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How to Estimate the Economic Value of Health Care Intervention: A Methodological Study and an Illustrative Empirical Application on Diabetes

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Abstract

Health plays a vital role in every human's life; and the centre for the maintaining and retaining our health lies in the health care. Thanks to innovations in health care technology and innovations of new drugs the health care is constantly improving, which contributes to improved survival probability and improved quality of life. However, this development in health care comes at a high price, since the development of drugs and technologies is costly which leads to rising prices in health care. This is one explanation to the fact that health care costs take up an increasing part of many countries GDP.

We are interested in how the gains, in terms of improved longevity and quality of life, from the improved health care can be measured and valued. We have chosen to compare two methods in this area, one econometric model developed by Frank R. Lichtenberg, and one model based on the theory of willingness to pay (WTP) developed by Kevin M. Murphy and Robert H. Topel. We then make an illustrative empirical application of one of the methods on the case of increasing longevity in diabetes in Sweden. With the data we found on diabetes longevity we came to the conclusion that Murphy and Topel's model is more applicable to this issue in the scope of our study. It would be interesting for further studies to applicate Lichtenberg's method; however it demands more extensive work and larger amounts of data.

In our empirical application on diabetes our resulting valuation of the increased longevity in diabetes in Sweden between the years 1897 to 1993 were 1,114 trillion SEK, which is a large number compared to any standard.

Keywords: *Value of health care intervention, Value of a statistical life year, Willingness to pay, Evaluating longevity gains*

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1. Introduction

To develop new technical- and medical innovations in health care is an expensive process and requires large investments from both public and private actors. Therefore significant sums of money are being put into medical research and technological innovations every year. At the same time longevity has been increasing worldwide (Murphy and Topel, 2003). Up until recently health economists seem to have believed that the contribution of medical care to the increase in longevity and other health improvements have been quite modest. However, more recent research is pointing in the direction that technological innovations and new medicines have had important positive impacts on health (Lichtenberg, 2005). Increasing longevity is one component to increased social welfare, but despite this it is not directly included in the most common measure on the wealth of a country, its GDP. Indirectly increased health is included since healthier people produce and consumes more, which effects the GDP, but no direct measure of health is included, since health does not have a market price (Nordhaus, 2003). Taking into account that health care costs represent such a growing proportion of GDP, it is of interest to evaluate the benefits of these large investments (Appendix, Figure 7.1). The benefits from improved treatments can lead to increased longevity, increased quality of life, reduced need of hospital treatment, increased productivity and less sick leaves from work and so on (Cutler and McClellan, 2001). In order to evaluate the total impact on society of technological innovations in health care both the costs and the benefits has to be taken into account. As long as the benefits outweigh the costs there is a positive net benefit. Since many studies focus on the costs of the investment in health care, we here focus on the gains and in this thesis especially on gains in terms of increased longevity.

There are many ways to evaluate the value of new technologies in health care. For different purposes and with varying intentions in mind, some studies have been made in the area of gauging gains from medical research. Kevin Murphy and Robert Topel, two well known authors who have been active in the health economic field of research, have developed a method based on willingness to pay (WTP) for measuring gains in health care and have edited a book, on the same subject, including other well renowned researchers, there among Lichtenberg. There are a few other famous researchers that have been active in this field; David Cutler, Mark McClellan, William Nordhaus and Frank Lichtenberg just to name a few (Cutler and McClellan, 2001) (Nordhaus, 2003).
We have chosen to focus on Lichtenberg and on Murphy and Topel much because their methods are different from the others as well as from each other. Lichtenberg's large perspective and

econometric model is very different from Murphy and Topel's top down perspective based on individual's willingness to pay. Because of their large differences, we feel that comparing them gives us a broader knowledge, better understanding and gives more information in the area, than if we would compare two more similar models.

1.1 The aim of the study

The aim of this master thesis is partly to discuss Lichtenberg and Murphy and Topel methods to evaluate gains in health care and partly to implement one of these methods empirically and thereby provide an answer to our question: what is the economic value from increased longevity? We use a bottom up perspective when illustrating this empirically and apply this method on the diabetes population in Sweden. We chose diabetes since it is a widespread disease with a growing prevalence that has been clearly affected by treatment innovations. Our hypothesis is that the improved treatment for diabetes has played a vital role in the increasing longevity for diabetic subjects.

1.2 The scope of the study

In our method analysis we limit ourselves to evaluating Lichtenberg and Murphy and Topel's methods thoroughly as opposed to making a complete literature review. This is to provide more space to make a comprehensive study of our selected methods. Further on, in our empirical implementation, we focus on one disease, diabetes, in one country, Sweden. The time period we study range from 1914, in order to include all important innovations and improvement in treatment of diabetes, and ends in 1993 due to data limitations.

We limit ourselves, in the empirical part when valuating health gains, by only looking at mortality decrease and chose to neglect improvements in quality of life, although we know improvements in health consists of both. This is since data on quality of life improvement is not easily accessible and includes subjective values. The effect from this limitation is that our results are an under estimation of the reality.

1.3 The outline of the study

We begin our thesis with background, theoretical concepts explaining further why we feel that calculating health gains is an important issue. This is followed by a presentation and discussion of the two methods we chose to analyse. Then we implement the method we find most appropriate to

get a value for increased longevity in diabetes subjects. The results of these calculations are followed by a discussion about how much of the value could be attributed to improved treatment. The thesis ends with a summary of our findings and our conclusion.

2. Relevant Concepts

In this part we discuss, for a better understanding of the methodological analysis, some important theoretical concepts: willingness to pay (WTP), willingness to accept (WTA) and quality adjusted life years (QALY).

2.1 Willingness to pay (WTP) and willingness to accept (WTA)

The concepts willingness to pay (WTP) and willingness to accept (WTA) have wide usage possibilities. Recent literature on economic evaluation in health and health care has shown increasing interest in the use of (WTP) as a measure of health benefits (Olsen et al, 1999). WTP is simply put, a measure for the maximum a person is willing to pay to avoid something undesired or to receive a good. It can for example be used to elicit how much money one is willing to sacrifice for increased lifetime. The opposite, willingness to accept (WTA), is a measure for the minimum a person requires as compensation for health deterioration or no health improvement. WTP and WTA are important ingredients of cost benefit analysis (CBA) which in turn is a tool for decision makers to value possible health interventions (Boardman et al. 2011).

When conducting a WTP or WTA economists use revealed preference (RP) method, stated preference (SP) method or a human capital approach. The RP method uses data from individual's actual behaviour while SP method uses answers from people from questions on how they would have behaved in different situations given certain hypothetical circumstances. The strengths in using RP techniques is that you know for sure what a person actually paid for a specific good, i.e. insurance premium or air bag for the car (Bateman et al. 2002).

The most common SP approach is contingent valuation (CV) studies, where the person is asked a dichotomous choice, a (yes/no) question, for example whether or not they would be willing to pay a certain amount for a certain risk reduction. Estimates based on SP studies generally give higher VSL estimates (de Blaeij et al. 2003) while RP is regarded to be more reliable (Sugden, 2005).
When using the human capital approach the present value of an individual's expected life earnings is summarized. This can be done for all different ages (Hultkrantz and Svensson, 2008).

2.2 Quality adjusted life years (QALY)

A quality adjusted life year (QALY) takes into account both the quantity and quality of life generated by health care interventions. It is the arithmetic product of life expectancy and a measure of the quality of the remaining life-years. A QALY weights the VSL by multiplying it with the quality of health during a particular year:

QALYt=wt*VOSL

(Boardman et al. 2011)

where the weight can take any value between 1 for perfect health and 0 when being dead.

Calculating life expectancy imposes few controversies since a person is either alive or not, whereas calculating quality of life is much more difficult since the definition of health is highly subjective and it has been argued that some health states are even worse than being dead. The QALY is needed in order to be able to take into account the fact that not all years are lived with the same high standard. Some years can be spent in better health than others. There is limited descriptive material on quality of life of persons with diabetes and very few (if any) isolated reports of the application of quality of life instruments in conjunction with interventions.

2.3 Value of a statistical life year

To put a value on a human life is a difficult and sensitive issue for many reasons. It imposes challenges in religious, moral and ethical beliefs to name a few. The allocation of scarce financial recourses is not always consistent with these beliefs. The arguments against valuing human life in terms of money are mainly two: Firstly, it is unethical and secondly, assessing a finite value on a human life is just simply wrong (Zweifel et al. 2009). Questions arise like why should the disabled, the old, the poor or other persons that have a lower productivity level's lives be valued less than any other's? Important then to keep in mind is that the trade off is normally not between life and money but instead between remaining life expectancy and money (Sund, 2010). This is perhaps an easier concept to accept morally.

Despite the fact that it is a sensitive and difficult issue, it is never the less an important and necessary one when it comes to evaluating health care. An intervention or investment, which aim is to reduce mortality risks needs a monetized value so that we can compare the social benefits they achieves with the economic costs they create. The monetized benefit of decreased mortality risk is

calculated using the value of a statistical life (VSL). VSL is in essence the value the society deems economically efficient to spend in order to prevent one premature death (Svensson, 2008). It can also be seen as the trade off between income and mortality risk reduction.

There are a numerous of different estimates for VSL. The range of values it can take varies tremendously across studies. It can take any value from 0 to 192 million SEK, but more recent studies narrows it down to between 22 million SEK (Boardman et al. 2011) to 58 million SEK (Viscusi and Aldy, 2003) for the US, where as in Sweden this figure is between 28 million SEK and 47 million SEK (Svensson, 2008). The reason for these differences is that there are different ways to go about measuring VSL.

3. Measuring the value of medical technologies

In this part we introduce and discuss the two methods chosen for our analysis. We start with the presentation of the method developed by Kevin M. Murphy and Robert H. Topel. Thereafter, we present two articles written by Frank R. Lichtenberg, who uses an econometric approach for estimating effects on longevity. Besides using different methods the objectives of their studies differ. The main objective with Murphy and Topel's model is to calculate a value for increased longevity whilst Lichtenberg's main objective is to examine the relationship between new drugs and increased longevity. We end this part with a discussion on the applicability of these methods to our objective.

