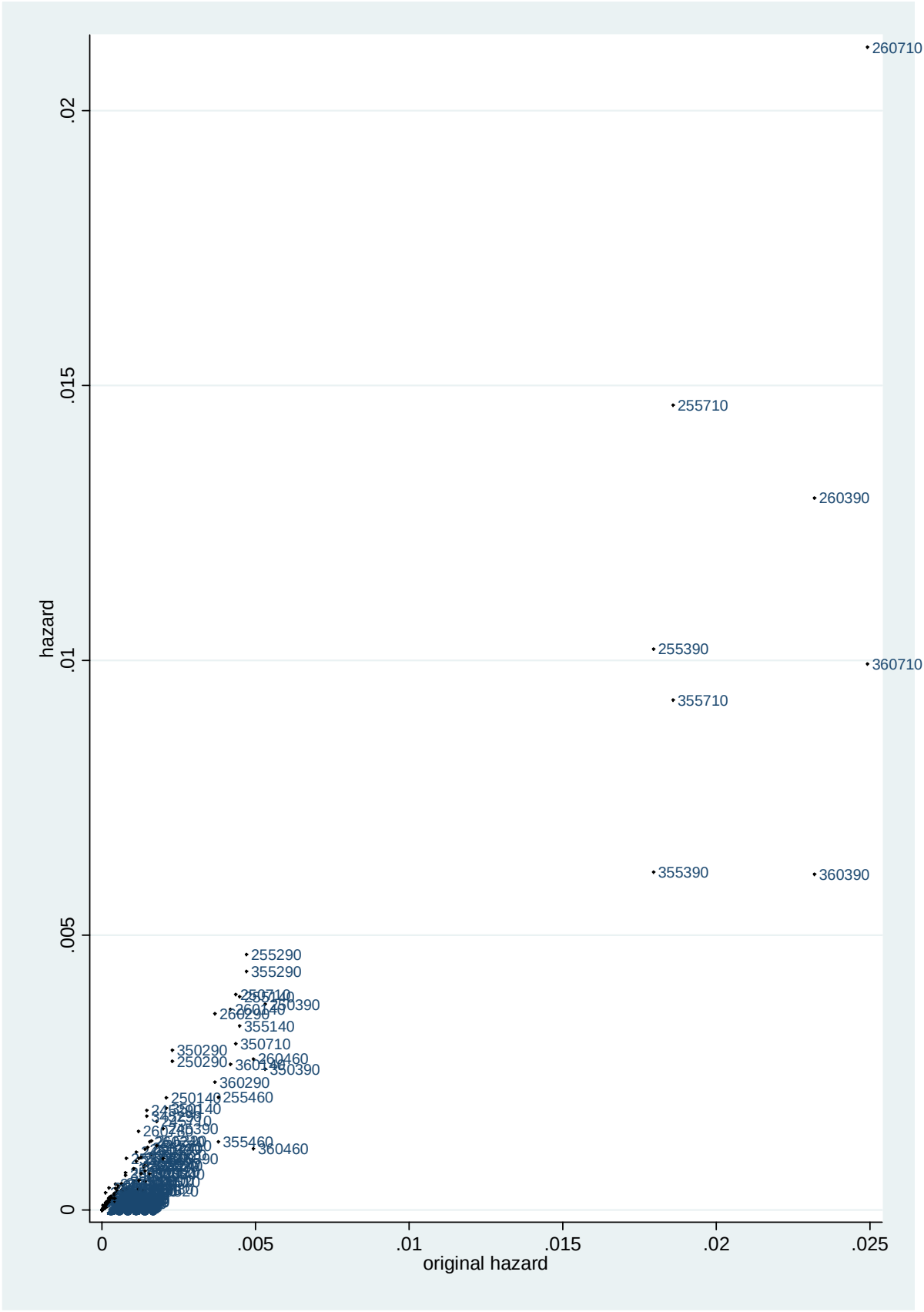


Figure 12: The Decline of Retirement Hazards for Women, by Age and Disease

