The Impact of Aging on Future Healthcare Expenditure

Lukas Steinmann, Harry Telser, and Peter Zweifel

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Abstract

The impact of aging on healthcare expenditure (HCE) has been at the center of a prolonged debate. This paper purports to shed light on several issues. First, it presents new evidence on the relative importance of the two components of HCE that have been distinguished by Zweifel, Felder and Meier (1999), viz. the cost of morbidity and the cost of mortality (their ‘red herring’ hypothesis claims that neglecting the mortality component results in excessive estimates of future growth of HCE). Second, it takes account of recent evidence suggesting that HCE does increase life expectancy, implying that time-to-death is an endogenous determinant of HCE. Third, it investigates the contribution of population aging to the future growth of HCE. For the case of Switzerland, it finds this contribution to be relatively small regardless of whether or not the cost of dying is accounted for, thus qualifying the ‘red herring’ hypothesis.

Keywords: Health econometrics, Aging, Cost of dying, Healthcare expenditure

JEL Classification: J14, I12.

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1 Introduction

Economic and technical progress in the industrialized world has enhanced both standards of living and effectiveness of health care, resulting in an enormous increase of life expectancy. In addition, fertility rates have been dropping since the end of the so-called baby-boom in the late 1960s. These two trends in combination cause a marked aging of population, i.e. the share of the elderly will increase dramatically in the two decades to come.

Since there is a strong cross-section relationship between age and per capita healthcare expenditure (henceforth $HCE$), aging populations are often predicted to have increasing costs of health.

However, the macroeconomic evidence is ambiguous. Only two macroeconometric cross-national studies found the age structure of the population in developed countries to be a consistently significant regressor [Hitiris and Posnett (1992) and Gerdtham, Søgaard, Jönsson and Andersson (1992b)]. The others found no significant relationship between age and per capita $HCE$ [cf. Gerdtham, Søgaard, Andersson and Jönsson (1992a); Gerdtham, Jönsson, MacFarlan and Oxley (1998); Getzen (1992); Leu (1986), O’Connell (1996); OECD (1987); Zweifel, Steinmann and Eugster (2005)].

One explanation that has been advanced is that the cross-section relationship between $HCE$ and age fails to control for an important variable that has varied in the past and may vary in the future. Indeed, much of the increase of average $HCE$ with age may be due to a changing composition of the insured population. At higher ages, the share of those approaching death rises substantially. But according to several studies, proximity to death goes along with a marked surge in $HCE$. Lubitz and Riley (1993) estimate that the 5.1 percent of Medicare recipients dying in a given year account for no less than 29 percent of total $HCE$. Similar findings were reported for long-term care [Stooker, van Acht and van Barneveld (2001)]. Therefore, in a population containing a larger share of individuals close to death, average $HCE$ must increase with age.

Now at a given point in time, higher age and proximity to death are the same. In the course of time, however, this need not to be true. For example, people on average are in their last year of life at age 75, while 20 years from now, they may be one year from death at age 85. Thus, they would simply enter the costly phase of their lives ten years later. Calculated on a lifetime basis, $HCE$ of future cohorts with higher life expectancy may even be lower than present—population aging would result in a lower present...
value of $HCE$!

In order to test this for possibility, three concepts of time need to be distinguished. Historical time (the year of observation) reflects the state of medical technology; time from birth (the age of the individual) stands for the true effect of age; and time to death ($TTD$) is the determinant of $HCE$ that can only be identified with independent information about remaining life expectancy. Using panel data on deceased members of two Swiss social health insurers covering the years 1983 to 1994, Zweifel et al. (1999) were the first to be able to separate these three concepts of time. Relating quarterly $HCE$ to sex, type of insurance, age, proximity to death, and year of observation, they found age not to be significant in determining $HCE$ during the last two years of life, at least for the population aged 65 and above. On the other hand, $TTD$ was highly significant, with its estimated effect consistently increasing in closeness to death. In a second analysis covering the last five years of life, these findings were confirmed. This led ZFM to argue that aging will not affect $HCE$ growth when controlling for proximity to death. This conclusion stands in stark contrast to naive predictions using raw $HCE$ profiles from descriptive statistics. ZFM dubbed aging a ‘red herring’, pointing instead to changes in medical technology as the main driver of cost over time.