3.1 Murphy & Topel - Estimating health gains using WTP

Murphy and Topel has developed a method using individual willingness to pay (WTP) for life to get a measure on health improvements (Murphy and Topel, 2005). The use of WTP to get a measure on risks is not a new phenomenon, on the contrary it is popular to use when valuing the trade off between longevity and goods in different areas, some examples are; the decision to smoke, tradeoffs between time and safety for modes of travel, trade-offs between costs and safety for consumer products, the choice of medical treatment options, or, probably the most used, for the choice among potentially risky jobs (Murphy and Topel, 2003). What Murphy and Topel do is that they develop a framework based on WTP that provides a value of increased health, or in other words, the value of increased longevity. With this framework past, present and prospective gains can all be calculated. The results of their calculations show large gains, and even when comparing it to the costs they are left with a big net profit. In the empirical part where they use their model to estimate the value of decreased mortality in the time interval 1970-2000 the results are extraordinary; 3,2 trillion dollar per year (in gains). This result indicates that even small changes in the survival probability may be the cause of large gains.

3.1.1 The framework building

Murphy and Topel's model has its foundation on the individual's maximization of lifetime expected utility. The utility is affected by the individual's consumption $c(t)$ and leisure $l(t)$. It is also affected by health which in this model is presented by $H(t)$ and $G(t)$. Medical advances that affect $H(t)$ improves the quality of life but not the chances for survival and advances that affect $G(t)$ improves the survival probability but do not affect quality of life. Naturally there are also medical advances that affect both $H(t)$ and $G(t)$ and they are also captured in the model. In (1) we can see the factors affecting the individual's utility.

(1) $\int e^{-\varrho(t-a)} H(t)\, u(c(t),\, l(t))\, S(t,a)\, dt$

$G(t)$ is not visible but it is included through the survival function $S(t,a)$ in the following way:

(2) $S(t,a) = \exp[\, -\int \lambda(\pi,\, G(\pi))d\pi]$

Where $\lambda(\pi,\, G(\pi))$ is the instantaneous mortality rate and $S(t,a)$ displays the probability that an individual survives from age a to time t. Murphy and Topel assume that the differential $d\lambda/dG$ is <0 which means that an increase in type-G health reduces mortality and increases the survivor function.

In order to maximise the remaining lifetime expected utility the individual chooses (1) subject to the budget constraint (3).

(3) $A(a) + \int [y(t) - c(t)]\, S(t,a)\, e^{-r(t-a)}\, dt = 0$

where:
r is the interest rate,
$A(a)$ is initial assets at age a,
$y(t)$ is life-contingent income at age t, determined by the choice of $l(t)$, $y(t) = w(t)[1-l(t)]+b(t)$

When the budget constraint is included we get a utility maximisation model for an individual, shown in model (4).

(4) $U(a) = \int \{H(t)u(c(t), l(t))e^{-\varrho(t-a)} + \mu[y(t)-c(t)]e^{-r(t-a)}\}\, S(t,a)dt + \mu A(a)$

where μ is the multiplier associated with the budget constraint. In this model (4) it is assumed that it is a perfect annuity market implying that at age a, the lifetime expected discounted value of future consumption must equal expected lifetime wealth. These necessary conditions when optimising (4) are shown in (5):

(5) $H(t)u'_c\,(c(t), l(t)) = \mu e^{-(r-\varrho)(-a)}$

$$H(t)u'_l(c(t), l(t)) = w(t)\,\mu e^{-(r-\varrho)(-a)}$$

By adding on to the model (4) Murphy and Topel are able to provide values for increased health. The basic thought is that if a representative individuals health increases then the value of that individuals remaining life will rise which means WTP for health improvement for this person will increase. This also implies that there is a complementary relationship between different diseases; if a new medicine increases survival probability for one disease, the person suffering from that disease is more likely to survive and therefore have increased WTP for other medical advances that increase health. Since WTP is determined by the expected discounted value of lifetime utility, the modelling continues by including valuation of a life year, valuation of remaining life and evaluating changes in these when health improves. We will go into detail with this in the next section.

3.1.2 Value of life and the willingness to pay for improvements in health

The expected utility of remaining lifetime for an individual at age *a* is expressed as a function of value of life year and the survival function in (6):

(6) $$V_\lambda(a) = \int v(t)e^{-r(t-a)}\,S(t,a)dt$$

where the value of a life year is $v(t)$:

(7) $$v(t) = (u(c(t), l(t))/u'_c) - c(t) + y(t)$$

What can be derived from the model for $v(t)$ is that the value of a life year depends of the instantaneous utility, $u(c, l)/u'_c$, plus net savings (y(t)-c(t)) that make it possible to consume in the future, with marginal utility μ. All of the variables included in $V_\lambda(a)$ are discounted, including $S(t,a)$. The term $H(t)$ is not included in the model of the value of life, implying that the quality of life is not affecting how individuals value their life. Factors that do affect the valuation of life in Murphy and Topel's model are age and income. Numerous other studies have also provided calculations on the value of life, but many of them do not include age as a variable. If age is not included it means that one values the life year of a 20 year old the same as the life of a 90 year old. Since the health and consumption actually do vary during the life it is more plausible to assume that the value of the 20 year old person's life is larger. Needless to say it is morally never an easy task to put an monetary value on a person's life and in addition rank whose life is more "valuable" than

another but since we are dealing with scarce resources it inevitably has to be done. Murphy and Topel extend the model to explain how the value of life changes during life:

(8) $v(t) = y(t)/v(t)\ [\ s_w(t)\ w(t) + (1\text{-}s_w(t))\ b(t)] + [1\text{-}\ y(t)\text{-}c(t)\ /\ v(t)\ ]\ [\ H(t) + r\text{-}\rho]$

The understanding of the first term of this model is that the income and age profile of the value of life is connected. The part $s_w(t)\ w(t)$ ties the value of a life year to the wage profile. For a working person $s_w(t) = 1$, and for a retired person $b(t)=0$. The second term of the model ties the $v(t)$ to changes in health, consumption and leisure.

The framework discussed so far, can be used to get an estimate on how much people are willing to pay for improvements in health, and thereof how much people are willing to pay for medical advances that leads to these improvements. To illustrate how improvements in health affect WTP they use a factor α that represents a medical advancement that can improve both quality of life as well as the longevity. Examples for what α could be is a new treatment technique, new medical knowledge, or improved access to medical care. Again, using the basic building block (4) they estimate the marginal value of α:

(9) $V_\alpha\ (a) \equiv U'_\alpha\ (a)/\ \mu = \int v(t)\ S(t,a)\Gamma_\alpha\ (t,a)\ dt + \int H'_\alpha\ (t)/\ H(t)\ u(c(t), l(t))/\ u_c\ S(t,a)\ dt$

where: $S(t,a)\Gamma_a\ (t,a) = dS(t,a)/d\alpha$
and: $H'_a\ (t) \equiv dH(t)/d\alpha$

A new treatment or technique affects health, in both G(t) and H(t). The change in G(t) is measured in $(dS(t,a)/d\alpha)$ which is the marginal change in the probability of survival as H(t) is held constant. The change in type H- health is seen in the second term of (9), and it measures how the quality of life changes as mortality is held constant. Proportional changes in both types of health are valued the same when savings are insignificant. With this model we get the monetary value of the increased lifetime expected utility of α, since it measures the resulting increase in expected lifetime utility.

To get a monetary value of the increased longevity, the value of a life year is used. Murphy and Topel calculate value of a life year $v(t)$ using shadow values for full consumption and full income (c^F, y^F):

(10) $c^F = c + z_l / z_c \, l = z_c^{-1} z$

(11) $y^F = y + z_l / z_c \, l$

where z is a factor that aggregates consumption and non market time. These shadow values are used in the following way to get an expression of value of a life year:

(12) $v = y^F + c^F \phi(z)$

where $\phi(z)$ = consumer surplus per unit of factor z. This is then substituted into equation (9):

(13) $V_\alpha(a) = \int [y^F(t) + c^F(t)\, \phi(z(t))]\, S(t,a) \Gamma_a(t,a)\, dt + \int H'_a(t)/H(t)\, c^F(t)[1 + \phi(z(t))]\, S(t,a)\, dt$

Equation (13) is an expression of the WTP for improvements in health by for individual at age a. The formula (12) can also help us to get an equation for the value of an age-a statistical life.

(14) $V_\lambda(a) = \int [y^F(t) + c^F(t)\, \phi(z(t))]\, S(t,a)\, dt$

The model (14) shows us that WTP for improvements in health is proportional to full income and full consumption. This can be interpreted as wealthier individuals (or societies) have a higher WTP than not so wealthy individuals (or societies). To clarify, the model for $V_\lambda(a)$ provides a way to calculate the value of remaining life, and that value changes, as mentioned before, with age. Murphy and Topel show in a simple example how this can be done. First they choose the conditions they have at hand. Let's say that we want to know the value of a life year for a 50 year old male. To get the value of $y^F(t)$ they use wage profiles, assuming that the individual earns 60,000 dollar for 2000 hours of work in a year. They also assume that $y=c$ indicating that they assume the individual does not save any of its income. That provides us with the value for a statistical life with the given conditions.

3.1.3 The social value of health improvements

By using the model to calculate the value of health improvements to a representative individual, the results can be transformed to a society level. One key factor to have in mind when it comes to evaluations of medical improvements, is that the effect may not be visible until further in the future. Murphy and Topel have thought of this and provide a way to estimate the value of α for past, present and future populations. The basic formula for WTP is extended in the following way:

(15) $W_\alpha(\tau) = \int_{a=0} N(a,\tau)\, V_\alpha(a)da + N^f(\tau)V_\alpha(0)$

where $N(a,\tau)$ = the population of age a and τ
and $N^f(\tau)$ = the discounted value of the number of births in future years

In both terms the V_α is included, first depending on age (a) and in the second term from birth.
The model (15) measures current social value of advances that improves health from time τ and forward. Since the model shows that the value of α is proportional to both $N^f(\tau)$ and $N(a,\tau)$, we know that the bigger the population, the larger is the value of α. This model provides a way to calculate the gains for a time period since it shows how much an individual with same age and gender would be willing to pay to have the survival rate of time A instead of the survival rate at time B. This last model is the last piece needed to make calculations on the value of α over time. This measures the gain, or the benefit of α, which is used to get a measure on the net benefits to society.

