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Health expenditure growth: Looking beyond the average through decomposition of the full distribution

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Abstract (150 words)

Explanations of growth in health expenditures have restricted attention to the mean. We explain change throughout the distribution of expenditures, providing insight into how growth and its explanation differ along the distribution. We analyse Dutch data on actual health expenditures linked to hospital discharge and mortality registers. Full distribution decomposition delivers findings that would be overlooked by examination of changes in the mean alone. The growth in expenditures on hospital care is strongest at the middle of the distribution and is driven mainly by changes in the distributions of determinants. Pharmaceutical expenditures increase most at the top of the distribution and are mainly attributable to structural changes, including technological progress, making treatment of the highest cost cases even more expensive. Changes in hospital practice styles make the largest contribution of all determinants to increased spending not only on hospital care but also on pharmaceuticals, suggesting important spill over effects.

Keywords: Health care expenditure, decomposition, aging, pharmaceuticals, the Netherlands

JEL codes: I10

Introduction

Expenditures on health care continue to increase substantially, both absolutely and relative to national income, throughout most of the developed world. In the Netherlands, for instance, spending on health care increased from 6.6 to 9.9 percent of Gross Domestic Product (GDP) between 1973 and 2008, and real per capita spending almost trebled over this thirty-five year period (OECD, 2011). Expenditure growth on this scale has profound implications for both health and economic policy. Not surprisingly, accounting for it has been the purpose of much research (e.g. Newhouse, 1992; Getzen, 2000; Mehrotra et al., 2003; Dormont et al., 2006). This research has either employed aggregate time series or cross-country comparison data to identify the relationship of changes in total spending to determinants at the national level, with the residual being attributed to technological progress (Newhouse, 1992; Gerdtham et al., 1992; O'Connell, 1996; Getzen, 2000), or it has estimated the relationship of mean expenditure to potential determinants at the individual level, with changes in the relationship being attributed to technological progress and other structural changes (Dormont et al., 2006). In this paper, we do not merely account for the growth in total (mean) health care expenditure (HCE) but also explain change in its full distribution. This allows us to identify whether the growth is being driven by increased spending at all levels or whether spending on high cost cases is rising relative to the average such that the distribution is becoming even more skewed. We examine the extent to which the drivers of HCE growth vary along the distribution. For example, we can address not only the question of how much population aging is contributing to the growth of mean expenditure but also whether this contribution is stronger at high expenditures than low expenditures and so whether aging stretches or squeezes the distribution. For health insurers and health care providers paid prospectively, it is important to know not only how average expenditures vary with observable characteristics but also how their dispersion differs.

We implement this detailed decomposition of change in the distribution of HCE using very rich individual level insurance claims data on the actual acute care expenditures for two-thirds of the Dutch population over a seven year period from 1998 to 2004[1]. These data are linked to the hospital discharge and mortality registers allowing HCE to be explained not only by demographics, time-to-death and cause-of-death, but also by hospital diagnosis and medical treatment patterns in the hospital. The latter allows us to open the 'black box' of medical technology by identifying the expenditure growth that can be directly attributed to changes in medical practices in the treatment of specific conditions. We decompose the growth in two large components of HCE – hospital care and pharmaceuticals – separately. Not only the rate of growth but also its explanation turns out to differ substantially between the two. We explore the extent to which changes in hospital practices (e.g. a

[1] This is the longest period for which we have comparable data.

higher proportion of outpatient care and reduced length of stay) appear to have spill over effects on pharmaceutical spending.

The distribution of HCE may change for two broad types of reason. First, the distributions of the determinants of HCE may change. Through its impact on population health, aging is the most obvious contributor to this source of change. Second, structural changes may alter the way in which given determinants impact on HCE. Medical technology, other changes in medical practice, and changes in health policy at both the micro level of hospitals and insurers, and the macro level of government and regulators are the most likely sources of shifts in the relationship of HCE to its determinants. Most attempts to forecast future trends in HCE, including those that aim to identify the contribution of population aging, estimate a model of HCE and use this to simulate HCE under alternative scenarios about future trends in determinants (e.g. Zweifel et al., 1999; Seshamani, Gray, 2004; Stearns, Norton, 2004; Breyer, Felder, 2006; Lafortune et al., 2007; Häkkinen et al., 2008). This assumes that the relationship of HCE to its determinants is stable, which is unlikely. At best, these forecasts indicate what will happen in the absence of structural changes within a sector that is noted for technological progress, high government regulation and many policy reforms.

We employ a decomposition method that separates the observed change in the distribution of HCE into the part due to changes in determinants and that due to their changed impact. The size of the second part gives an indication of how wrong a forecast of HCE growth would be if it were made on the assumption of a stable relationship. In this respect, our study is similar to that of Dormont et al. (2006), who decomposed the growth in French HCE over the period 1992-2000 using a method in the spirit of Oaxaca (1973) and Blinder (1973). While Dormont et al. explained the change in mean HCE, we decompose the change in the full marginal distribution. This allows us to establish whether the contribution of shifts in determinants, such as aging and population health, is constant across the distribution and also whether the structural shifts in the relationship are more evident for high or low cost cases. We implement this by using distributional regression to generate counterfactual distributions (Chernozhukov et al., 2009).

This approach delivers findings that could not have been uncovered by a standard decomposition of mean HCE. The growth in expenditures on hospital care is strongest at the middle of the distribution and is driven mainly by changes in the distributions of determinants. In contrast, pharmaceutical expenditures increase most at the top of the distribution and are mainly attributable to structural changes most likely attributable to treatment of the highest cost cases with even more expensive drugs. Population aging contributes more to the growth of spending on pharmaceuticals than it does to that on hospital care. Aging (and all its correlates) explains 12 percent of the increase in median HCE in the Netherlands between 1998 and 2004. Of all the determinants, changes in hospital practice styles contribute most to the increase not only in spending on hospital care but also on pharmaceuticals. An

increased rate of outpatient visits is the single most important of these determinants. The fact that increased reliance on outpatient care can explain greater spending on pharmaceuticals suggests that there are indeed important spill over effects.

In the next section, we describe the most important structural changes in the Dutch health sector within the study period that may have altered the determination of HCE. In the third section, we present the decomposition method employed. The data are described in the fourth section and the results are presented in the fifth. The final section concludes by drawing lessons about the drivers of HCE and acknowledgement of some limitations.

Structural changes in the determination of health care expenditure in the Netherlands

Government policy

Until 2001, the volume of hospital care was constrained by fixed global budgets that enforced an income ceiling on hospitals. Hospitals' activity was further contained by the fact that they were permitted to keep the surplus when the amount of care provided was lower than *a priori* agreed. Since 1995, hospital specialists have been paid by fixed lump sums rather than fee-for-service. This budget funding successfully contained costs; real HCE grew by an average of 3.7 percent annually and remained fairly constant at 7.7-8.3 percent of GDP over the period 1983-2000 (OECD, 2011).

Tight budgets alongside population aging and technological progress resulted in lengthening waiting lists for inpatient care. Pressure on the government arose both from public dissatisfaction and rulings from the national court and EU Court that patients have an enforceable right to timely care (European Court of Justice, 2001; Van de Vijsel et al., 2011). Maximum waiting time standards were developed. The mounting pressure on the government resulted in a sudden relaxation of hospital budgets in 2001. Hospitals received posterior compensation when output exceeded their *a priori* set budget (House of Representatives, 2000). Although the additional revenue could only be spent on waiting list reduction, in practice, budgets became open-ended. Medical specialist fees were tied to actual production removing financial incentives to under produce. These changes in hospital funding led to substantial increases in HCE, with real spending growth of 6.3, 7.0 and 10.5 percent in 2001, 2002 and 2003, respectively (OECD, 2011).[2] This policy change offers a rare opportunity to trace how an injection of funding gets distributed across patients. We are able to identify how changes in admission rates impacted on not only the level but also the distribution of expenditures.

[2] Mackenbach et al. (2011) also reports a substantial increase in the growth rates from 2001. They use the national rather than international terminology of HCE which is why their growth rates differ from the ones reported here.

The sudden relaxation of hospital budgets apparently facilitated a reduction in waiting times. Although waiting lists for acute care only slightly decreased between 1998 and 2002 (2.7 percent), the majority of patients was treated within the allowed waiting period (TCOZ, 2004; Van de Vijsel et al., 2011). Hospitalization rates increased disproportionately for the elderly and for treatments with long waiting times (e.g. for cardiovascular, orthopaedic, cataract and plastic surgery).

Medical technology and practice

Although technological progress can, in principle, reduce costs (Cutler, McClellan, 2001; Cutler, 2007), it tends to introduce more expensive treatments and promotes more widespread use, resulting in higher HCE (Bodenheimer, 2005), particularly on pharmaceuticals (Dormont et al., 2006; Häkkinen et al., 2008). To give one example, TNF alpha blockers, which were introduced in 2000, improved the health status of rheumatoid arthritis patients considerably, but increased treatment costs substantially. In 2010, two TNF alpha blockers were the two most expensive non hospital drugs in the Netherlands, together costing 340 million euro (Health Care Insurance Board, 2011).