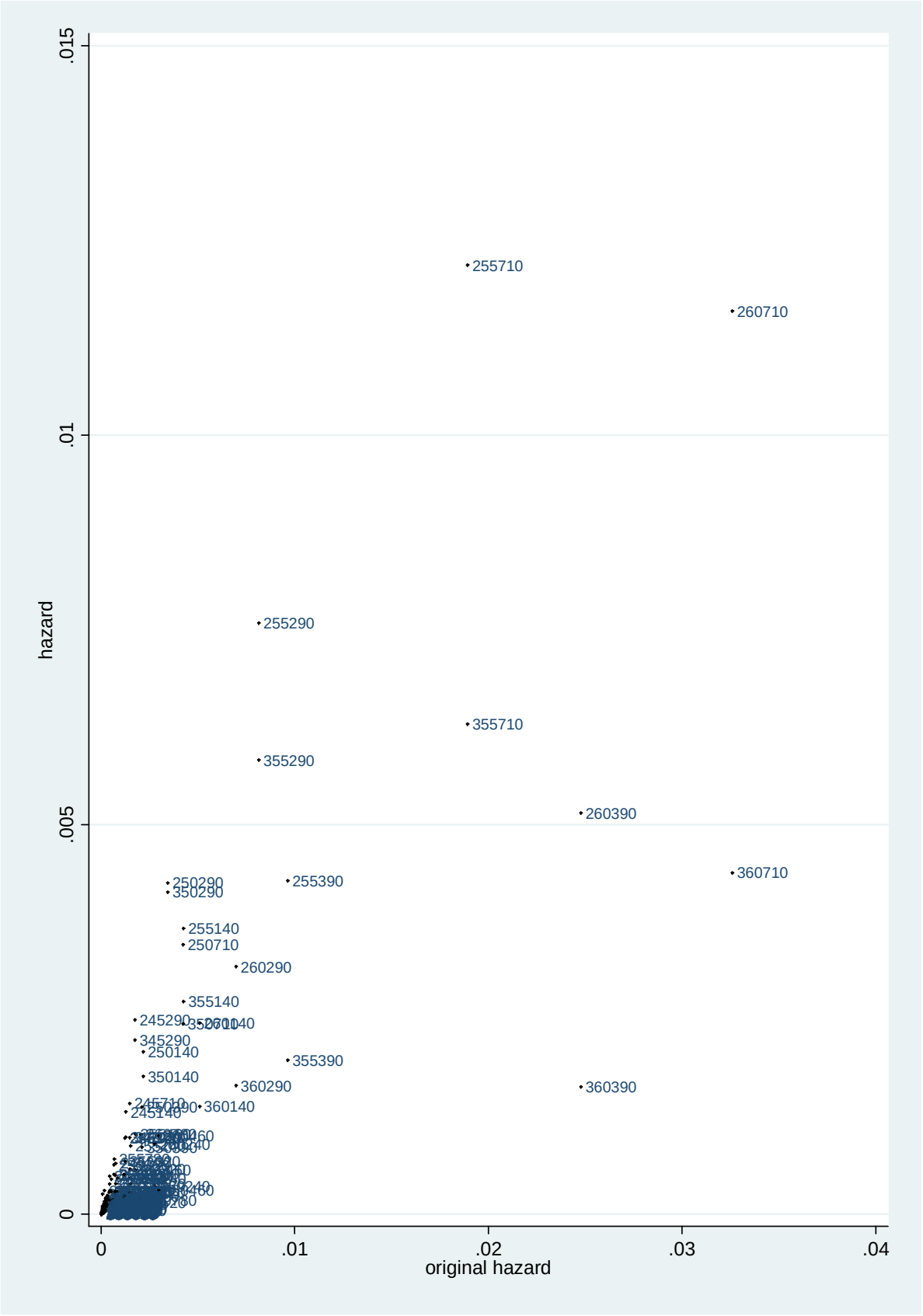


Figure 13: Male Retirement Hazards for Rare Diseases, by Agegroup and Cohort over Time