3.1.4 Empirical results from WTP model

Murphy and Topel apply their model both to calculate gains over time and disease specific gains. To get the net benefits of the value of increased longevity between years 1970-2000 in the US, they use mortality data that is both age and gender specific. Since it is possible to calculate the age profiled value with the model, and to measure for the past population, they can estimate the gains for the entire period. The value of increased longevity at past dates is measured in current WTP for different ages. This estimates the value for individual's year 2000 for the increased longevity that has been achieved in the past. Further on, the present discounted value of changes in survival for different ages, for the whole decennium.

Focusing on the years between 1970 and 2000, the total increased longevity has one major explanation; increased survival for heart disease. The reduced mortality from heart disease alone

accounts for 2/3 of the overall decline during this period. Only for these 30 years the total social gains is calculated to be 95 trillion dollars, which is 3,2 trillions per year (measured by (15)).

In economics, we know that the gains are only one side of the equation. To get a measure on the net benefits these gains have to be compared to the costs. Murphy and Topel provide a model that includes the costs of α.
The health expenditures at time t, $k(t)$ is included in the model (13):

(16) $$V_\alpha(a) = \int [y^f(t) - k(t) + c^F(t)\,\phi(z(t))]\,(S_\alpha(t,a)\,k_\alpha(t))dt - \int k_\alpha(t)S(t,a)dt + \int (H'_\alpha(t) + H'_k(t)\,k_\alpha(t)) / H(t)\,c^F(t)\,[1 + \phi(z(t))]\,S(t,a)dt$$

Where $k_\alpha(t)$ is change in health spending at age t. The costs are defined as both development and implementing costs. However, Murphy and Topel never implement this model in their calculation, because it demands data on the specific costs for α, which can be hard to find. Instead aggregated costs for all different medical advancement are used. They get the estimates for the costs using data from medical expenditure survey from 1977 and 1987, and then they use the age profile to get an estimate on aggregated health costs for the whole period 1970-2000. This results in an estimate of total health care costs of 34 trillion dollars. Putting this in relation to the gains it results in net gains of 61 trillion dollars.

3.2 Lichtenberg-Estimating health gains using an econometric approach

A well recognized name in the field of health economics, Frank R. Lichtenberg have received much attention for his studies on the relationship between new drugs and increased longevity. Below we present a summary of two of his articles that in particular analyses the specific increase in longevity and medical spending due to new medical innovations. The first article from 2001 has the objective to analyse if the benefits from new drugs are worth their costs, while the second article from 2005 analyse the impact of new drug launches on longevity. The aim with his method is to illustrate the benefit of drugs in terms of longevity and to put it in relation to the cost for developing them.

3.2.1 Are the benefits of newer drugs worth their costs?

It is well known that new medicines are research intensive and constitute a significant part of the explanation of rising costs in health care around the world. Despite this fact, according to Lichtenberg, little focus has been put on trying to estimate the possible benefits these new drugs

accounts for. The aim with Lichtenberg's article ”Are the benefits of newer drugs worth their cost” is to evaluate if the age of a drug affect the possible drug offset, which is if the benefits of a new drug outweigh the costs for developing it (Lichtenberg, 2001). Lichtenberg's hypothesis is that new drugs are more effective which lead to less costs for overall treatments and therefore might lead to a drug offset.

Lichtenberg uses an econometric framework to test the hypothesis. He uses panel data from a medical expenditure panel survey; MEPS for the US population for the year 1996. The survey contains information about different diseases that individuals participating in the survey have suffered from, as well as detailed information associated with that particular disease, such as which drug were prescribed for what condition, possible loss of work days, nondrug treatment like hospital stay etcetera. The unit that is interesting for this analysis is the events of prescribed medicines. Each observation on prescribed medicine contains information on the amount paid, who paid for it and the national drug code. The drug code is then used for deciding the age of the drug, which is defined as the logarithm of the number of years prior to 1996 that the active ingredient in a prescription was approved by the food and drug administration (FDA). The data available in the MEPS is used to analyse the effect drug age has on three variables: mortality (if a person was dead in the end of survey), morbidity (missed school days, missed job days) and total medical expenditure (nondrug medical events associated with the condition). These factors are possible to control for since the MEPS data contains detailed information on people dying and costs for the different conditions. The results of the analyse support Lichtenberg's hypothesis showing that the use of newer drugs contributes to reduced nondrug spending.

3.2.2 Impact of new drug launches on longevity

Lichtenberg's article from 2005 is a continuation of the results from the 2001 article, but the objective comes closer to the type of analysis we are interested in (Lichtenberg, 2005). The basic hypothesis is that if new drugs are more efficient than older ones than a large amount of new drugs launched would be positively correlated with reduced mortality. The question he poses is; have new drugs contributed to longevity in 52 countries during the time period 1982-2001. In 1994, the US spent almost a third of their total health R&D and more than half of industry health R&D expenditures on pharmaceutical industry R&D expenditure. Although the costs for new drugs are high there are clinical studies indicating that new drugs increase longevity for several diseases

compared to the older drugs. So, these expenditures could be offset if the benefits in terms of increased longevity, are sufficiently high from the new drugs.

Lichtenberg designs an econometric model using panel data with fixed effects to test the hypothesis. The advantage of panel data, compared to time series and a cross sectional data sets, is that they allow for identification of certain parameters without having to make limitative assumptions. They can identify whether a rise in life expectancy from one year to another, for example, is a result of an overall increase for the population or if the increase is larger for the younger and less for the elderly. In other words, panel data are not only able to explain why individual units behave differently but can also explain why a given unit behaves differently in different time periods. With panel data it is possible to exploit the particular nature of the data because of the repeated observations on the same individual.

(1) $AGE\_DEATH = \ln \beta(N\_DRUG_{ij,t-k}) + \gamma X_{ijt} + \varepsilon_{ijt}$

In Lichtenberg's model the dependent variable AGE_DEATH stands for the age distribution of deaths from disease *i* in country *j* year *t*. The model measures how the AGE_DEATH is affected by N_DRUG_{ijt}, which is new drugs that was launched to treat disease *i* in country *j* year *t-k*. The variable N_DRUG is lagged with *k* years since it normally takes some time for new drugs to be established on the market. The variable X_{ijt} is a vector for all other factors that can affect the age distribution of deaths, for example: gender, education, nutrition etcetera. The last and final term in the model is an error term, the residuals, which captures everything that cannot be explained by the model itself.

Lichtenberg states some hypothesis for the model. First, the model is based on the hypothesis that the age distribution from disease *i*, in country *j* year *t* depends on the cumulative amount of drugs launched to treat disease *i*, in country *j* year *t-k*. A second hypothesis is that all other factors in X_{ijt} are invariant across diseases within a country and year, invariant across countries within a disease and year, as well as invariant across years within a disease and country. These assumptions are explained in the model (2), and then substituted into model (1) controlling for all other factors. Lichtenberg solves the problem with individual changes by capturing all observable disease or country differences over time by using fixed effects. For instance, if assumed that income is an

important factor affecting the age distribution of death, and that income is correlated with number of drugs, then it would be considered invariant and therefore be controlled for by δ'_{jt}.

(2) $X_{ijt}=\alpha'_{it}+\delta'_{jt}+\theta'_{ij}+\nu'_{ijt}$

α'_{it} = fixed effect for disease *i* in year *t*
δ'_{jt} = fixed effect for country j year t
θ'_{ij} = fixed effect for disease i in country j
ν'_{ijt} = error term

The data used is from IMS health drug database which has records on all new drugs in 52 countries from 1982, and it contains detailed information including primary disease for each new drug, the country where the drug was produced, which year etcetera. The purpose with using this data is to make estimates on number of drugs launched for disease *i*, in country *j* year *t-k*. First Lichtenberg constructs a list of all different ingredients in IMS, and one list for each country. These lists were then merged together with a list for the chemicals attribute and the numbers of new chemical launched (NCEs). NCEs are an important factor in the model since the aim is to measure the effects of new drugs on longevity. A drug that has been launched during the time interval 1982-2001 is not necessarily new, it could be a re-launch. Therefore the drugs launched in the time interval of interest are divided into the groups NCEs and non-NCEs. This is included in the model as follows:

(3) $AGE\_DEATH_{ijt} = \beta_{NCE}\ln(CUM\_NCE_{ij,t-k}) + \alpha'_{it} + \delta'_{jt} + \theta_{ij} + \mu_{ijt}$

(4) $AGE\_DEATH_{ijt} = \beta_{NCE}\ln(CUM\_NCE_{ij,t-k}) + \beta_{NONln}(CUM\_non\text{-}NCE_{ij,t-k}) + \alpha'_{it} + \delta'_{jt} + \theta_{ij} + \mu_{ijt}$

CUM_NCE = cumulative number of NCEs launched
CUM_non - NCE= cumulative number of non-NCEs launched

In (4) the AGE_DEATH depends on both NCEs and nonNCEs which makes it possible to isolate the amount NCEs has contributed to AGE_DEATH. Here, one more hypothesis is made, namely that NCEs has a larger effect on AGE_DEATH than non-NCEs, and this is based on the belief that the amount NCEs consumed is positively correlated with the number of NCEs launched. If nonNCEs are launched it takes marketplace from NCEs and therefore has a negative impact on longevity.

To get an expression of the AGE_DEATH two different samples of life tables are used, time series of decennial life tables for US from year 1900-2000 and cross section of life tables for 191 countries year 2000 from WHO mortality database. One issue is that the samples show the length of life and survival probabilities for the entire population in different countries, but not for different disease specific groups. Lichtenberg solves this problem by dividing deaths and drugs into 11 broad disease categories. Later on in the article Lichtenberg makes a regression of the variables life expectancy at age a (LEa), and probability of survival from birth until age 65 (SURV65). With this calculated regression lines it is possible not only to estimate the impact of new drug launches on life expectancy at given ages, but also for the life expectancy for the entire population (LEpop). This is done by calculating weighted average of the regression coefficients weighted by the share of population in each age group.