Technological progress can also impact on HCE through changes in medical practice. It probably helped facilitate shifts from overnight admissions to day care admissions and policlinic visits (TCOZ, 2004; Borghans et al., 2008) and reduced lengths of stay that occurred between 1998 and 2004. In addition to these substitutions between hospital treatments, there has also been a movement from hospital to GP treatment for some specific diagnoses. An important example is the increased responsibility of GPs for the detection of diabetes within their patient population and its treatment (Rutten et al., 1999; Niessen et al., 2003).

Integrated care programs, treatment protocols and drug formularies are particularly relevant changes in medical practice. A well-known example of an integrated care program is the specialized stroke unit, which was introduced in the Netherlands from 2000 (Van Exel et al., 2005). This could either lower or raise treatment costs since average length of stay of admitted stroke patients decreased from 22 to 12 days between 1998 and 2004, while treatment intensified during the shorter stay. The treatment costs of stroke will also have been influenced by GP guidelines to admit all stroke patients to the hospital, which increased hospitalization rates but reduced the average severity of stroke patients admitted.

These are just a few examples of the multitude of policy, technology and medical practice changes that are frequently occurring within the health sector and may alter how health spending responds to determinants such as population demographics and health, which are also changing. A Oaxaca (1973) – Blinder (1973) decomposition could be used to distinguish the contribution of such structural changes to the shift in mean HCE but this would not allow us to examine whether changes at the top of

the HCE distribution are explained differently from those at the bottom or middle. It seems likely that, say, technological progress impacts on HCE differentially at different points of the distribution. For example, extremely expensive innovative medicines typically treat serious, high cost conditions resulting in an outward shift of pharmaceutical expenditures given observable determinants that is more pronounced at the top of the distribution.

Decomposition method

A number of methods extending the Oaxaca-Blinder decomposition to explain the difference in full marginal distributions, mostly of wages, have been proposed (e.g. DiNardo et al., 1996; Gosling et al., 2000; Machado, Mata, 2005; O'Donnell et al. 2009; Fortin et al, 2011). Chernozhukov et al. (2012) provide the inference for methods based on regression models, and introduce one that uses distributional regression (Foresi and Peracchi, 1995). The decomposition derives from the fact that the marginal distribution of an outcome (Y), in our case HCE, is equal to the integral of its conditional distribution over the distribution of covariates (X), $F_{Y_t}(y) = \int F_{Y_t|X_t}(y|x) dF_{X_t}(x)$. Counterfactual distributions can be constructed by integrating the conditional distribution in one year over the distribution of covariates from another. For example, the hypothetical distribution of HCE that would have materialised if the distribution of covariates had remained as it was in 1998 but the relationship of HCE with these covariates was as in 2004 is, $F^{98}_{Y_{04}}(y) = \int F_{Y_{04}|X_{04}}(y|x) dF_{X_{98}}(x)$. The decomposition involves comparison between marginal and counterfactual distributions. For example, the change in the distribution of HCE between 1998 and 2004 that is attributable to change in the distribution of determinants is given by, $F_{Y_{04}}(y) - F^{98}_{Y_{04}}(y)$.

Implementation of the decomposition requires estimators of the conditional distribution of HCE and of the marginal distributions of its determinants. For the former, we use distributional regression. This involves directly estimating the conditional distribution function through a series of binary response models for the probability that the outcome lies below increasing thresholds (y), $e.g.\ F_{Y|X}(y|x) = \Lambda(x\beta(y))$ where $\Lambda(\ )$ is some link function, which can be specified as logistic, standard normal or the identity, and $\beta(y)$ is a functional parameter that is allowed for vary freely with the value of y. This is very similar to quantile regression, with the essential difference being that the latter models effects on the inverse of the conditional distribution. Both methods permit covariates to change the location, scale and entire shape of the distribution. We use the distributional regression (DR) estimator since quantile regression can provide a poor approximation to a conditional

distribution with mass points (Chernozhukov et al, 2012), which in our application occurs at zero expenditure. DR does not require smoothness of the conditional density, since the approximation is done point wise at the threshold y.[3] A further advantage of DR in this application, given our large sample size, is computational speed.

DR is used to estimate the impact of covariates on the probability that HCE lies below each of 400 percentile points of the unconditional distribution. These probabilities are estimated by the linear probability model i.e. the link function $\Lambda(.)$ is specified as the identity function, since, with our large sample, estimation time is considerably faster than with logit or probit, while the decomposition results are virtually identical. The distributions of covariates are estimated from the empirical distributions.

The simulated marginal distribution for each year $\left(\hat{F}_{Y_t}(y)\right)$ is obtained, following the plug-in-rule, by integrating the DR estimates of the conditional HCE distribution $\left(\hat{F}_{Y_t|X_t}(y|x)\right)$ for that year over the empirical distribution of the covaraites for the same year $\left(\hat{F}_{X_t}(x)\right)$ (Chernozhukov et al, 2012). The counterfactual distribution of HCE in 2004 if the distribution of covariates had remained as it was in 1998 is obtained following the same principle by combining the estimated 2004 conditional distribution of HCE with the 1998 distribution of covariates, $\hat{F}^{98}_{Y_{04}}(y) = \int \hat{F}_{Y_{04}}(y|x) d\hat{F}_{X_{98}}(x)$.[4] The change in the distribution of HCE observed between 1998 and 2004 can then be decomposed as follows:

$$F_{Y_{04}}(y) - F_{Y_{98}}(y) = \left[\hat{F}_{Y_{04}}(y) - \hat{F}^{98}_{Y_{04}}(y)\right] + \left[\hat{F}^{98}_{Y_{04}}(y) - \hat{F}_{Y_{98}}(y)\right] + \hat{\varepsilon} \quad (1)$$

where $\hat{\varepsilon}$ is a residual caused by differences between the empirical and simulated distributions. The first term on the right-hand side is the estimated change in the distribution of HCE attributable to changes in the distributions of its determinants. The second term is the change due to the structural shifts in the relationship of HCE to its determinants. Standard errors are obtained by the bootstrap (100 iterations), the validity of which for this decomposition procedure is established by Chernozhukov et al. (2012).

We present decompositions of changes in quantiles. These are obtained using the fact that the quantile function is the inverse of the cumulative distribution, $Q_{Y_t}(\tau) = F^{\leftarrow}_{Y_t}(\tau), \tau \in (0,1)$ where $F^{\leftarrow}_{Y_t}$ is the left

[3] We did experiment with the use of quantile regression to estimate the conditional distribution functions. It gave less precise estimates of the decomposition with larger standard errors of the contributions than those obtained using DR.

inverse of F_{Y_t} (Chernozhukov et al, 2012). Analogous to (1), the decomposition of changes in quantiles is

$$Q_{Y_{04}}(\tau)-Q_{Y_{98}}(\tau)=\left[\hat{Q}_{Y_{04}}(\tau)-\hat{Q}^{98}_{Y_{04}}(\tau)\right]+\left[\hat{Q}^{98}_{Y_{04}}(\tau)-\hat{Q}_{Y_{98}}(\tau)\right]+\hat{v}\,. \quad (1)$$

In addition to the broad distinction between changes in HCE attributable to changes in determinants and the effects of those determinants, we are interested in identifying the specific contributions of changes in population demographics, the disease burden, hospital admission rates, etc. This is done by comparing the first term in (1) (or (2)), which gives the effect of changing all covariates, with a similar difference in which all but one covariate is changed under the counterfactual. To clarify, divide the covariate vector into two components, $X_t=(Z_t,W_t)$. W_t represents a single covariate or a group of covariates (such as all age-gender groups) and Z_t are the remaining covariates. We re-sample from the 1998 empirical distribution of covariates such that *W* is distributed as it is in 2004.[5] This gives a counterfactual estimated distribution of covariates that would be observed if *W* were the only covariate to have changed between 1998 and 2004, $\hat{F}_{Z_{98},W_{04}}(z,w)$. Combining this with the estimated 2004 conditional distribution gives a counterfactual distribution of HCE in 2004 if all covariates but for *W* had been distributed as they were in 1998, $\hat{F}^{Z_{98},W_{04}}_{Y_{04}}(y)=\int \hat{F}_{Y_{04}|Z_{04},W_{04}}(y|z,w)\,d\hat{F}_{Z_{98},W_{04}}(z,w)$. Subtracting this from the simulated marginal 2004 distribution gives the effect of changing all covariates but for *W*, $\left(\hat{F}_{Y_{04}}(y)-\hat{F}^{Z_{98},W_{04}}_{Y_{04}}(y)\right)$. Comparing this with the first term from (1) gives the contribution of the change in *W* alone to the total contribution of the change in all covariates,

$$\left[\hat{F}_{Y_{04}}(y)-\hat{F}^{98}_{Y_{04}}(y)\right]-\left[\hat{F}_{Y_{04}}(y)-\hat{F}^{Z_{98},W_{04}}_{Y_{04}}(y)\right]=\hat{F}^{Z_{98},W_{04}}_{Y_{04}}(y)-\hat{F}^{98}_{Y_{04}}(y) \quad (2)$$

Analogous contributions of a single covariate to changes in quantiles can be derived. This procedure is repeated a number of times to compute the contribution of the change in each (group of) covariate(s). Since covariates are not changed cumulatively, there is no path dependence in this process. The procedure to resample from the 1998 sample such that a specific covariate is distributed as in 2004 holds correlations between this covariate and the remaining covariates constant. Hence, the distribution of the correlates also changes. Combined with the non-cumulative changes in covariates, this implies that the detailed decomposition does not possess the adding-up property (Fortin et al,

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[4] Computationally, this is simply, $\hat{F}^{98}_{Y_{04}}(y)=n_{98}^{-1}\sum_{i=1}^{n_{98}}\hat{F}_{Y_{04}|X_{04}}(y|X_{98i})$ where n_{98} is the 1998 sample size (Chernozhukov et al, 2012).