The ZFM argument has given rise to several conceptual and methodological issues [cf. Salas and Raftery (2001), Dow and Norton (2002), Seshamani and Gray (2004a), Seshamani and Gray (2004b), and Stearns and Norton (2004)]. Zweifel, Felder and Werblow (2004) addressed these issues with a more sophisticated econometric analysis that also included survivors. They concluded that the ZFM argument still stands.

In this paper we return to the basic ZFM argument. Is it really necessary to distinguish between the cost of dying and ‘normal’ $HCE$ for forecasting future $HCE$? At the same time, the following four issues will be addressed.

1. With increasing life expectancy, the cost of dying will accrue at a higher age. Thus, naive forecasts of $HCE$ that fail to consider this shift have an upward bias, simply because the share of deathbound at a given age will be smaller than today. However, the cost of dying itself may increase even faster than ‘normal’ $HCE$, thus counterbalancing this upward bias.

2. Recent research into the so-called Sisyphus syndrome [cf. Zweifel and Ferrari (1992); Zweifel et al. (2005); see also Frech and Miller (1999)] has found that $HCE$ does positively affect remaining life expectancy, in
particular at age 60. This implies that TTD is an endogenous variable, a fact that only Zweifel et al. (2004) tried to deal with by replacing a series of time dummies by one single continuous variable TTD. While this solution serves to mitigate the endogeneity problem of TTD, it comes at a price because the influence of historical time (and hence medical technology) on HCE cannot be estimated anymore.

3. Estimation based on a sample of dying and surviving individuals runs the risk of underestimating the influence of TTD on HCE. This is because information about TTD is only available for those individuals who died within the observation period. For those to die after the end of the observation period, the indicator ”one year to death” (death\(_1\), see below) typically used in existing studies is not defined. Deleting the last year of observation for those who did not die solves the problem. Deleting the last two years ensures that ”two years to death” (death\(_2\)) is defined as well.

4. There are two important econometric issues. First, a substantial number of individuals have zero HCE. This calls either for sample selection modeling [typically of the Heckit variety as in ZFM, with the risk of incurring identification problems, as argued by Salas and Raftery (2001)]. The alternative, pursued below, is to specify a two-part-model. In the first part, the probability of nonzero HCE is estimated. In the second part, the amount of HCE is estimated for all individuals with positive HCE. Second, HCE values usually are heavily skewed. Transforming the data is the common procedure for handling this problem. However, this entails the difficulty of retransformation after estimation. The solution proposed here is to estimate a generalized linear model.

The remainder of this paper is organized as follows: in section 2 the data and the econometric method applied are presented. In section 3 the results are discussed altogether with the corresponding age profile of HCE. In section 4 the effect of aging on HCE is discussed. Section 5 contains concluding remarks.

## 2 Data and econometric model

The present study is based on a data set comprising about 450,000 Swiss individuals from a major sickness fund. Spanning the years 1997 through
2004, it results in a sample of more than 3,700,000 observations.

In Switzerland health insurance is mandatory. Premiums are not risk-based, but must be uniform for all adults of a given insurer with residence in a region. There is a risk adjustment scheme designed to prevent risk selection by competing health insurers. The minimum annual deductible is CHF 230 (starting 2004: CHF 300; 1 Swiss franc CHF=0.8 US$ at 2004 exchange rates), combined with a copayment of 10 percent of \( HCE \) (capped at CHF 700). Thus, total out-of-pocket payments by an individual with a standard contract is limited to a maximum of CHF 930 per year (effective 2004: CHF 1,000), or some 1.5 percent of average income. Additionally, the insured may choose deductibles of up to CHF 1,500. Managed-care contracts, viz. health maintenance organizations (HMO), preferred provider organizations (PPO), and physician networks are also available.