3.2.3 Empirical results using the econometric model

The main objective of Lichtenberg's analysis is to get an estimate of how much of the increase in longevity in the long run that can be explained by the launches of NCEs. By putting the resulting estimates in relation to the costs it is possible to get a value of the cost per life year gained from the launch of NCEs. Lichtenberg suggests two different approaches that results in unequal results. The difference in the approaches is whether the estimate reflects the effect of medical innovation in general or the effect of new drug launches.

Using the last mentioned approach, that the estimate reflects the effect of new drug launches, the model provides the result 0,2145 representing the effect of NCEs on average life expectancy for the entire population. This is calculated using the aggregated life tables;
Life expectancy at birth (LE_0) = 63,2 * βNCE

The number 63,2 is derived from the regression of life expectancy. It is the average of the slope of the regression for the US and the international data, and βNCE = 0,0065 when using a 4 year lag. The 4 year lag is used since the calculations show that it generally took 3 years for a drug to be established on the market. The cost per life year gained from NCEs is calculated by using the average per capita pharmaceutical expenditure in OECD countries, which is 250 dollar. This is divided with the average annual increase in longevity for the entire population 0,056, which is derived from calculations on how much NCEs has contributed to the increasing longevity as a whole. This is calculated with the average growth of death rates from disease *i* country *j* year *t* that occurred at age 65+.

The estimate on how much NCEs has contributed to the increased survival probabilities is then calculated 0,0065*λt-λ1986, and the contribution of NCEs to increased LEpop is calculated with 33 (mean life expectancy)*0,0065 *λt-λ1986, where λt-λ1986 is an estimate of the change in survival rates in the time interval 1986 -year t (the number 0,0065 is equal to β_{NCE} with a 4 year lag). The result is that NCE launches accounts for 0,79 percent of the entire longevity increase between the years 1986-2000. The annual effect for the entire population is 0,79/14= 0,056. Finally, it results in an estimate for cost per life year gained by NCEs: $250/0,056 = $4500. This cost may be even lower since there may be evidence that new drugs accounts for only half of all drug costs which would implicate that the real cost per life year gained may be only $2250.

Lichtenberg briefly discusses how these results would change if using the other approach mentioned; that the estimates reflect the effect of all medical innovation. Since it is not possible to measure the non-pharmaceutical innovations, Lichtenberg uses an estimate of how large the share of the US health R&D is in relation to pharmaceutical R&D, which is 1/3. From that perspective he makes the assumption that:

$$\frac{\text{Effect on LE of new drug launches}}{\text{Effect on LE of all medical innovations}} = \frac{\text{Pharmaceutical R\&D expenditure}}{\text{Total R\&D expenditure}}.$$

That implies that only a third of the estimated effect of new drug launches on longevity is attributable to new drugs, the remaining two thirds are attributable to all medical innovations. That of course would increase the cost per life year gained since it reduces the benefit of drugs. Lichtenberg estimates that the cost per life year gained then would be $6750, which is still far lower than most estimate of the value of a statistical life-year.

3.3 Methodological discussion

We have now presented two different methods which both concerns the gains in longevity from medical advancements in different ways. We continue with a discussion about some critical points of both methods, and also explain how and if they are applicable for analysing the gains of increased longevity due to decreased mortality from diabetes.

3.3.1 Lichtenberg

When using an econometric approach it is possible to get an estimate for how much the independent variables affect the dependent variable. Lichtenberg, in his articles, analyses how much more effective new drugs are compared to the older models in terms of increased longevity, and how much of the long-run increase in longevity is due to the number of new drugs on the market.

Although we find some critical points, Lichtenberg's analysis is very detailed and extensive based on very large datasets on aggregated and disease level longitudinal data from 52 countries. By including additional variables in the model it enables Lichtenberg to control for effects of other potential determinants that are invariant across, country and gender in order to end up with only significant variables. It is evident that this type of method serves the purpose well, when the purpose is, like in Lichtenberg's case, to find a correlation between variables. In this case he uses this method to find a value for new drugs.

One critical point is that Lichtenberg only confirms that on aggregate level, *all* new drugs lead to an increase in longevity. He does not discuss the possibility that drugs may vary in effect. It could be that one drug has contributed more to the results and that others have contributed marginally or not at all. It could be that a successful NCE has a higher pay off overshadowing the less successful NCEs and thus, the more NCEs that are produced the higher the probability that one is successful.

Lichtenberg points out that heterogeneity with respect to NCE launches appear to explain very little of the variation across countries. This could simply be due to technology dissemination. It is probably safe to assume technologies do not get stuck at the borders. Even if a country is incapable of developing and producing NCE:s themselves, as long as that country approves of NCE:s from another countries it can still benefit from them. On the other hand time series differences do appear to account for a significant fraction of the long run increases in longevity, indicating that more NCE launches results in increased longevity. Another issue that arises when using an econometric method is the risk of causality. In this case it could very well be that since people live longer they are more likely to invest more money in health improvements (Murphy and Topel, 2005). With this in mind it is not certain that the number of drugs affect the longevity, it could also be that the increased longevity affects the demand of new drugs and therefore more new drugs might be produced.

3.3.2 Murphy and Topel

Murphy and Topel's framework has its main strength in its applicability. Since it has its foundation on willingness to pay, it can be applied on an individual level or on a population without much difficulty. A particular strength of the model is the inclusion of age in the calculation for value of life as well as the value of non market time for non working individuals. This all affects WTP and therefore reflects the reality to a broader extent. Although the model Murphy and Topel presents is detailed and captures many factors, we would like to discuss and draw some attention to some of its weaknesses.

Firstly, one critical point the authors themselves point out is the exclusion of the quality of life in the calculations, which is also a problem in Lichtenberg's case, but in contrast to Lichtenberg Murphy and Topel discusses the issue. The reason for the excluding of quality of life is that mortality data is easy to find and the increased longevity is directly measurable. Although it is possible to put a monetary value on the changes in the quality of life, it is not directly measurable (Murphy and Topel, 2005). Nevertheless, there is no doubt that increasing quality of life is valuable and that if included in the model the gains would be even larger. Even if they do not calculate exact values Murphy and Topel provide a way to roughly estimate changes in the quality of life by using mortality data that shows mortality at age *t* both 1970 and 2000, knowing that the mortality has decreased, and assuming that mortality is associated with the quality of life. When getting a measure for how much longer people at age *t* live in the year 2000 than in the year 1970 this can be interpreted as how much "younger" people have become. The results from the age profile showed that the value of life is higher for younger people, therefore giving a total higher value. This increase is then the result of increased quality of life. However, this is given as a hypothetical model and not something empirically measured. The fact that the effect on quality of life is not included in the final calculations implies that the results are an underestimation of the total value. Murphy and Topel even suggest that the value of the increased quality of life may be even larger than the value of the increased longevity.

Secondly, as opposed to Lichtenberg, Murphy and Topel assumes that all gains in longevity can be dedicated to improvements in health care and medical innovations. It is not too farfetched to believe other things can affect longevity, such as nutrition, housing and public information campaigns to mention a few (Siegler et al. 2003). This makes us compare apples and pears when comparing the benefits from increased longevity, which can be a result of many factors, and the costs for health care alone. The costs of the other factors are not included in the calculations and therefore the result may be overestimated. In a critical article by Siegler et al. (2003) they suggest that Murphy and

Topel's model should be applied to a disease that is less effected by external factors like for example diabetes type 1, since the mortality development from that disease nearly only depends on medical advances. The problem is then to find data on only diabetes type 1, which is as we mentioned earlier a known problem.

Lastly, another part of the model that we want to address is the part designed to calculate net gains. In equation (16) Murphy and Topel provide an extension of their model that includes the costs of the medical improvement α, but they never use this model in their calculations since they find that the costs for one specific improvement or drug is both hard to define and to measure. They therefore never calculate net gains on a disease level, only on a social level comparing the gains with the aggregated health care costs over one period of time. Since they do calculate gains on a disease level one would assume that the net gains could be obtained, but the model does not offer this possibility.

3.3.3 Concluding remarks on Lichtenberg and Murphy and Topel

In our case, it is clear that there is a positive relationship between the treatment development for diabetes and the increased longevity among subjects with diabetes. It would not be impossible to redesign Lichtenberg's model to fit one single disease and one single drug, but it requires large amount of very specific data for the results to be reliable, which is difficult to find in the case of diabetes. We believe this goes beyond the scope of our thesis though, having said that, it would be an interesting study to pursue. For example one would need data specifically for all drugs and treatments on diabetes and the effects of these drugs and treatments. If that kind of data was available a possible model following Lichtenberg's method could look like this:

$$AGE\_DEATH = \beta(DIABETES\_DRUGS) + \beta(OTHER\_DRUGS)\ \gamma X_{ijt} + \varepsilon_{ijt}$$

With a model like this we could test the relationship between the number of diabetes drugs and the age of death distribution. Although, one issue would be that it would not be evident how the mortality changes for diabetic subjects, which is what we are interested in. Also, there is a correlation between diabetic drugs and other drugs in a way that diabetic drugs may be more efficient when it is used together with some other drugs.

When it comes to Murphy and Topel's model it simplifies the reality and it provides a way to get a monetary result on the gains from increased longevity. An advantage compared to using an

econometric method is that it is possible to calculate the gains for one individual or for a chosen population. It requires mortality data for the population at interest and a value of life measure, which is far less than Lichtenberg's method requires. We find that an econometric approach would be favourable if our objective would be on an aggregated level. In our case Murphy and Topel's model is favourable, since our objective is to analyse the increased longevity from one specific disease. Their method is also more suitable since it is possible to find the data needed and it is doable in our time frame.

4. Illustrative Empirical Application on Diabetes

Inspired by Murphy and Topel's method we here study the changed longevity for diabetic subjects in Sweden, and thereafter put a monetary value on it. We present some basic information about diabetes and its treatment. That is followed by a presentation of our method and the data we use. In this part we also discuss the important issue of valuation of a statistical life. The results are followed by a discussion around the results.