[5] We do this in the same way as Machado and Mata (2005) by drawing from the 1998 distribution such that the relative frequencies (percentiles) of *W* are equal to those observed in the 2004 sample when *W* is categorical (continuous).

2011) - the sum of the contributions of changes in specific covariates will not equal the aggregate contribution of changes in all covariates.

Data

Sample

A variety of data sources linked at the individual level is used. Expenditures on hospital and other acute care are obtained from claims documented in the sickness fund records (Vektis) of the entire covered population, which accounted for approximately two-thirds of the Dutch population during the period of analysis. Until 2006, sickness fund insurance was compulsory for people below an earnings threshold. The remaining one-third of the population was not eligible for sickness fund insurance and could purchase a private voluntary insurance. Their expenditures are not recorded in the data. For individuals with an inpatient hospital admission – i.e. day care or overnight admission – diagnosis and the procedure administered are obtained from the hospital discharge records. Vital status, time-to-death and cause of death are obtained from the mortality register. Finally, age, sex and co-residence status are obtained from the municipality register.

A probability linkage process is used to link these four sources. Linking variables were date of birth, sex, zip code and survival status. Due to incompleteness of date of birth in Vektis, only 49 percent of the sickness fund records could be uniquely identified (N= 9,082,279). Of the linked individuals, we randomly selected 165,000 for each year to obtain a sample for which computation was feasible. We deliberately oversampled decedents and those with an inpatient admission by a factor of two in order to estimate the effects of covariates related to mortality and inpatient care more precisely.

Iterative proportional fitting (IPF) weights were derived to correct for the sample selection caused by the linking process and deliberate oversampling. IPF corrects the marginal distribution of the weighting variables in the study sample to that of the total sickness fund population (Deming and Stephan, 1940; Bethlehem, 2008). We use age*sex*decedent status, the linking variables, plus hospitalization status as weighting variables.

Measures of health care expenditure

We decompose change in the distribution of expenditure on: i) acute HCE; ii) hospital and other secondary acute care (hereafter: hospital expenditures); iii) pharmaceuticals. In each case the measure

is logarithm of the individual's average *monthly* spending over the course of the year.[6] The logarithm of expenditure is used because of the extreme skewness of the untransformed distribution.[7] Acute HCE comprises the sum of spending on hospital care, pharmaceuticals, transport, devices, obstetrics and maternity care covered by the basic benefit package. Dental and paramedic care are excluded because they were partly removed from the basic benefit package in 2004 resulting in an apparent drop in the level of these expenditures in 2004. GP expenditures are excluded because, given that GP's were mainly funded on a capitation base during the study period, payments made by the insurer do not correspond to the cost of the GP care provided. Hospital expenditures also include pharmaceutical spending during hospital admissions and include spending on other secondary acute care provided by rehabilitation centres and private clinics that supply treatment covered by public insurance. Pharmaceutical expenditures refer to spending on pharmaceuticals issued or prescribed at outpatient visits, including those prescribed by GP's.

The influence of technology and other changes in practices to HCE growth can be better captured when correcting for price changes unrelated to changes in treatment patterns, i.e. general and health care specific inflation. Apart from those on pharmaceuticals, all expenditures were deflated to 1998 values using the consumer price index (CPI). Due to several pricing policies, the price of pharmaceuticals decreased by 22 percent in the period 1998-2004 (SFK, 2009). Pharmaceutical expenditures were therefore inflated by this proportion. In addition to correcting for general price changes, we control for the Baumol (1967) effect as this raises HCE simply through the rising relative price of health care. We do so by assuming a 0.8 percent annual increase in the cost of labour intensive services only (Douven et al., 2006).

Changes in the empirical distribution of expenditures

Table I reports summary statistics for each category of expenditure in each year for the full sample and those with positive expenditures. Figure 1 presents the empirical quantile functions of the logarithm of the three spending categories for 1998 and 2004. In the middle of the distribution, the quantile function of log acute HCE clearly shifts to the left indicating a substantial increase in HCE in the period 1998-2004. Mean real expenditure increased by 28 percent.[8] This growth at the mean is mostly driven by increases in the upper half of the distribution. The 25th percentile decreased by 14 percent, while the 50th and 75th percentiles increased by 38, and 39 percent respectively. Restricting attention to

[6] For those observed the full year, yearly expenditures are divided by 12. For individuals observed part of the year (mainly decedents), yearly expenditures were divided by the number of days observed and multiplied by 365/12 to approximate average monthly expenditures.
[7] Simply from a practical perspective, this makes graphing of the distribution on the raw scale difficult.

individuals with any expenditure, growth in spending is positive across the entire distribution and is somewhat greater at the centre of the distribution.

Figure 2 shows the change in the distributions in more detail by plotting the vertical difference between the quantile functions presented in figure 1. For the entire sample, the downward spike around the 20th quantile for the change in log HCE is due to an increase in the proportion for which no HCE were incurred. A similar spike is evident at a slightly higher quantile in the distribution of pharmaceutical expenditures. Both are almost entirely attributable to the removal of the contraceptive pill from the basic benefit package in 2004 with the result that the cost of contraceptives is not evident in the claims data in the later year.[9] In contrast, there is a positive spike in the difference in the quantile functions for hospital expenditures, which reflects the increased propensity to receive hospital care mainly as a result of the initiatives to reduce waiting lists.

Hospital and pharmaceutical spending together comprise approximately 90 percent of all acute HCE. Their distributions evolve in entirely different ways over the period. Mean pharmaceutical spending increased by no less than 69 percent, mean hospital spending only by 18 percent. The growth in hospital expenditures is strongest at the centre of the distribution, again a reflection of the increased propensity to be treated. Restricting attention to individuals with positive expenditures, spending on hospital care displays moderate and fairly constant growth over much of the distribution (figure 2). In contrast, the growth of pharmaceutical expenditures is greatest at the top of the distribution, even after conditioning on positive expenditures. This suggests more expensive and/or intensive pharmaceutical treatment of the already high cost cases. The shift in the distribution of total HCE is an average of these very distinct patterns in the growth of hospital and pharmaceutical spending.

Covariates

We use data on demographics, disability, time-to-death (TTD), cause of death, utilisation of hospital care, and hospital diagnosis and procedure to explain the distribution of HCE. Some of these (e.g. TTD) are potentially endogenous to the expenditures on health care. Since we do not interpret the estimates as causal effects but rather associations that can be used to decompose the change in HCE, we are not concerned about endogeneity.

Table II presents year specific means of some of the covariates. See appendix A for a detailed description of the covariates. There is a significant increase of around 10 months in the mean age of

[8] Our estimated nominal growth rate of mean spending for the period (42%) is similar to that calculated by Statistics Netherlands (2011).
[9] The likelihood of positive claims for pharmaceuticals expenditures is significantly decreased only for females aged 25-34.

the sample over the seven year period and a significant decrease in the proportion males. In the models, we capture the impact of demographics on HCE through sex specific dummy variables defined for 10 years age bands, plus 0-14 years and 85+ years. Longevity is measured by an indicator of whether the person dies within 5 years of the time of observation and the TTD in months and its square.

Co-residence status is used since co-habiting couples are often found to be healthier than those living alone. Institutionalized individuals are also identified. While they should have a greater care need, basic in-house care provided by residential and nursing homes, covered by long-term care insurance, may substitute for acute services. The proportion of individuals living alone increased over the period.

HCE vary with the intensity and type of health care utilised. The quantity and nature of hospital care is measured by: number of first outpatient (policlinic) visits, number of overnight and day care admissions, type of hospital, and length of stay (LOS) The share of individuals with an outpatient visit increased by 6.8 percentage points. The propensity to have day care increased by 1.5 percentage points while the chances of being admitted for an overnight stay decreased by 0.5 percentage points. Average LOS for those admitted overnight decreased from 9.1 to 7.1 days. A greater share of patients was admitted to teaching hospitals in 2004 than in 1998, and fewer were admitted to general hospitals.