There is a vivid debate about the effect of such non-conventional contacts. It revolves about the issue of whether the substantial cost savings are achieved due to risk selection or due to innovations in the guise of changed incentives. A recent study concludes that between one-third (physician networks) and two-thirds (HMO) of cost savings cannot be traced to risk selection effects and therefore may reflect true innovation [Lehmann and Zweifel (2004)]. Therefore, the subsamples with higher deductibles and managed-care alternatives may be characterized by self-selection. In order to avoid modeling the pertinent selection mechanisms, all observations having deductibles in excess of CHF 230 or a managed-care option were deleted from the sample.

As argued in the preceding section, \( TTD \) is only defined for those individuals who died within the observation period. Since the indicators ”one year to death” \( (death_1) \) and ”two years to death” \( (death_2) \) are to be used, the observations of 2003 and 2004 need to be dropped for all individuals who did not die by the end of 2004.

These deletions left 1,528,949 observations for estimation. On average, an individual has 4.9 observations, causing the data set to be of the panel type. This calls for a random-effects specification for two reasons. First, it is important to control for unobserved heterogeneity caused by unobserved health, which may be poorly approximated by the socioeconomic variables available. Second, cohort and age effects can be disentangled in this way.

There is a significant number of observations whose \( HCE \) does not exceed the deductible. These individuals paid out of pocket or were not sick at all. Accordingly, the first step of the two-part model was estimated using the
following random-effects probit\footnote{Choice of the appropriate model in this context is discussed in Seshamani and Gray (2004a); Dow and Norton (2002); or Jones (2000).}

\[ Pr(HCE_{i,t} > \text{Deductible}) = \Phi\{\alpha + \beta X_{i,t} + v_i + \epsilon_{i,t}\} \]  \hspace{1cm} (1)

where \( X_i \) contains \( TTD \) (represented by three variables \( death, death_1, \) \( death_2 \) indicating whether individual is 0, 1, 2 years away from death), \( age \) (up to cubic), a dummy variable \( sexf \) for women, and interaction terms involving \( age \), and \( sexf \), plus a set of dummy variables controlling for regional effects. This was necessary because morbidity and the level of \( HCE \) differ considerably between regions. \( v_i \) is the random-effect for insured individual \( i \), and \( \epsilon_{i,t} \) is the iid residual of individual \( i \) in year \( t \). Moreover, all \( TTD \) variables are interacted with \( age \), \( age^2 \), \( sexf \), \( sexfage \), and \( sexfage^2 \) to allow for more flexibility in modeling the cost of dying.

As noted in the preceding section, \( TTD \) likely depends on \( HCE \). For ensuring identification, \( death_j \) \((j = 0, 1, 2) \) is related to \( HCE \) of the previous year \( (HCE_{-1}) \), \( sexf, age, age^2 \), and interaction terms (with regional dummies excluded on the premise that living conditions and medical technology are homogenous across the cantons of Switzerland). One therefore has

\[ death_{i,t} = \Phi\{\kappa + \lambda Z_{i,t} + \varphi_i + \mu_{i,t}\} \]  \hspace{1cm} (2)

with \( Z_{i,t} \) containing \( sexf, age, age^2, sexfage, HCE_{-1} \), and its interaction with age of individual \( i \). The predicted values \( \hat{death}_{i,t} \) enter equations 1 and 3.

In the second step of the two-part model, the amount of \( HCE | HCE > \text{Deductible} \) was estimated using again a random-effects specification and the same set of regressors as in the first part,

\[ HCE_{i,t} | HCE_{i,t} > \text{Deductible} = \alpha + \gamma X_{i,t} + v_i + \epsilon_{i,t}. \]  \hspace{1cm} (3)

Since \( HCE \) is skewed heavily, estimated parameters \( (\alpha, \beta) \) may not be robust; moreover, significance tests based on the normality assumption are inadequate. A Box-Cox test indicated that a log transformation results in the best approximation to normality. However, standard estimation procedures predict \( E(log(y)|x) \) rather than \( log(E(y)|x) \), calling for a difficult retransformation after estimation.\footnote{Duan’s smearing procedure to retransform the predicted values may be a solution, but has desirable statistical properties only in absence of heteroscedasticity.} The alternative is to estimate a generalized linear
model with a log-link. The relationship between the means and variances of $HCE$ and the regressors suggests using the gamma distribution for the error term [i.e. $V(y|x)$ is proportional to $(E(y|x))^2$; cf. Manning and Mullahy (2001)].