4.1 Diabetes Mellitus

Being a world spread chronic illness; diabetes causes not only death but also large costs to countries all over the world. The prevalence of diabetes is rising, and is estimated to rise even more so in the future (*Figure 1)*.

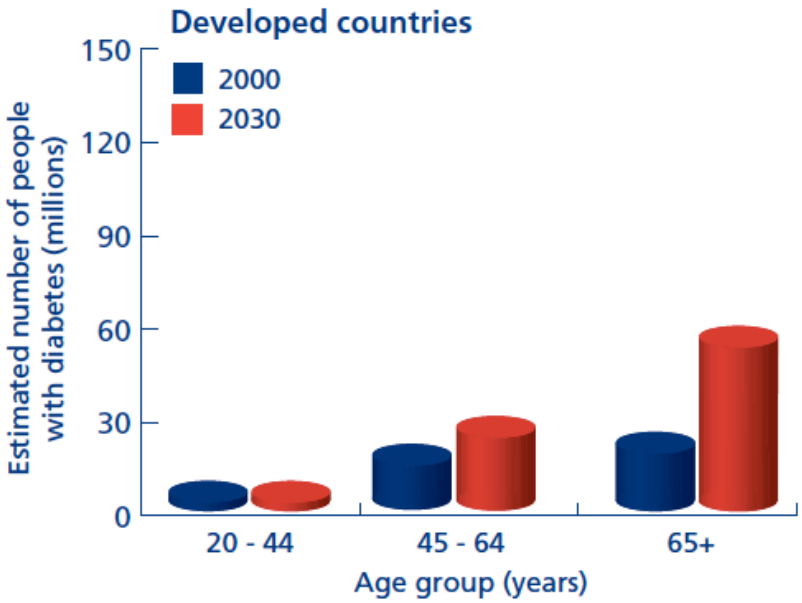

Figure 1. Showing the estimated number of people with diabetes in the different age groups in year 2000 and 2030 (www.diabetesatlas.org).

Although some developed countries have documented an improved survival of subjects with diabetes, the increased prevalence is most likely due to a rise in incidence rather than improved survival (www.diabetesatlas.org). According to Bolin et al. (2009) there were around 250 000 diabetics in Sweden year 2005. This is an increase from 1987 when only about 150 000 people were diagnosed with diabetes (Bolin et al. 2009).

Diabetes mellitus is a classification for the different variants of the disease diabetes. It can be distinguished between two types, type 1 and type 2 diabetes (www.diabetes.se, 1). The majority of cases are diagnosed with type 2 diabetes while the less common type, type 1, accounts for about 10-15 percent of all diabetes cases (Bolin et al. 2009). The difference between the two types is mainly the underlying factors that cause the disease. The reason for type 1 diabetes is that the immune system attacks the pancreas where the insulin is produced. It leads to a decreased insulin production, and finally to a complete stop. Type 1 diabetes cannot be prevented nor can it currently be cured and is thus often more severe than type 2. The underlying factors for diabetes type 2 are numerous. A key feature is that not only does the body produce too little insulin, but also the body cells have a reduced ability to absorb it (www.diabetes.se, 2). Individuals diagnosed with type 2 diabetes are often also overweight, have high blood pressure and a high level of body fat (www.skane.se ”Hälso- och sjukvårdsprogrammet för diabetes mellitus”, 2002).

4.1.1 Diabetes treatment and mortality

The human body needs insulin to control the blood sugar level and it is vital for the body to function. Without insulin the blood sugar will rise and high blood sugar is associated with complications and can in severe cases be very serious and even fatal. Artificial insulin has been used as a treatment for diabetes for nearly 90 years and has continuously been improved and tailored. Today it has increasing similarities to normal insulin production (www.insulin.se). Insulin has improved the conditions for people living with the diabetes. Most commonly, insulin treatment is given to persons with type 1 diabetes, although today an increasing number of persons with type 2, are also treated with insulin (www.diabetes.se, 2). When given to a type 2 person, it has probably been preceded with actions to change the lifestyle of the individual, for example improved nutrition or exercise or other medical treatment (www.skane.se, ”Hälso- och sjukvårdsprogrammet för diabetes mellitus”, 2002).

In 1920, before the introduction of insulin, the expected survival for a child with diabetes was only one to two years after diagnose (Mark, 1964). After the introduction the disease went from being a deadly disease to ”only” a chronic disease. Regular insulin treatments began in 1922 and according to Nishimura et al. (2001) the effect was immediate. In 1930 the survival probability for someone diagnosed with diabetes was already 90 percent and has been increasing ever since (Nishimura et al. 2001). Extensive research has been made worldwide with the objective to measure the mortality for subjects with diabetes over a longer period of time (Jansson et al. 2010, Eliasson et al. 2008, Marks,

1964, Thomas et al. 2003, Chan and LaPorte, 1996, Weiderpass et al. 2000, Nishimura et al. 2001, Borch-Johnsen et al. 1986, Panzram, 1987, Panzram and Zabel-Langhennig, 1981 and Edward et al. 2007). Their results differ some but give an overall picture that the mortality has decreased but that diabetic subjects still have an excess mortality relative the average population.

To measure mortality due to diabetes is not an uncomplicated task since the individual rarely dies from the actual disease itself but from its various complications. Health statistics therefore often underestimate the burden of mortality. Largely because diabetes is often omitted on death certificates as contributing to death (Weiderpass et al. 2001). The most common complications are cardiovascular disease, metabolic complications and neurological symptoms (Bolin et al. 2009). Another issue that is often pointed out is the difficulty in distinguishing the type of diabetes, for example the international classification of drug codes (ICD) which is used to classify diseases in death records make no difference between the type 1 and type 2 diabetes. These problems indicate that there is a risk that mortality statistics on diabetes may be biased (Panzram, 1987). A solution to this problem is to classify the types by age or age at the onset of the disease. Weiderpass et al. (2001) classify the studied population into insulin dependent diabetes mellitus patients (IDDM) which includes those under the age of 40, and those over the age of 40 are classified as non-IDDM. This method provides results showing that the mortality for IDDM patients was higher but also decreased the most, indicating that the treatment has been most effective for this group. This result is also consistent with the results from a study made by Borch-Johnsens et al. (1986). This indicates that diabetes type 1 is more severe but, at the same time, that insulin treatments have had a major impact on the mortality for diabetes subjects.

4.2 Method

Inspired by Murphy and Topel's method, our aim is to evaluate the increased longevity for diabetic subjects in Sweden compared to the average population over a longer period of time. The treatment for diabetes improved when insulin was launched in 1922. We look at the time period 1897 to 1993 in order to get an idea of the situation before and after this launch.

In Murphy and Topel's empirical part of their article from 2005 they first calculate the additional life years due to decreased mortality. The additional life years are then valued by using a value of life year estimate. Since Murphy and Topel use data on USA to calculate the value of a statistical life year, we make our own estimates on the value of a life year that is more suitable for Sweden.

4.2.1 Calculating the Value of a statistical life year

As we have mentioned earlier there are a numerous of different estimates for VSL. The range of values it can take varies tremendously across studies. It can take any value from 0 to 192 million SEK, but more recent studies narrows it down to between 22 million SEK (Boardman et al. 2011) to 58 million SEK (Viscusi and Aldy, 2003) for the US, where as in Sweden this figure is between 28 million SEK and 47 million SEK (Svensson, 2008). The reason for these differences is that there are different ways to go about measuring VSL.

We use a human capital approach to find a value of a statistical life year, which is the present value of an individual's expected earnings (Hultkrantz and Svensson, 2008). This is similar to Murphy and Topel's calculations on the value of a statistical life, since it is based on income and consumption, with the assumption that the consumption is equal to the earnings (Murphy and Topel, 2003). Since we are interested in the Swedish population we use mean income data from Swedish bureau of statistics for all different profession, for both women and men and for all sectors for the year 2009. This amount is 27 900 SEK per month. To get the value for a year we multiply 27 900 with 12,2 months, which then also includes holiday pay (www.spp.se). The result of 12,2 * 27 900 is 340 380 SEK which is our value of a life year. We include payroll taxes which 2009 was 31,42 (www.ekonomifakta.se). The final result for the value of a statistical life year is then 447 327 SEK. In Murphy and Topel's analysis they calculate the value of a life year for different ages. In our calculation we do not take ages into consideration since we will only use average results.

Our resulting value of a life year is significantly smaller than Murphy and Topel's measures on the value of a life year. They find that the value for a life year for a 50 year old male is between 1 240 200 SEK and 2 313 320 SEK. However, our value is larger than the value given by Lichtenberg which is 160 648 SEK (Murphy and Topel, 2005). It is clear that the value of a life year changes depending on what is included and how it is measured.

4.2.2 Life table calculations

Like in Murphy and Topel's calculation we want to get a value for the additional life years due to decreased mortality in diabetes. Since we do not have data directly for life expectancy for diabetics we transform mortality figures to life expectancy figures. This is done with life table calculations (Appendix 7.3). We make this type of transformation on both mortality data for diabetic subjects and for the average population. This transformation demands mortality data for different age

intervals, and between the years 1897-1960 we only have mortality data for specific ages for diabetic subjects. This is solved by making a linear interpolation between the age observations available.

Another issue in the transformation from mortality figures to life expectancy figures, is that the mortality data on diabetic subjects up to year 1960 only extends to age 60. This becomes a problem since the age 60 then becomes the maximum age in the calculation, which results in too low life expectancy estimates. We solve this issue by assuming the mortality (q) for diabetic subjects increased with age at the same rate as for non diabetic subjects after age 60. The difference in the resulting life expectancy due to this assumption is not huge but it improves the life expectancy calculations. In order for this not to affect the credibility of our further analysis we only look at the change in life expectancy for diabetic subjects between the ages 10 to 60.

These calculations provide us with a value for life expectancy at different ages and not from birth which is more common. To make it more understandable we have used the mean of these results.