The data allow us to identify not only receipt of hospital treatment but also the nature of the treatment received. In the models for acute HCE and hospital expenditures, we control for hospital procedures. Over 10.000 different procedures could be distinguished. Procedures are grouped into 47 categories based on the International Classification of Health Interventions (WHO, 2011). Given limited space, Table II only shows the means of a few of these procedures for which long waiting times existed. Appendix B presents the means for all 47 procedures. As a proportion of the total number of inpatient admissions, the share of percutaneous transluminal coronary angioplasty (PTCA) and surgery of the eye and lens, for which long waiting times existed, increased significantly. The share admitted for diagnostics also increased while those that did not receive a procedure (e.g. pharmaceutical hospital treatment, admission to observe rather than to treat) decreased.

The only health indicator available for the entire sample is whether the individual has a work disability, which shows no change over time. Diagnosis is available for day care and overnight hospitalized individuals and cause of death can be established for the deceased. Because diagnosis is observable only for those admitted to hospital, it is not a clean indicator of the population disease burden. It also reflects changes in admission rates and the state of technology available to treat a specific health problem. The disproportionate growth in certain diagnoses reflects the reduction in waiting lists for treatment of these conditions. Diagnoses are grouped into 39 categories according to ICD-10 chapter and prevalence rates (see appendix A). Table II presents prevalence rates per ICD chapter (appendix B presents them for all 39 diagnoses). The most notable increases are in the vague

categories – symptoms/ill-defined conditions and not disease related admissions. Diagnoses related to procedures for which long waiting times existed also increased significantly (e.g. other cardiovascular disease, eye disorders, osteoarthritis, other disease of the nervous system (carpal tunnel syndrome)). The largest decreases are in respiratory disease and conditions related to pregnancy and child birth.

Cause-of-death is grouped into 10 categories. There was a very large reduction in deaths due to cardiovascular disease. There was also a fall in the proportion of deaths recorded as due to ill-defined conditions, which is also a category for which there was a steep rise in hospital admissions. The proportion dying from mental and behavioural disorders and a disease of the nervous system/sense organs increased.

Results

Figure 3 and Tables III-IV present the decomposition given by equation (2) of the change in the distribution of each category of health expenditure into that due to change in the distribution of determinants and that due to change in the conditional distribution. The figure shows the decomposition of the change in the quantile function for the full sample and for those individuals with positive expenditures. 95% confidence intervals are indicated by shading. Estimates at certain quantiles are given in Tables III and IV for the two samples respectively. For all three categories of expenditure and both samples, the change in expenditure and its explanation varies across the distribution making evident the superiority of this decomposition approach over one that explains change in the mean only. Heterogeneity in the relationship of health expenditure to covariates is confirmed by a Kolmogorov-Smirnov test of the null of equality of effects at all quantiles ($p=0.000$ for all three expenditure categories). In all cases the residual of the decomposition is small, indicating that the simulated distribution is very close to the empirical distribution. Standard errors are also very small such that estimated contributions at almost all quantiles are statistically significant.

Decomposition of change in the distribution of acute HCE

For the entire sample, including those without any acute HCE (top left panel of Figure 3 and top panel of Table III), changes in the distributions of determinants raise expenditures at all quantiles while change in the conditional HCE distribution decreases expenditures at lower quantiles but raises them at higher quantiles. The downward shift in HCE conditional on covariates at low quantiles is due to the withdrawal of contraceptives from the benefit package, which resulted in a greater proportion of individuals with no health expenditure claims.

Up to the median, the positive effect of changes in the distributions of determinants is offset by the negative effect of changes in their effect on HCE. Median HCE increased by 29 percent, of which 26 percentage points can be explained by changes in covariates, supplemented by a now small positive contribution of 3 percentage points from change in the conditional HCE distribution. From the median upwards, the contribution of change in the conditional distribution remains positive and grows in magnitude, while that of the change in the distributions of covariates steadily decreases. From the 80th percentile, change in the conditional distribution explains more of the increase in HCE than do changes in covariates. Changes in the relationships of covariates with HCE explain 20 percentage points of the 27 percent increase at the 90th percentile.

Restricting attention to individuals with any claims for HCE (top left panel of Figure 3 and top panel of Table IV), changes in the distributions of covariates and in the conditional HCE distribution both contribute positively to the increase in spending throughout the distribution. The contribution of changes in the distributions of determinants is generally larger than that of change in the effects of these determinants and reaches a peak at the centre of the distribution. After increasing up to the 40th percentile, the contribution of change in the conditional HCE distribution is roughly constant with the result that it is the dominant effect from the 80th percentile upward. Increases in health expenditures at the very top of the distribution are mainly driven by higher costs for given observable population and health care characteristics. Given we control for the procedures conducted in hospital, as well as diagnoses, demographics and cause of death among patient characteristics, this indicates that the costs of conducting given procedures have increased for the highest cost conditions. This suggests that technological developments are particularly pertinent to the explanation of growth in the highest expenditures.

Decomposition of changes in hospital and pharmaceutical expenditures

Marked differences in the explanations of the changes in the distributions of hospital and pharmaceutical expenditures emerge. The growth in hospital expenditures throughout most the distribution is mainly driven by changes in the distributions of determinants, the effect of which is offset by a much smaller negative effect of change in the conditional HCE distribution (middle panel of Figure 3). This is true for the entire sample (middle panel of Table III) and for those with positive hospital expenditure claims (middle panel of Table IV). The dominance of changes in determinants is not surprising given expenditures are modelled as a function of a large number of hospital-related characteristics, including the number and type of admissions, the type of hospital and the procedure administered. Changes in receipt of treatment (admissions), what is done (procedures), how it is done (outpatient, day-care or overnight) and where it is done (general, specialist or teaching hospital) should

indeed drive changes in hospital expenditures. Changes in these and other determinants constrain hospital expenditure growth only in the top quintile of the distribution of positive expenditures, which mainly comprises inpatient hospital users. This negative contribution results from a higher proportion of day care admissions in 2004 and a reduction in LOS. In this top quintile, change in the conditional distribution raises hospital expenditures, again suggesting that technological progress further increases costs of treating the most expensive hospital patients. The negative contribution of change in the distribution conditional on the number of and type of admissions, procedures, etc. at lower expenditures is indicative of improved efficiency.

As for hospital expenditures, changes in the distributions of determinants raise pharmaceutical expenditures (bottom panel of Figure 3). But unlike for hospital expenditures, changes in the effects of determinants mostly contribute positively to growth in spending on pharmaceutical. The decrease in spending on pharmaceuticals at the bottom of the distribution is explained by the shift in the conditional distribution, which is attributable to the withdrawal of contraceptives from the benefit package. This change makes it more interesting to concentrate attention on the distribution of positive pharmaceutical expenditure claims (bottom right panel of Figure 3 and bottom panel of Table IV). Pharmaceutical expenditures rise markedly and above the 40^{th} percentile change in the conditional distribution accounts for the greater part of the increase, although changes in determinants continue to play an important role. Changes in the relationship of pharmaceutical expenditures to determinants explain approximately 60 percent of the increase in the 60^{th} percentile and around 75 percent of the increase in the 90^{th} percentile.

It is understandable that covariates play a relatively more important role in explaining spending on hospitals than they do in explaining spending on pharmaceuticals since the models for hospital expenditures include many hospital and procedure level characteristics while the models for pharmaceutical expenditures do not include information on the pharmaceuticals consumed. The positive and relatively strong role of change in the conditional distribution in explaining increasing spending on medicines indicates that the cost of pharmaceutical treatment for given diagnoses and other patient characteristics is rising. But, it should be kept in mind that we have tried to keep prices constant by inflating 2004 pharmaceutical expenditures to allow for the average 22 percent fall in the prices of medicines since 1998.

Detailed decomposition of the contribution of changes in covariates

Equation (3) is used to estimate the contribution of a specific (group of) covariate(s) to the change in the distribution of expenditure. Figure 4 shows the results for the three most interesting groups of covariates, which we label population aging (age composition), disease burden (work disability and

cause of death)[10] and hospital admissions (outpatient visits, and day-care and overnight admissions). In each figure, the contribution of the change in the distribution of all covariates to the change in the quantile function is indicated by the solid line. This simply reproduces the 'covariates' line from the left-hand panel of Figure 3. The dotted line indicates the contribution of changes in all covariates other than those indicated by the sub-heading. The shaded difference between these lines, which corresponds to the equivalent of equation (3) for quantiles, indicates the contribution of changes in the determinants specified by the sub-heading.

Of the population characteristics, changes related to aging contribute most to the growth in health expenditures. But this aging effect is confined to pharmaceutical expenditures. Changes in the age composition do not explain change in hospital spending at all. One might expect changes in the population disease burden, such as the large fall in deaths from cardiovascular disease, to explain change in the distribution of health expenditures but there is little or no evidence of this. Absence of this evidence is likely to be related to the endogeneity issue: health care use might have resulted in the fall of cardiovascular deaths (Mackenbach et al 2011). There is no impact of changes in population disease burden on the change in hospital expenditures and only a very small constraining effect on pharmaceutical spending growth. From the right-hand panel of Figure 4 it is evident that most of the contribution of changes in determinants to growth in all types of spending is accounted for by rates of hospital use. Increased use accounts almost entirely for the spike in the change in all three functions at low quantiles. The spike for hospital expenditures reflects the fall in the proportion with zero spending and this is explained by the rise in admissions and outpatient visits. Because pharmaceutical expenditures are also positively correlated with hospital admissions, the rise in the admissions rate is predicted to increase spending on medicines although, as is evident from the bottom left panel of Figure 3, this does not materialise. For hospital spending, it is evident that at higher percentiles, changes in covariates other than increased hospitalization rates exerted a negative influence on expenditures.