3 Estimation results

Starting with the auxiliary equation 2, there is a significant positive relationship between death and age, sex, $HCE_{-1}$ and its interaction with age (not reported here). However, predictive power is very low: for the younger individuals, too few deaths are predicted while for the elderly the contrary is true. This may be expected since younger individuals more often die due to accidents and suicides. This means they have low $HCE$ in the year preceding death. Thus, only the few young individuals with (very) high $HCE_{-1}$ are predicted to be in the last years of their life while the rest of those about to die are predicted to survive (type II error). The elderly on the other hand have a relatively high probability of being in their last years of life. Moreover, they rarely die without having incurred considerable $HCE_{-1}$, which is positively related to the probability of being in their last years of life. Thus, among the elderly, equation 2 results in too many predicted deaths (type I error).

By trying to solve the endogeneity problem, one thus introduces error. Obviously, death at young age is strongly influenced by unobserved factors, such as bad luck or a lack of prudence rather than by $HCE$. Since among the young, only individuals with very high $HCE$ are predicted to be in the last year of their life, estimation of $HCE$ leads to exorbitant values for the young and values too close to zero for those at the top end of the age distribution. Thus, it seemed preferable to use observed rather than predicted values of death $j$ ($j = 0, 1, 2$) in equations 1 and 3 in spite of the existence of an endogeneity problem.

Turning to now the probit estimate of equation 1 (the first three columns in table I), one notes the extremely large $z$ values of age, $age^2$, $age^3$, and their interaction terms with sexf. On the other hand, death, death$_1$, and death$_2$ and their interaction terms often fall short of standard significance levels, likely because of multicollinearity with age-related regressors. However, they are jointly significant, as indicated by a series of Wald tests. In figure 1, the estimated relationship between age and the probability of having $HCE > 7$
Figure 1: Probability of having $HCE > Deductible$

$Deductible$ is plotted for both genders. As expected, this probability declines with age, reaches a minimum (at age 25 for women and 30 for men), and increases again. At ages beyond 75, both women and men are very likely to have $HCE > Deductible$.

When looking at the estimation results for the second part of the model (equation [3], the last three columns in table [1]), one first notices that again most regressors involving age have high $z$ values. However, several regressors fall short of conventional significance levels. This may have two different reasons. First, there indeed may not be a relationship between the regressor and $HCE$. For example, this seems to be the case for $age^3$. A regression omitting this particular variable indicated that the parameters of $age$ and $age^2$ remained stable. Thus, $age^3$ indeed does not influence $HCE$ of men. A second reason for lack of significance might be multicollinearity. While
Table 1: Estimation results

<table>
<thead>
<tr>
<th>Variable</th>
<th>First part (equation 1)</th>
<th>Second part(^a) (equation 3)</th>
<th>z</th>
<th>Coeff</th>
<th>SE</th>
<th>z</th>
<th>Coeff</th>
<th>SE(^b)</th>
<th>z</th>
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<td>-2.61</td>
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<td>0.0023</td>
<td>15.33</td>
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<td>0.0001</td>
<td>-2.39</td>
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Observations 1,528,949 1,195,988
Groups 310,348 290,712
Log-Likelihood: -624,200 AIC: 17.34

\(^a\) Estimation using the Newton-Raphson algorithm.
\(^b\) Estimation using the Huber-White sandwich estimator.
multicollinearity does not result in biased parameters, it serves to strongly increase estimated standard errors. Thus, in the presence of multicollinearity, the probability of type II error looms large. For example, omitting \( age^3 \) caused the parameter of \( age^2 \) to have much higher \( z \) value than the one shown in table 1.