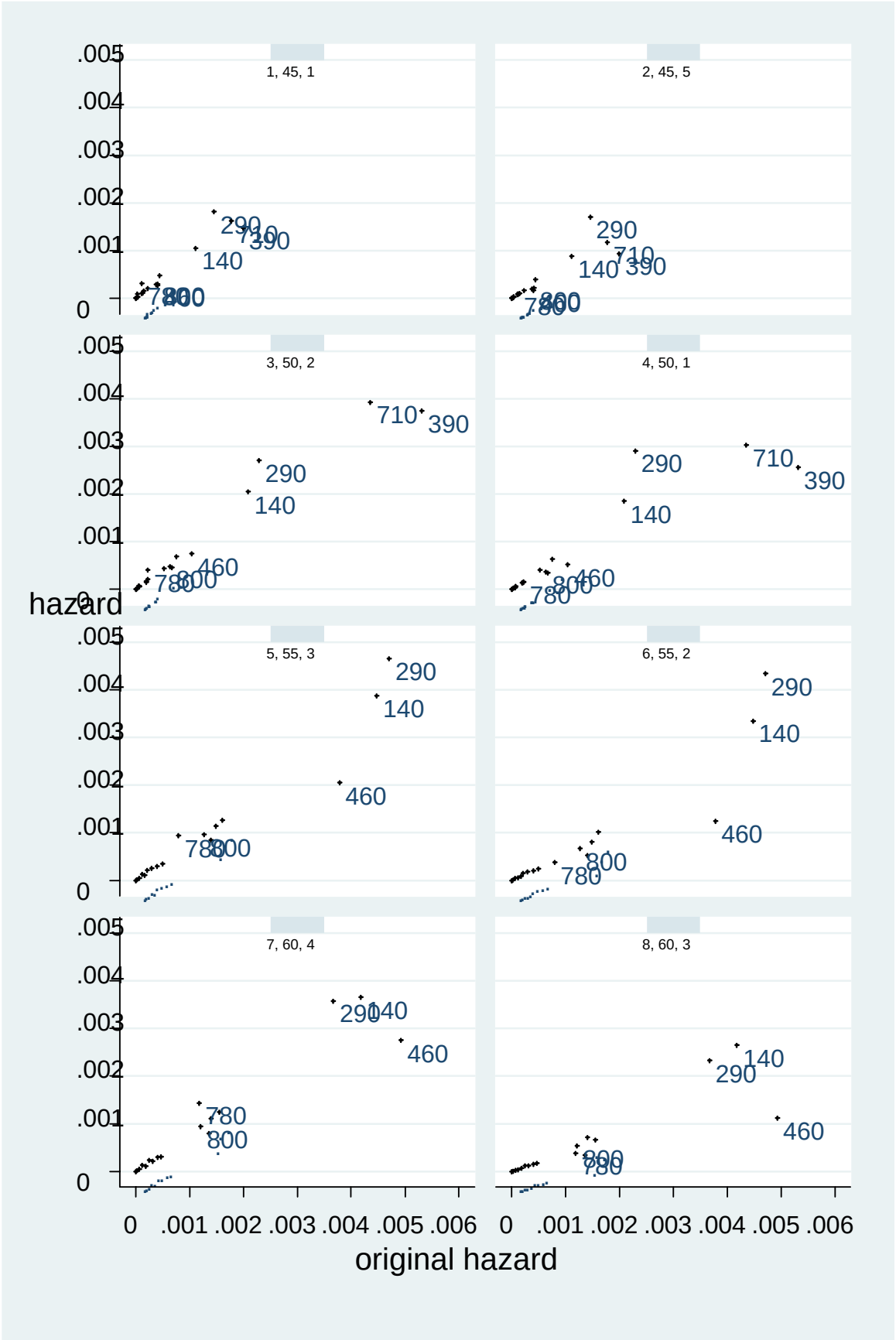


Figure 14: Female Retirement Hazards for Rare Diseases, by Agegroup and Cohort over Time