In Figures 5 (hospital spending) and 6 (pharmaceutical spending) we further disentangle the contribution of changes in hospital-related factors. For the purpose of comparison, the upper left graph in each figure again presents the contribution of changes in hospital use rates. The top right graph identifies the contribution of changes in inpatient only. Comparing the two reveals that changes in inpatient care are responsible for only a small proportion of the growth in both hospital and pharmaceutical expenditures. Hence, the 6.8 percentage point increase in outpatient visits must be responsible for the majority of the spending growth generated by changes in determinants throughout

[10] Hospital diagnoses are not included since, as noted above, they reflect hospital admissions policy and not only the population disease burden.

the distribution of both types of expenditures. As would be expected, given the high cost of inpatient care, the contribution of changes in inpatient admissions is concentrated at higher quantiles.

Changes in other hospital-related determinants contribute very differently to hospital and pharmaceutical expenditure growth. The middle right graph in each figure shows the effect of the reduction in LOS of an overnight admission. Without this reduction, there would have been a greater increase in hospital spending at the very top of the distribution. Due to the skewed nature of health expenditures – 10 percent of the population is responsible for approximately 70 percent of health expenditures – a small reduction in the top of the distribution implies a large impact on total HCE. In this case, counter to the hypothesis of substitution, there is no discernible impact of reduced LOS on pharmaceutical spending. The bottom left graph in each figure shows the effect of a decrease in the propensity of overnight admissions – a shift towards more day care and policlinic visits – holding the hospital rate constant. For hospital expenditures, an effect is evident at nearly all quantiles. At high quantiles, the dotted line (all changes but reduced proportion of overnight admissions) lies above the solid line (all changes) indicating the extent to which expenditures would have been even higher if all changes had taken place but overnight admissions as a proportion of hospital admissions had not changed. The relative reduction of overnight admissions has however increased spending at the lower quantiles (the dotted line lies below the solid line), probably by treating more severe patients in day care and policlinic that would otherwise been treated during an overnight admission. The higher propensity to treat patients in day care and policlinic contributed to the rise in pharmaceutical expenditures across the whole distribution. This is suggestive of pharmaceutical treatment substituting for treatment in hospital. In addition, the middle left-hand graph shows the effect of a greater share of day care admissions of inpatient admissions only. For hospital expenditures, an effect is only evident at high quantiles: if all changes had taken place but overnight admissions as a proportion of inpatient admissions had not changed hospital expenditures at the top of the distributions would be higher. There is less impact on the distribution of pharmaceutical expenditures indicating that the increased propensity of outpatient hospital use contributed most to the rise in pharmaceutical expenditures. The bottom right-hand graph in Figure 5 indicates that, holding the number and type of admissions constant, changes in procedures (e.g. higher proportion of bypass surgery, PTCA and hip replacement) had no impact on hospital expenditures except at the very top end of the distribution. Since some procedures increased while others fell, the implication is that any change in the mix of procedures did not have a noticeable impact on spending.

Tests of changes in relationship of HCE to its determinants

The decompositions reveal that changes in the conditional distribution contribute to the change in the distribution of health, particularly pharmaceutical, expenditures. It would be instructive to identify which particular relationships between HCE and its determinants are most important in driving change in the marginal distribution. This is not possible with the methods currently available for full distribution decomposition. In order to give some insight on which relationships have changed and in which direction, we concentrate on two parameters of the distribution – the probability of positive expenditure and the conditional mean of positive expenditures. For each, we test the null of no change in the effect of each covariate between 1998 and 2004 and we note the direction of significant changes. The probability of positive expenditure is estimated by probit and the conditional mean of log expenditures is estimated by OLS.[11] In each part we include full interactions with a time dummy and test stability by the significance of coefficients on these interactions. Table V presents these stability tests for (groups of) determinants.

Of the population characteristics, the increased effect of age, especially older age, on both the probability and the mean level of positive spending is most evident. The positive effect of work disability on the probability and the level of total health and pharmaceutical expenditures increased. The positive effect of being deceased within five years on the probability of positive spending for hospital and pharmaceutical care decreased but there are little or no changes in the impacts on the conditional means. This drop in the positive effect of being deceased on the probability of positive spending is likely related to the reduction of waiting lists: individuals are only placed on the waiting list when this is medically justified. Hence, the large increase in admissions for procedures for which waiting lists existed has benefited individuals that could have waited for treatment without being at sufficient risk for death.

Concerning hospital-related determinants, the positive effect of an inpatient admission (day-care and overnight) on mean (positive) total acute HCE and hospital expenditures fell. This is consistent with the rise in cheaper day care admissions. In addition, milder cases might have been brought into the hospital, resulting in lower average treatment costs. Costs of overnight admissions also fell. Treatment of more severe cases in policlinics probably accounts for the increased positive effect of outpatient visits on expenditures. The increased positive effect of polyclinic visits on pharmaceutical spending probably reflects a partial substitution of pharmaceutical for hospital treatment.

The joint effect of hospital procedures changed over time, with some increasing in cost and others decreasing. The joint effect of hospital diagnoses on hospital expenditures changed over time, with treatment costs rising for most. Most diagnoses were treated at lower pharmaceutical cost. Remember

[11] OLS residuals are not skewed (k=3.08) and so OLS on the log of HCE is an appropriate choice for the second part of this two-part model (Manning and Mullahy, 2001).

however that pharmaceutical treatment costs of nearly all age groups rose which might indicate that pharmaceutical costs have risen for nearly all population groups regardless of the disease.

Discussion

This is not the first paper to decompose the trend in HCE but it is the first to do so across the full expenditure distribution. This delivers findings that could not have been uncovered by a standard decomposition of mean HCE. Although the growth in acute HCE in the Netherlands was fairly evenly spread across the distribution, this is not true for hospital and pharmaceutical spending separately. Hospital spending growth, mainly driven by an increased propensity to use, was greatest around the centre of its distribution. This was related to the relatively large increase in outpatient rather than inpatient treatment. By contrast, pharmaceutical spending growth was highest at the higher quantiles, implying that the largest rise occurred among cases that were already very intensive and expensive drug users.

The explanation of the growth in acute HCE differs across the distribution. While changes in the distribution of determinants and the changing impacts of determinants both play an important role, the contribution of the former generally decreases along the distribution while that of the latter increases. There is a marked difference in the explanation of the rise in hospital and in pharmaceutical expenditures. Hospital expenditure growth is almost entirely explained by changes in determinants. Their contribution, however, falls at higher quantiles where the contribution of structural changes in the effect of determinants on hospital expenditure growth is apparent. This suggests that technological progress in the hospital sector is targeted at high-cost users and will increase their treatment costs even further. By contrast, changes in both the distribution of determinants and their effects are important in the explanation of pharmaceutical expenditure growth. The greater importance of changes in determinants for hospital expenditure growth is partly related to our selection of determinants that does include changes in hospital practices but not changes in pharmacotherapeutic treatment styles. Instead, the introduction of new expensive medicines is captured by the contribution of the changing effects of determinants. The higher price of innovative medicines due to patent rights and monopolistic price setting explains the relatively larger rise in the contribution of coefficients at the higher quantiles of the pharmaceutical expenditure distribution. While previous studies identified that increases in drug expenditures are largely driven by technological progress (Dormont et al., 2006; Häkkinen et al., 2008), we have demonstrated that technological progress accounts mostly for growth at the top end of the drug expenditure distribution. The relatively large contribution of technological progress to drug expenditure growth, especially at the top end of the expenditure distribution, is likely to hold for other countries as well. In more recent year, technological progress seems to have an even

larger effect on drug expenditure growth. Consider for instance the introduction of very expensive drugs for cancer treatment (Wilking et al., 2009).