This finding is confirmed by a Wald test\(^3\) which indicated that the coefficients pertaining to three the age-related regressors jointly are not significantly different from the first two \( [H_0 : \beta_{age} + \beta_{age^2} + \beta_{age^3} = \beta_{age} + \beta_{age^2} \text{ need not be rejected}, \chi^2(1) = 0.09, Prob > \chi^2 = 0.7634] \). Since the coefficients of both the the first two and also of all three age-related terms are jointly different from zero, a quadratic specification for estimating the relationship between age and \( HCE \) for men is sufficient.

For women, however, the cubic specification seems to be more adequate since \( H_0 : \beta_{sex\cdot age} + \beta_{sex\cdot age^2} + \beta_{sex\cdot age^3} = \beta_{sex\cdot age} + \beta_{sex\cdot age^2} \text{ is rejected with very low error probability} \ [\chi^2(1) = 248.89, Prob > \chi^2 = 0.0000] \).

With respect to death, for men, most regressors including interactions with age-related variables are significantly different from zero. Moreover, the significance of these regressors differs between men and women \( [i.e. H_0 : \beta_{death} + \beta_{death\cdot age} + \beta_{death\cdot age^2} = \beta_{death\cdot sex} + \beta_{death\cdot sex\cdot age} + \beta_{death\cdot sex\cdot age^2} \text{ may be rejected for all } t = 0, 1, 2 \text{ with } P < 0.008] \), justifying the specification presented in table 1 which allows for gender-specific \( HCE \) patterns.

The estimation results of table 1 may now be used to compute expected healthcare expenditure (\( \hat{HCE} \)) as shown in equation 4 below. The first term on the right hand side denotes expected morbidity cost (\( C_{MORB} \)), which is obtained by weighting estimated \( HCE \) \( \text{[cf. equation 3]} \) with estimated probability \( \text{[cf. equation 1]} \). The cost of dying (\( COD \)), being controlled for in the estimation, must be considered separately. This is done in the second term (\( C_{MORT} \)), which combines \( COD \) as estimated in equation 3 and mortality rates \( Pr(M) \) (observed values for the year 2000, forecasts for 2001 to 2060),

\(^3\)Wald tests may not be appropriate in GLM estimations of the non-Gaussian type because of the so-called Hauck-Donner phenomenon \([\text{cf. Hauck and Donner (1977)}]\). However, using the Huber-White sandwich variance estimator \( \text{as in table 1} \) helps to overcome the Hauck-Donner phenomenon, thus restoring appropriateness of the Wald test \([\text{cf. Statalist 2003]}\).
The age profiles for $C_{MORB}$ are plotted in figure 2. On the whole, $C_{MORB}$ (solid lines) increase with age for both sexes. For women, $C_{MORB}$ in general runs higher and increases more steadily, while for men up to age 30, there seems to be no relationship between age and $C_{MORB}$. At ages beyond 80, women (but not men) exhibit exponentially increasing $C_{MORB}$. This important difference was detected only based on the cubic specification, which allows for more flexibility.
As to $C_{MORT}$, only the $COD$ component is shown in figure 2. $Pr(MORT)$ is known to increase exponentially beyond age 60. Interestingly, the $COD$ curves tend to be inversely U-shaped, with a peak at age 51 for men and at age 41 for women, amounting to approximately CHF 21,000 for women and CHF 14,000 for men, respectively. Finally, $C_{MORB}$ and $COD$ can be compared. As has been known in the literature [Lubitz and Riley (1993); Zweifel et al. (1999)], the cost of dying ($COD$) is a several multiples of $C_{MORB}$ at most ages. However, since $COD$ declines past ages 41 and 51, it falls below $C_{MORB}$ at very high ages (93 for men, 89 for women). This means that the factor of proportionality is not a constant, but varies with age, an observation also made by Zweifel et al. (2004).

These findings are consistent with economic theory, which predicts that the relationship between the value of a statistical life and age is inversely U-shaped, with a maximum value around the age of forty [Schelling (1968) and Shepard and Zeckhauser (1982)]. Therefore, willingness to pay for survival (which is reflected in $COD$ if physicians act as reasonably good agents of their patients) should exhibit the same pattern. This prediction has been empirically confirmed by several authors [cf. Carthy, Chilton and Cookson (1999), Jones-Lee, Hammerton and Philips (1985), Mount, Weng and Schulze (2000)].