4.2.3 Evaluating the gains in longevity for diabetic subjects

We are first and foremost interested in the changing life expectancy for diabetic subjects in Sweden, but since the life expectancy for the average population also has improved we put the development for diabetic subjects in relation to the average population. Since the mortality data we use for diabetic subjects are based on the US population we first make a comparison between the life expectancy of the average US population and the average Swedish population to ensure that they do not differ too much. We compare the mean life expectancy for every year, for all ages. The results of this comparison show no large differences (see figure 1). With this in mind we feel safe using the data from the US diabetes subjects as a proxy for the Swedish diabetes population when comparing it to our Swedish data further on in our calculations.

We compare the development of life expectancy for the diabetic subjects with the average Swedish population (see figure 2). Throughout our calculations we use the mean remaining life years for all ages for different years, as opposed to life expectancy at birth. This is done by adding all remaining life years for a year and dividing this sum with all the ages. When comparing the diabetic subjects and the average population we get the difference in remaining life years. We put a monetary value on these years with our calculated value of a life year, 447 327 SEK.

If we were to offer a person with diabetes in the time period 1991-1993 the option of having his current life expectancy or having the life expectancy from the time period 1897-1914 our results suggests that he would not accept this offer for a sum smaller than 7 425 628 SEK. This figure is derived from multiplying the decrease in the gap between the remaining life of the average- and the diabetic population, 16,6 years, with the value of a life year, 447 327 SEK. We do not discount this result since we are only interested in the value from the accumulated years gained and 1 year 1897 is still valued as 1 year 1993.

In 1987 there were 150 000 diagnosed diabetic persons in Sweden and in 2005 this number was 254 000 (Bolin et al. 2009). If we assume there were around 150 000 persons suffering from diabetes in 1993 the total gains would be about 1,114 trillion SEK. In 1993 the excess mortality for diabetic subjects compared to the average population in Sweden was 14,2 years. If a cure for diabetes were to be found and thereby eliminating this gap, this would be worth about 953 billion SEK.

4.3 Data

The mortality data we use for diabetic subjects is derived from two sources. For the time span from 1897 to 1960 we use mortality data collected by a physician in Boston named Elliott P. Joslin, who during his work systematically followed up on his diabetic patients, which by his death were about 50 000 persons. The population included in the data is fairly representative for the US population (Marks, 1965). For the period 1960 to 1993 we use mortality data for diabetic subjects from vital statistics for the US (www.cdc.gov).

The mortality data for the average population in Sweden for different ages and years is taken from life tables from the Swedish bureau of statistics (Statistics Sweden). Corresponding data for the US population is from the department of demographics, university of California, Berkely (www.demog.berkely).

4.4 Results

In figure 1, the life expectancy for a 10 year old person in the USA is compared to the life expectancy for a Swedish 10 year old. As we can see, the life expectancy for a 10 year old in Sweden and USA have developed in a similar way, except maybe for the time period 1921-1930 where we can see a slight gap.

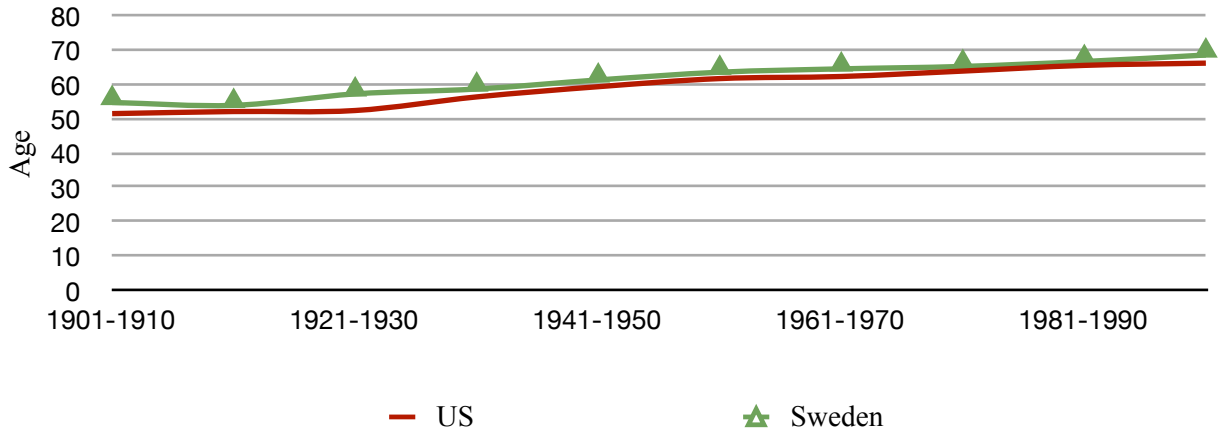

Figure 1. Comparing Swedish and the USA life expectancy for a 10 year old person, during 1901-1990 (Data from SCB.se and demog.berkeley.edu).

In figure 2 below we can see the results of our life table calculations where we have transformed the mortality data for diabetic subjects into life expectancy data (according to appendix 3). It is evident that the life expectancy for diabetic subjects has increased significantly since 1922, when insulin treatment was launched. The green (light) staples show the development for the average Swedish population for the same periods which, as we can see, has also increased although at a much slower rate.

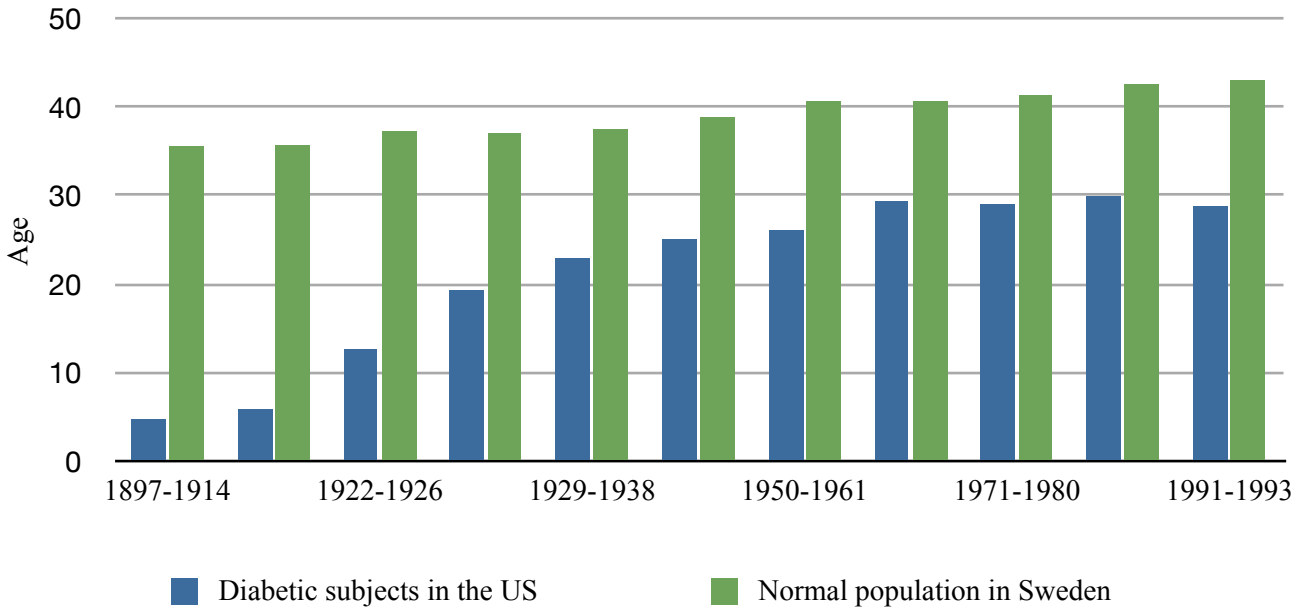

Figure 2. Mean life expectancy for the normal population and diabetic subjects (Data from Mark, 1964, SCB.se and vital statistics of the US).

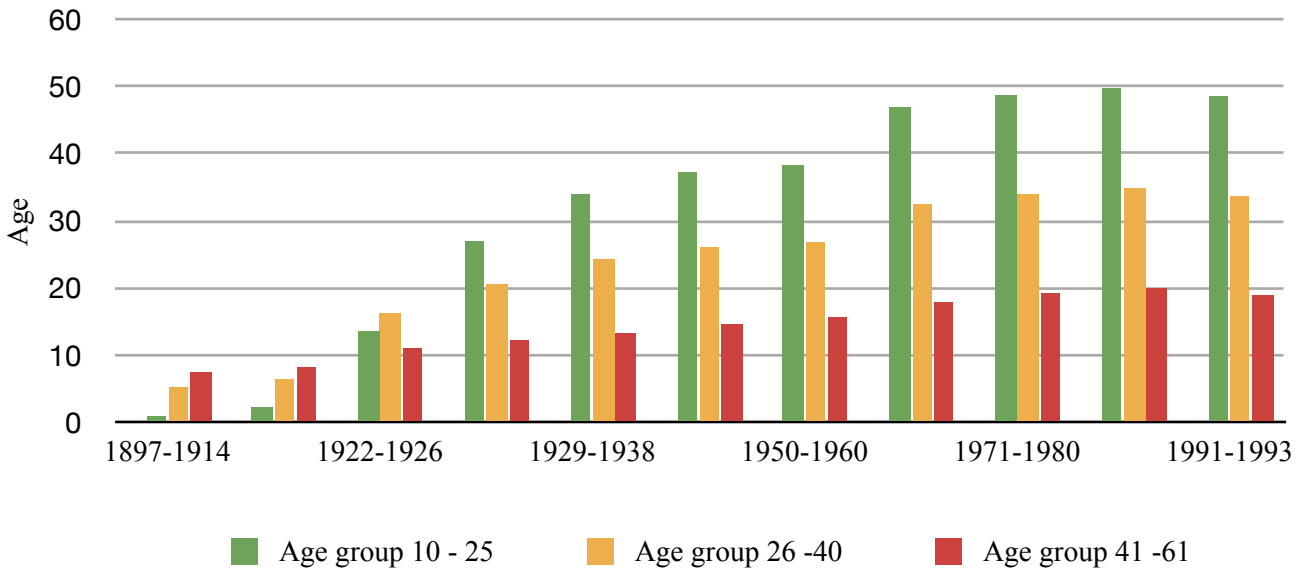

Figure 3. Mean life expectancy for the diabetes population in separated age groups (Data from Marks, 1964 and Vital Statistics of the US).