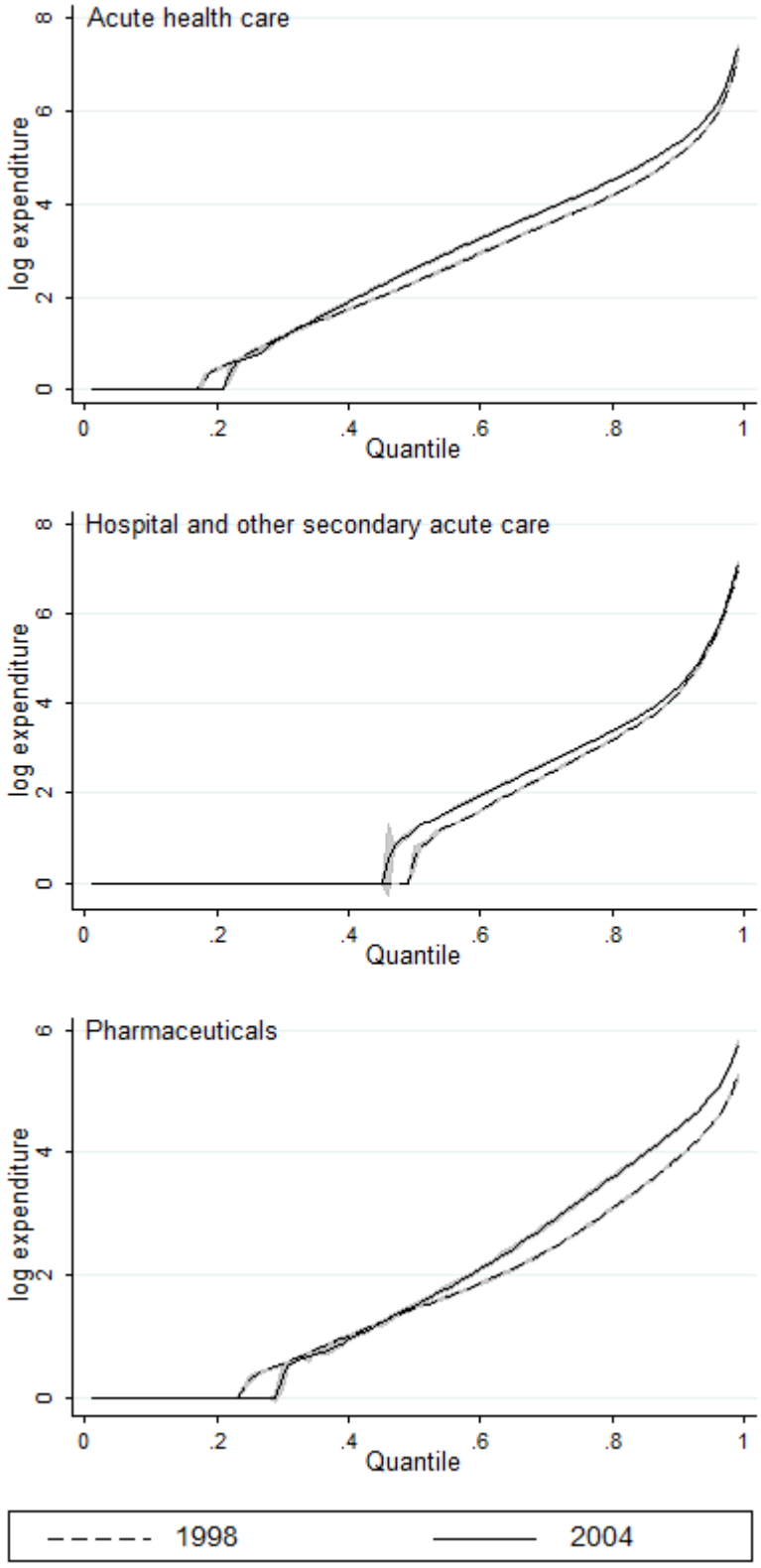
Changes in determinants that capture population characteristics only marginally contribute to the rise in acute HCE but, among these, population aging is the most important. Changes in hospital practice styles are by far the most important of the changes in determinants. While Dormont et al. (2006) found that changes in medical practices appear to play a dominant role in explaining HCE growth, our rich data allow us to take this further by disentangling the contribution of specific hospital practices. Increased propensity to use hospital care, in particular outpatient hospital care, explains nearly the entire contribution of changes in determinants to the rise in acute HCE and in hospital spending. However, changes in other hospital-related factors, have constrained the rise of expenditures in the higher quantiles. Shortened LOS of overnight admissions and an increased propensity to be treated in day care or the policlinic shifted hospital expenditures from the top to the middle of the distribution. The increased use of day care and policlinics contributed to the rise in pharmaceutical expenditures across the whole distribution, suggesting that hospital care has to some extent been substituted by pharmaceutical care. We cannot, however, conclude anything about the direction of the effect: the introduction of expensive innovative pharmaceuticals may have saved hospital costs, as has been concluded for the US (e.g. Lichtenberg, 2006, 2007, 2009), or the less intensive hospital treatment may have shifted costs to the pharmaceutical sector.

Our study has its limitations. First, although we have unique and rich data, the absence of health information on non-hospitalized patients somewhat impedes the interpretation. In the absence of general population health measures, much of the health effect may be captured by the age effect. Second, changes in the prevalence rates of inpatient hospital diagnoses and changes in the effect of these diagnoses on HCE should be interpreted cautiously. A fall in a specific hospital diagnosis does not necessarily imply a decrease in the overall prevalence of that condition. Similarly, changes in the effect of inpatient hospital diagnoses may not fully capture (changes in) the effect of a disease on acute HCE, hospital expenditures and pharmaceutical. Take the example of outpatient TNF-alpha blockers: while their introduction has increased pharmaceutical treatment costs for rheumatoid arthritis, this cannot be inferred from a change in the effect of a rheumatoid arthritis diagnosis as improved pharmaceutical treatment is likely to have reduced the probability of an inpatient admission for that condition. Third, due to the lack of data on GP spending (funded by a capitation system), we could not explicitly examine possible substitution of hospital treatment by GP treatment, although some of the prescribing consequences are captured indirectly in pharmaceutical spending patterns.

In conclusion, we find that changes in medical practices, in part resulting from technological progress and the relaxation of budgets, were the dominant drivers of acute HCE growth in the Netherlands. While there is a discernible contribution of population aging to spending growth, it is moderate:

population aging could explain 3.5 percentage point of the 29.4 percent growth in median real expenditure. However, its impact cannot be entirely isolated from that of technological progress and the relaxation of hospital budgets. Both developments seem to have disproportionately benefited the elderly given that expenditures on older groups rose more than those on younger groups, and both the expanded waiting list treatments (e.g. cataract and orthopaedic surgery, PTCA) and more liberal prescribing (e.g. of lipid-lowering drugs) were more concentrated among the elderly (Mackenbach et al., 2011). The latter is a reminder that HCE growth does not merely respond to population aging and developments in medical technology but is the result of deliberate government policy shifts. Predictions of HCE solely based on changes in population characteristics are very naive and are likely to grossly underestimate future HCE growth. In our case, such projections would have estimated a 6 percent growth at the median instead of the 29 percent growth actually observed between 1998 and 2004.

Figure 1 Empirical quantile function of log monthly expenditures in 1998 and 2004



Note: 95% bootstrapped confidence intervals are indicated by shading. Sample size is 165,000 for each year.

Figure 2 Change in quantile function of log monthly expenditures by category between 1998 and 2004