Estimated $C_{MORB}$ and $C_{MORT}$ sum up to $\hat{HCE}$. Comparison of $\hat{HCE}$ and country-wide age-specific $HCE$ published by the Swiss risk adjustment fund may serve as a check on the external validity of the two-part-model estimated. As can be seen from figure 3, the profiles match quite well.

4 What impact of aging on future health care expenditure?

Zweifel et al. (1999) claimed that the impact of aging on future $HCE$ is overestimated. Since then, a consensus has evolved that time to death ($TTD$) must be controlled for when estimating $HCE$ profiles designed to serve as a basis for prediction of the effect of aging on $HCE$. However, controlling for $TTD$ (and hence cost of dying $C_{MORT}$) in estimation requires that $C_{MORT}$ is included in forecasts of $HCE$ (as done in equation 4). Mortality will increase in the future because the baby-boomers will be coming of age. This trend
Figure 3: Country-wide and estimated age profiles of HCE (2000)
is illustrated for the case of Switzerland, which may be seen as a typical developed country except for its above-average high life expectancy. Also, Switzerland is aging faster than the United States due to a lower fertility, but more slowly than some other European countries (such as Germany or Italy). As can be seen in figure 4, Swiss mortality rates decreased (or at least were stable) until the year 2000. They are predicted to increase from below 900 per 100,000 at present to more than 1,200 by 2050. Later, when the last baby-boomers will have died, mortality rates will decline again.

Integrating the cost of dying into forecasts of HCE therefore causes a surge in future HCE that was not accounted for by ZFM, who implicitly
assumed the mortality rate to remain constant. The relative increase in 
HCE (A) due to the aging of population is defined as

\[ A = \frac{\overline{HCE}_t}{\overline{HCE}_{2000}} , t = 2000, ..., 2060 \tag{5} \]

Now equation 5 needs to be adjusted for changes in the future cohort 
structure of the population, yielding

\[ \overline{HCE}_t = \frac{1}{P_t} \sum_{c=1}^{n} p_{c,t} \ast \overline{C_{MORB_c,t}} + \frac{1}{P_t} \sum_{c=1}^{n} d_{c,t} \ast \overline{C_{MORT_c,t}} \tag{6} \]

\[ P_t = \sum_{i=1}^{n} p_{c,t}. \]

Here, \( P_t \) is the number of individuals alive in future period \( t \), composed 
of the \( n \) cohorts comprising \( p_{c,t} \) people, of which \( d_{c,t} \) die in period \( t \). \( C_{MORB} \)
denotes the predicted per capita cost of morbidity, and \( \overline{C_{MORT}} \) stands for 
the cost associated with dying as estimated in equations 1 and 3.

Age-specific \( C_{MORB} \) and \( C_{MORT} \) are held constant (\( C_{MORB_c} = C_{MORB_{2000}} \) 
and \( C_{MORT_t} = C_{MORT_{2000}} \)), i.e. any future change in preferences and medical 
technology that might influence \( C_{MORB} \) and \( C_{MORT} \) are assumed away in 
order to filter out the pure aging effect.

The degree to which the distinction between the cost of morbidity and 
mortality affects the magnitude of the aging effect depends on the age-specific 
cost of dying. If \( COD \) were low at higher ages, then the development of total 
HCE would follow closely that of \( C_{MORB} \). Thus, the naïve forecast and the 
ZFM approach would converge since \( C_{MORT} \) is largely irrelevant. If, however, 
\( COD \) were highest at high ages, causing it to increase strongly with a future 
rise in mortality rates, predicted HCE will diverge more markedly from an 
extrapolation that is based on \( C_{MORB} \) only, neglecting the mortality cost 
\( C_{MORT} \). Moreover, age groups characterized by a high rate of mortality will 
grow in size in the future. This constitutes an additional effect not taken 
into account by ZFM, who limited their analysis to a period of 10 years—too 
short to recognize such shifts in population structure.