The life table calculations also provide us with information about the gains in life expectancy for different age groups. We can see that the age group that gained the most in terms of extended life expectation is the youngest age group 10-25. Applying the results from other studies assuming that diabetic subjects under age 40 are IDDM (type 1 diabetics), our result suggests that a large part of the increased longevity could be credited to insulin treatment. In figure 4 we show the changes in life expectancy for the youngest age included in our study, a 10 year old, for the entire period.

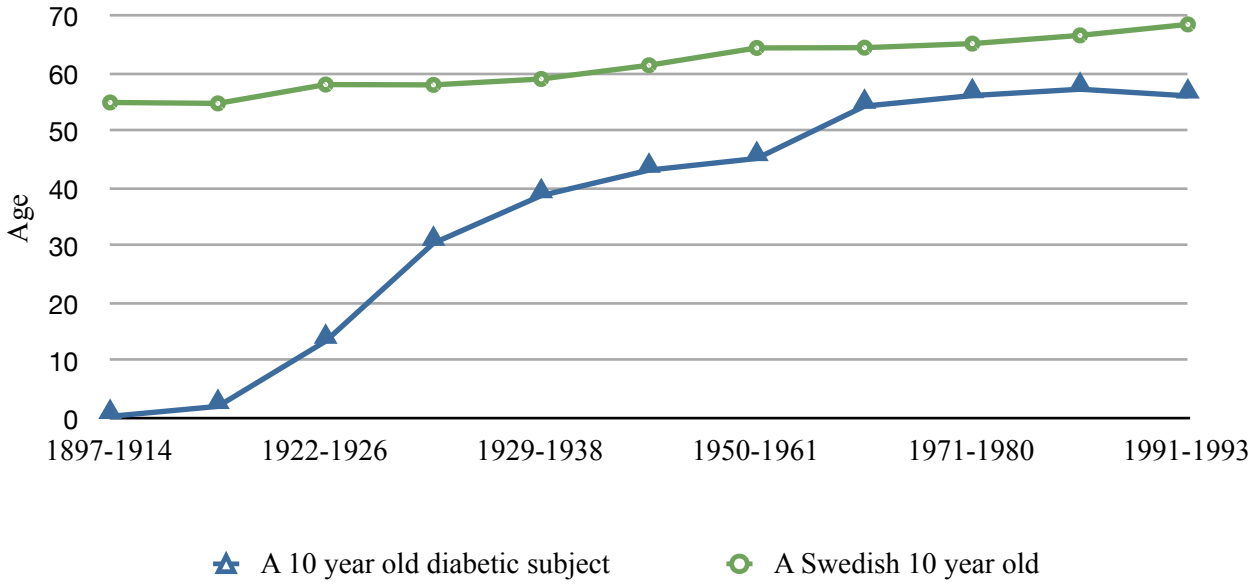

Figure 4. Life expectancy for a 10 year old diabetic subject compared to the life expectancy for a 10 year old average Swedish person (Data from Mark, 1964, SCB.se and vital statistics of the US).

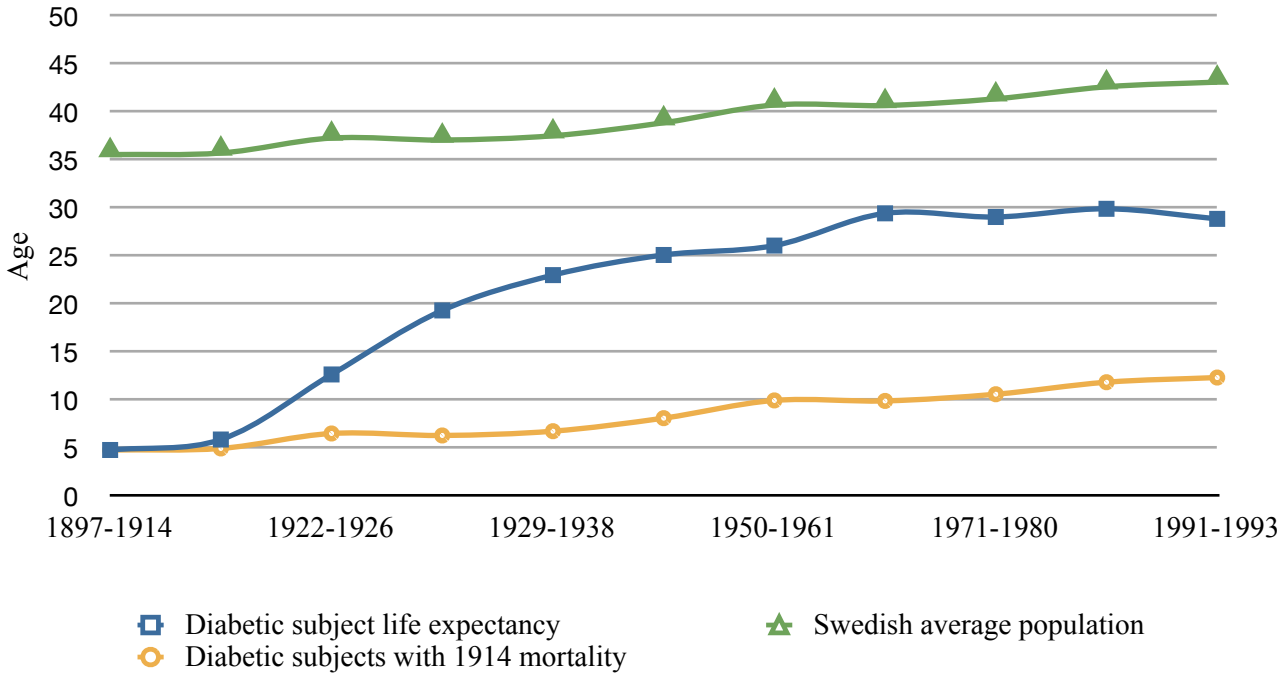

Figure 5. Comparing the actual life expectancy development for diabetic subjects, to a hypothetical development where the relative difference at 1914 is held constant (Data from Mark, 1964, SCB.se and vital statistics of the US).

In figure 5, we can see the life expectancy for the Swedish population compared to the life expectancy for diabetic subjects. The green line (triangles) shows the life expectancy development for the average Swedish population. The blue line (squares) shows the life expectancy development for diabetic subjects. The yellow line (circles) at the bottom shows how the life expectancy development for diabetic subjects would have been if the relative difference between the green line and the blue line in year 1914 would have stayed constant. The difference between the yellow (circles) line and the blue (squares) line can be interpreted as the effect of improved treatment, where we believe insulin has played a big role.

Time periods	Mean remaining life years for diabetic subjects	Mean remaining life years for the average Swedish population	Difference between diabetic - Swedish population
1897-1914	4,7	35,5	30,8
1914-1922	5,8	35,7	29,8
1922-1926	12,6	37,2	24,6
1926-1929	19,3	37,0	17,7
1929-1938	22,9	37,5	14,5
1939-1947	25,0	38,8	13,8
1950-1961	26,0	40,7	14,6
1961-1970	29,4	40,6	11,2
1971-1980	29,0	41,3	12,3
1981-1990	29,8	42,6	12,7
1991-1993	28,8	43,0	14,2

Table 2. Mean results of remaining life years. (Data from Mark, 1964, SCB.se and vital statistics of the US).

In the period 1897 to 1914 our results show that a subject with diabetes only had 4,7 mean remaining life years compared to the average population that had 35,5 mean remaining life years. In the year 1993 the situation had clearly improved for diabetic subjects, who now had 28,8 mean remaining life years. The difference in remaining life for a diabetic subject in the period 1897-1914 until the period 1991-1993 is 24,1 years. Additionally, the gap in remaining life between the average population and diabetics have decreased from 30,8 years in the first period 1897-1914, and only 14,2 years in the period 1990-1993, which means that the gap have decreased with 16,6 years. Valuing this in monetary terms we use our calculations of the value of a life year (447 327 SEK). Multiplying this with 16,6 years we get 7 425 628 SEK. This can be interpreted as the WTP of the increased longevity in diabetes during this specific time period for one person. If a cure were to be discovered eliminating the whole gap this would be worth about 953 billion SEK based on an assumed prevalence of 150 000 diabetic subjects in 1993.

4.5 Discussion

With the use of mortality data on USA and Sweden we have calculated estimates of the gains from increased longevity for diabetics in the time interval 1914-1993. The calculations are based on some assumptions which may affect the results in different ways.

The first assumption is that the mortality data on diabetics from USA can be applied to represent the Swedish diabetic population. We base this on both the results of the comparison between the life expectancy for the US and Swedish population that showed resembling development, and the fact that insulin treatment became available at the same time in both countries. Although we compare two developed countries, it is important to keep in mind that the health care systems in USA and Sweden are different. Since Sweden has a public health care system with limited private options it is fairly reasonable to believe that the actual development of the decrease in mortality for diabetics in Sweden has been even better (Nishimura, 2001). This suggests that our results may be an underestimation of the Swedish situation, implying that the actual gains are even greater.

The second assumption is that the relative mortality for diabetics compared to the Swedish population was constant after the age of 60. This was necessary since the mortality data for diabetics extended only to age 60, and if we left the calculations without any figure after age 60 the results for the remaining life years for ages 10-60 became distorted.

Like Murphy and Topel, we do not include the gains from increased quality of life, basically because it is not measurable in the same way as reduced mortality. The improved treatment did not only increase the life expectancy, but it also reduced the risk for complications from the disease (www.diabetes.se). This can be assumed lead to increased quality of life. The basic thought of Murphy and Topel's model is that health consists of two parts, as they refer to as G(t) and H(t), and to get a proper measure of the gains in health the quality of life should be included. This suggests that the actual gains are even bigger. It is therefore important to keep in mind that the gains we have measured are solely for the increased longevity, and not the entire health.