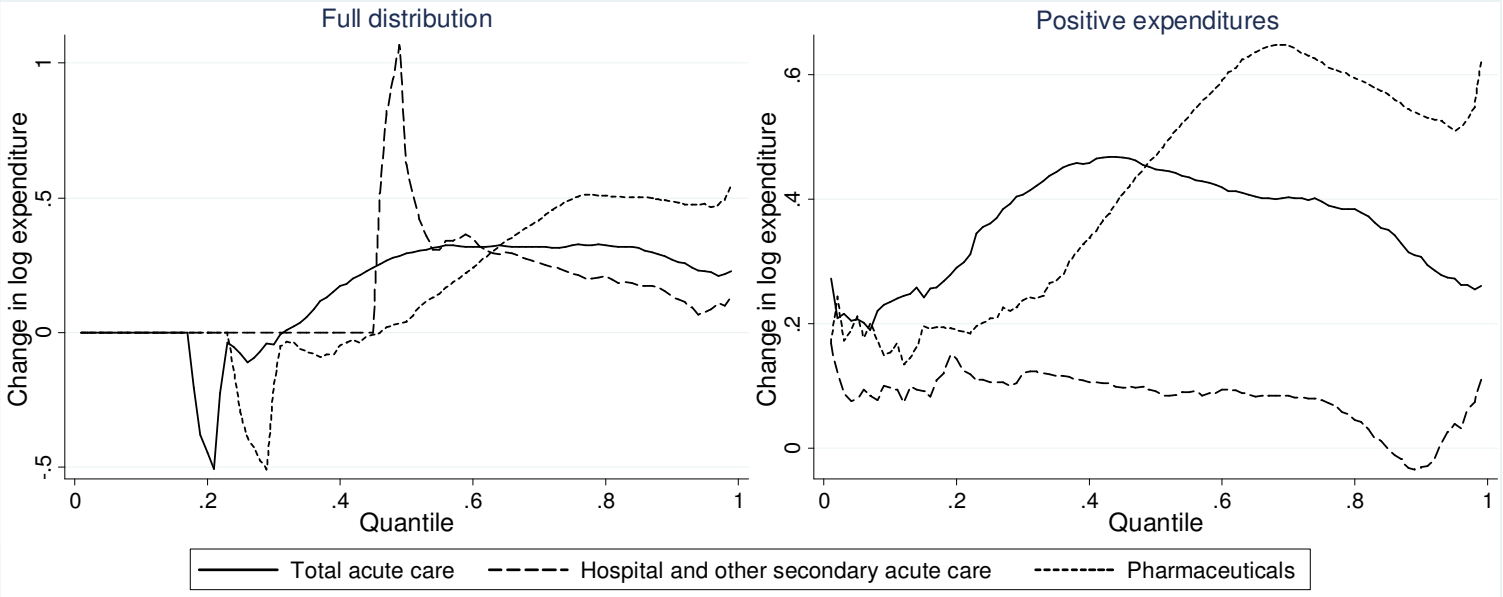
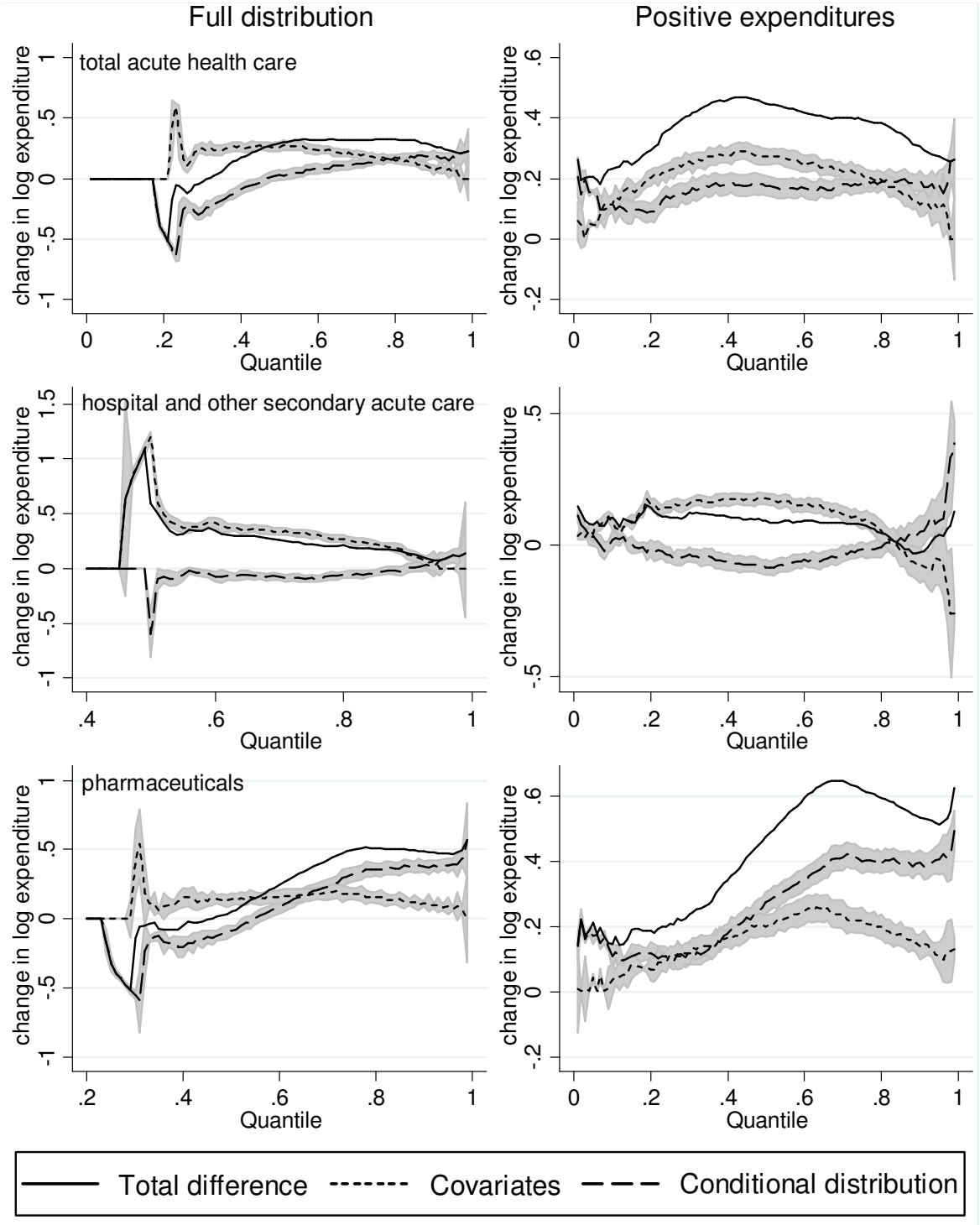
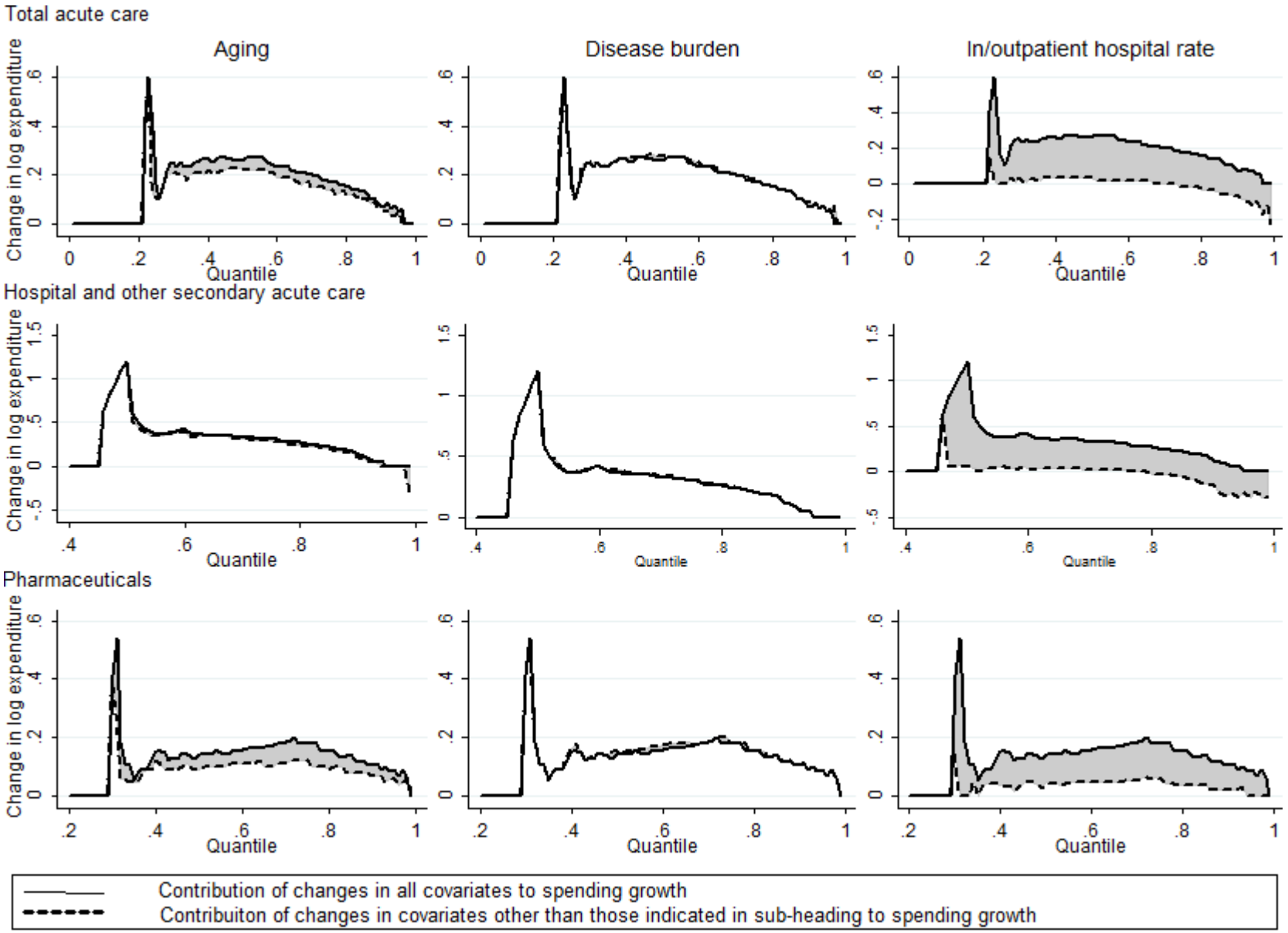


Figure 3 Decomposition of change in quantile function of log expenditure between 1998 and 2004 into contribution of changes in covariates and change in the conditional distribution