In figure 5, the predicted development of HCE is plotted (estimated 
according to equations 1 and 3 and computed using equation 6). In contrast 
to the official forecasts prepared by the Swiss Federal Office of Statistics,
lower mortality rates at higher ages are applied [scenario W, cf. Münz and Ulrich (2001)], causing the effect of aging on $HCE$ to be more pronounced.

The naïve forecast (i.e. without controlling for $C_{MORT}$) results in an increase in $HCE$ from about CHF 2,200 up to CHF 3,000 by 2060. Controlling the cost for mortality leads to predicted $HCE$ reaching CHF 2,700 by 2060. Therefore, the effect of aging on $HCE$ is relatively small in both scenarios, especially if compared with an extrapolation based on the 5 percent growth rate of the period 1996-2003, which would have $HCE$ increase to CHF 41,000 (cf. trend 5% in figure 5). Only if future growth of Swiss $HCE$ were to be pushed down to 1 percent p.a. would the pure aging effect make a noticeable
difference. And in that case, taking the cost of dying into account becomes relevant.

Indeed, the naïve approach results in a contribution of aging to $HCE$ growth amounting to 0.51 percent p.a., while calculations filtering out the cost of dying (cf. equation 4) yield 0.37 percent. This average difference is subject to considerable variation over time, however. As evidenced in figure 5, it will increase strongly until 2009, with a maximum of 0.24 percentage points ($= 0.86 - 0.62$).

Beyond 2032, the distinction between the costs of morbidity and mortality matters less and less, with the difference in growth rates falling from 0.24 percentage points in 2031 to zero by the year 2057. The reason for this reduction is that most baby-boomers will have died by mid 2050.
On the whole, these findings serve to qualify the conclusion reached by Zweifel et al. (2004) who in their re-examination of ZFM state that the "naïve estimation that does not control for proximity to death will grossly overestimate the effect of population ageing on aggregate health care expenditure". In the case of Switzerland at least, this statement holds true only if future HCE growth is markedly below the values experienced in the recent past.

5 Conclusions

Aging will not contribute much to future growth of per capita healthcare expenditure (HCE). When computing the naïve aging effect, i.e. neglecting the distinction between morbidity and mortality cost as proposed by Zweifel et al. (1999), HCE growth in Switzerland will be around 0.5 percent per annum over the next 55 years. If proximity to death is controlled for when estimating age profiles of HCE and taken into account when forecasting the effect of aging, predicted growth falls to 0.37 percent per annum. The reason for this rather small decrease is that mortality rates will increase dramatically in the coming decades, when the baby-boomers reach higher ages and eventually die. Thus, the downward adjustment of the aging effect in future HCE requested by ZFM is partly offset by an increasing cost of mortality. This finding proves quite robust with respect to different specifications at least in the case of Switzerland (whose experience however is representative of ‘mature’ industrial countries). Thus, while the ZFM approach leads to a minimum aging effect, it cannot substantially modify a naïve extrapolation. According to Beck (2004), Swiss HCE increased by 5.5 percent p.a. between 1992 and 1999, to which aging contributed a mere 0.6 percentage points, vindicating ZFM.

However, there are reasons to believe that aging will indirectly increase HCE. It is quite possible that HCE growth for the elderly will be faster than for the younger, i.e. that age profiles will become steeper in the future, causing the aging effect to increase. In their international comparison, Seshamani and Gray (2002) report mixed evidence, age-specific growth of HCE being linked to the type of healthcare system. Since aging effects turn out to be relatively less important in the in England and Wales with its National Health Service, this difference may be interpreted as reflecting
age-based rationing not imposed by insurance-based systems. This leads to the conclusion that not aging but rather institutional characteristics cause differences in age-specific growth of $HCE$.

There is one additional channel of influence that may affect the age gradient of $HCE$. It is the so-called Sisyphus syndrome which claims that aging shifts voting power to the elderly who in turn seek—by democratic means—to allocate more and more resources to (public) healthcare services [cf. Zweifel and Ferrari (1992) and Zweifel et al. (2005)]. This means that the young members of society will increasingly have to carry the burden of aging. Along with the additional burden of having to finance old-age pensions for beneficiaries whose remaining life expectancy after retirement continues to increase, this may well become a major challenge for aging societies.
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