In our calculations we assume that most of the increase in longevity for diabetics is a result of improved health care, which might not be entirely true. Just as Murphy and Topel point out there are other factors that can have had an impact, like improved environment, changed lifestyle, actions against smoking etcetera. In Siegler et al. (2003), a critiquing article on Murphy and Topel, they suggest in order to avoid this issue a disease that is almost entirely affected by improved medical advances could be chosen for analysis, like for example diabetes type 1. We agree that it would make the results more credible, since diabetes type 1 is almost not at all affected by external factors, but as many other researchers have pointed out it is not an easy task to distinguish between the two types (Panzram, 1987). If we had mortality data on only type 1 available, indeed it would be

interesting to compare mortality data on both types, to explore how much of the improvement in longevity in fact can be explained by the medical advances, primarily insulin.

Despite these limitations we find that our results provide an overall picture of the development that is of interest, and even if the mortality data for diabetics is not perfect its strength is that is covers the period before and after insulin was launched. In figure 2 it is clear that the development for diabetic subjects have been very positive during the time interval 1914-1993, with a rapid increasing life expectancy, compared to the more constant development for the average Swedish population. One important finding is that the age group that had the lowest life expectancy in the beginning of the 2000 century, had by far the best improvement during the years up to 1993 (figure 4). This is similar to other research results showing that the younger cohort, sometimes defined as IDDM-patients or type 1 patients had the highest mortality before insulin, and the launch of insulin had the largest impact on this group. This findings indirectly shows the impact of insulin, since the increase in life expectancy is evident after the launch in 1922, and the fact that the age group that were affected the most was the youngest.

In figure 5 we show how the life expectancy for a diabetic subject would be in year during the continuing time if the mortality relative to the average population had been constant since 1914. If we assume that most part of the actual increase stems from insulin, this gives a picture of how the situation would have been if insulin had not been launched. In all of the figures we can see that the mortality have decreased over the entire period, but with a diminishing rate. This is consistent with more recent studies on the mortality in diabetes, and the overall results show that the mortality for a diabetic patient is around 3 times higher than for an average person (Eliasson et al. 2008) (Weiderpass et al. 2001). One could hypothesise that the diminishing decrease in mortality in diabetes seen the latest decades also can be attributable to insulin. After the introduction of insulin, no such dramatic new medical treatment has been launched, but the technology of how the insulin is given has improved, as has the insulin itself.

4.5.1 Costs relative to gains

Diabetes Mellitus is a chronic disease and as mentioned earlier the diagnosis and treatment is a large and growing economic burden to many countries (Begunde, 2007). In our analysis we have focused only on the gains, in terms of increased longevity, but to get an overall picture on the economic net impact of the disease we need to look not only at the benefits but also at the costs.

A common way to measure costs in health care is *cost of illness* (COI) studies. Cost of illness studies measure the social burden of a specific disease taking both direct as well as indirect costs into account. Jönsson and Henrikssons (1998) cost of illness study done on diabetes in Sweden showed that the total costs were 5746 MSEK (1994) which could be divided in to 2455 MSEK for direct costs i.e. health care and hospitalization and the remaining 3291 MSEK for indirect costs i.e. loss of productivity and income (Hälso & sjukvårdsprogrammet för diabetes mellitus, 2002). Insulin accounted for the greater part of the drug costs. Expenditures for complications as well as for treatment when diabetes was not the main diagnosis were excluded in these figures which suggest that the total costs may be larger.

Another way to calculate the cost is by including all the relevant costs for persons suffering from diabetes, whether diabetes is the main diagnose or not. The upside is that everyone with diabetes is included although the results may then be overestimated. This type of analysis was done in a study from Malmöhus län, but where only the direct cost was measured. The results showed an average cost of 27 885 SEK/patient and year. This number was fairly constant for all ages, except for the youngest cohort, under the age of five, which had more than double the costs (Hälso- och sjukvårdsprogrammet för diabetes mellitus, 2002). This finding is constant with many other studies because the excess mortality for a diabetic patient is higher if the age of onset is low, which suggest that the disease is then more severe and requires more resources.

5. Conclusion

The purpose with this thesis has been to do a methodological analysis of different approaches to estimate the gains in longevity. We chose to analyse two methods thoroughly, both which have the focus on how new drugs effect longevity. The objectives and implementations vary in many aspects. Lichtenberg's econometric model is designed to evaluate the impact of new drugs on longevity by investigating the relationship between the number of new drugs and increased longevity. Lichtenberg has a top down perspective and uses data from many countries and many different drugs and disease groups in his analysis. Even if Lichtenberg divides the results into different disease groups it is not possible to extract the effect of one single drug, i.e. insulin. Since we want to analyse the impact of one drug on one disease in one country, our perspective is different from Lichtenberg's, and we find that the econometric approach is hard to use for our smaller perspective. It is not impossible to redesign the econometric model to fit our purpose better, but to get reliable results we would need large amount of data which is hard to find for diabetes from early 1900.

Murphy and Topel use a bottom up approach to estimate the value of increased longevity. The strength of this method is that it can be used for any individual or population at interest. The method also provides results in monetary value, since it measures the willingness to pay for the increased longevity, which is different from Lichtenberg's method. Based on the data at hand, we find that making a similar calculation to Murphy and Topel's would provide us with more reliable and relevant results then an econometric model would.

In our empirical analysis we started off by transforming the mortality data on diabetics to remaining life years for different ages. This was with the intention to get a measure on the additional life years due to decreased mortality in diabetes, like Murphy and Topel presented for various diseases over time. Despite some necessary assumptions, we find that the results are reasonable and consistent to other studies.

Our results confirm our hypothesis that there has been an increase in longevity for diabetics during 1914-1993. We cannot statistically confirm the underlying cause for this increase, although a lot is implying that insulin has played an important part. First, we see a dramatic effect short after the launch of insulin in 1922. Second, the effect was the largest for the youngest cohort which is presumably type 1, insulin depended patients. These facts together suggest that a large part of the

increase in longevity can be attributable to insulin. Needless to say insulin has played a vital role in the lives of diabetic patients. The results also suggest, as did Murphy and Topel's analysis, that the gains from increased longevity are tremendous, indicating that although the cost and efforts put into research and development make up a substantial part of countries GDP, they are well worth it.

For further analysis it would be interesting to do a similar analysis specifically for type 1 diabetes subjects since external factors like improved lifestyle do not have a big impact on the mortality. It would therefore be easier to isolate the effect of insulin. Finally, it would then be interesting to compare these gains with more specific expenditure data on insulin to make some kind of cost effectiveness analyses.

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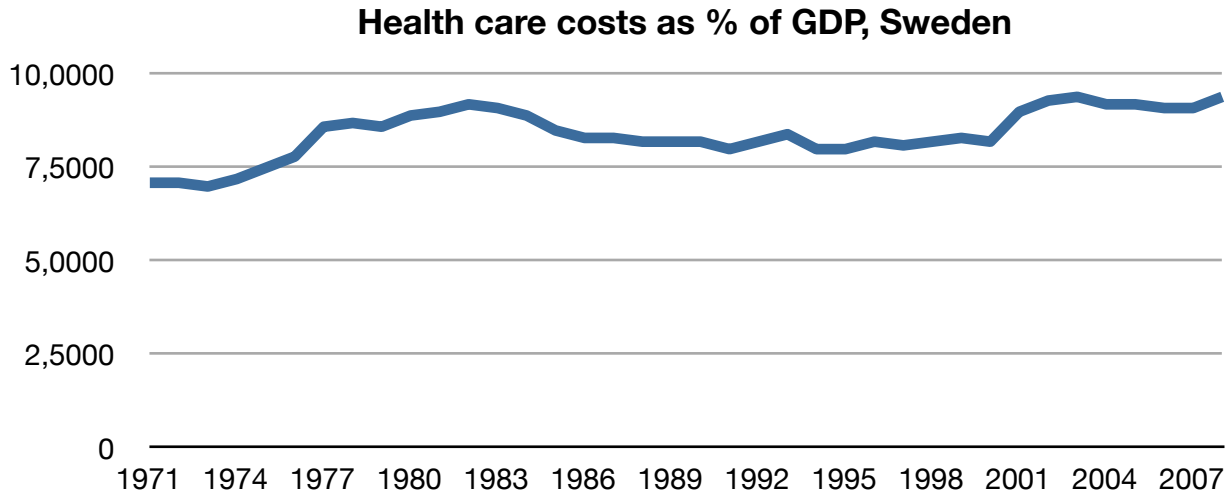
7. Appendix

7.1 Health care costs



(Data from WHO)

7.2 Mortality data

Table 1—Mortality Among Diabetic Patients at Specified Ages in Successive Periods, 1897-1961: Experience of Joslin Clinic, Boston, Mass.

	Death Rate per 1,000						
	Naunyn Era	Allen Era	Insulin Era				
Age	1897–1914	1914–1922	1922–1926	1926–1929	1929–1938	1939–1947	1950–1961
10	824.0	386.1	61.4	19.1	8.1	3.3	1.0
20	614.0	410.8	89.4	18.3	12.6	7.9	3.4
30	359.8	236.8	74.8	33.4	13.9	11.3	14.4
40	165.7	115.1	34.7	23.8	16.6	13.6	15.3
50	96.1	77.4	45.3	41.0	30.6	26.7	21.5
60	88.8	112.5	85.2	70.1	66.6	51.6	43.0

NOTE: Except for 1950-1961 rates are graduated from life table computations; in all groups deaths within one week of first observation or hospital discharge are excluded.

(Marks, 1965)

7.3 Life table calculations

q_x = Age specific mortality risk

p_x = Survival probability ($1- q_x$)

l_x = Number of individuals surviving until time x. $l_{x+1} = l_x \text{ X } p_x$ ($l_0 = 100\ 000$)

d_x = Number of individuals who died at age x. dx

L_x = Person years lived between x & x+1.

T_x = Person years lived above age x. $T_x = \sum^{\infty}_{a=x} L_a$

e_x = Life expectancy at age x. T_x/l_x

(Kirk Scott, Associate Professor at the Department of Economic Demogaphy at Lund University)