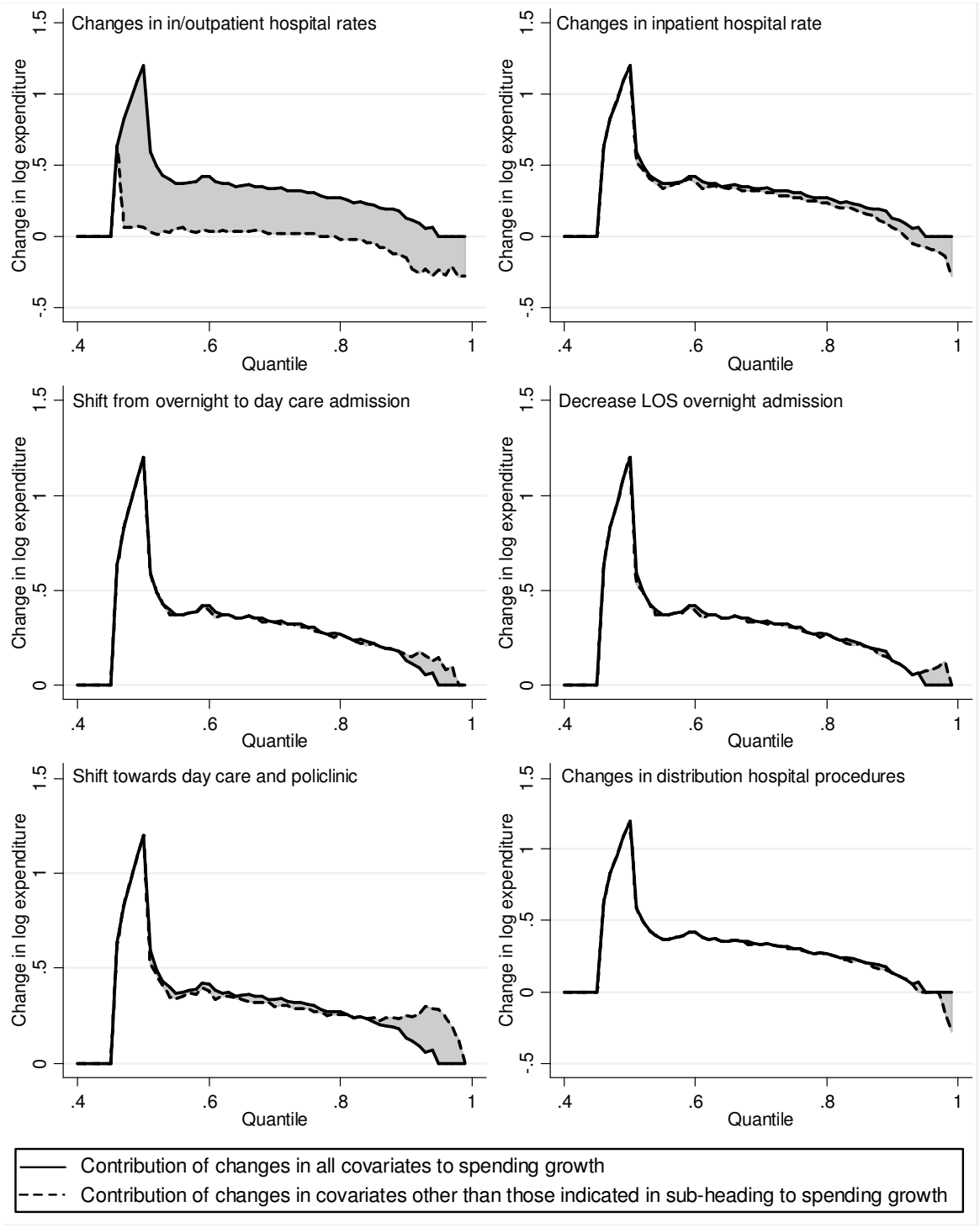
Note: Results are derived from the decomposition method defined by equation (2). The total difference is derived from the left hand side of (2), the contribution of changes in covariates from the first term on the right-hand side and the contribution of change in the conditional distribution from the second term on the RHS.
95% confidence intervals are indicated by shading.

Figure 4 Contributions of changes in specific covariates to change in quantile function of log monthly expenditure between 1998 and 2004



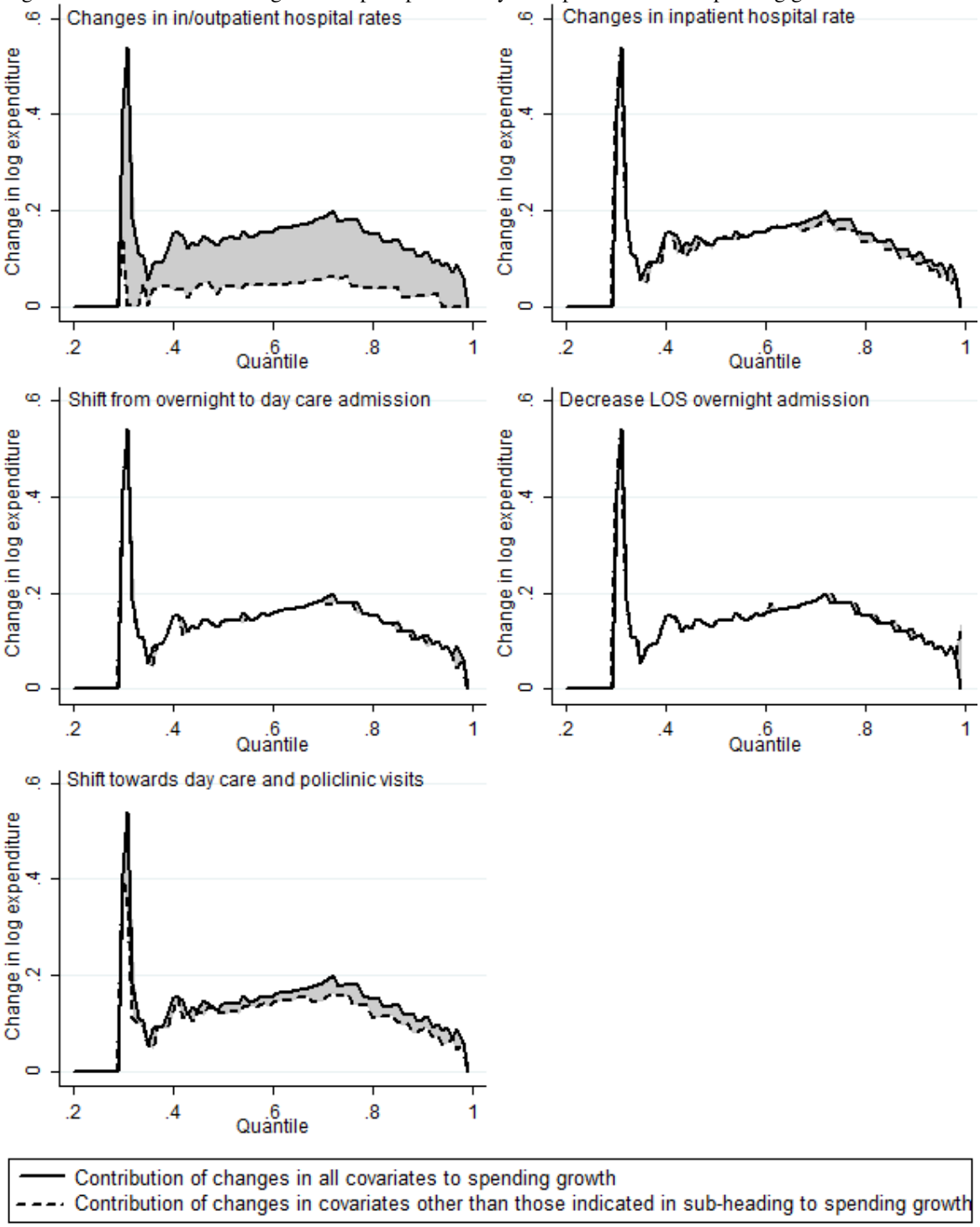
Note: The shaded area is derived from the equivalent of equation (3) for quantiles. Aging refers to changes in the age-sex composition. Disease burden refers to changes in work disability and cause of death. In/outpatient hospital rate refers to changes in in- and outpatient hospital use rates.

Figure 5 Contribution of changes in hospital level characteristics to change in quantile function of log monthly expenditures on hospital and other secondary acute care spending



Note: The shaded area is derived from the equivalent of equation (3) for quantiles. In/outpatient hospital rate refers to changes in in- and outpatient hospital use rates. Shift from overnight to day care admission refers to changes in the distribution of type of inpatient hospital admission (day care or overnight admission) holding the inpatient hospital rate constant. Decrease LOS overnight admission refers to the reduction of length of stay for overnight admissions holding the rate of overnight admissions constant. Shift towards day care and policlinic visits refers to changes in the distribution of type of hospital use (outpatient, day care or overnight admission), holding the hospitalization rate constant.

Figure 6 Contribution of changes in hospital practice styles to pharmaceutical spending growth



Note: The shaded area is derived from the equivalent of equation (3) for quantiles. In/outpatient hospital rate refers to changes in in- and outpatient hospital use rates. Shift from overnight to day care admission refers to changes in the distribution of type of inpatient hospital admission (day care or overnight admission) holding the inpatient hospital rate constant. Decrease LOS overnight admission refers to the reduction of length of stay for overnight admissions holding the rate of overnight admissions constant. Shift towards day care and policlinic visits refers to changes in the distribution of type of hospital use (outpatient, day care or overnight admission), holding the hospitalization rate constant.

Table I Summary statistics for per capita monthly health expenditure (1998 prices) stratified by year and spending category

	Total acute health care expenditures			Hospital and other secondary acute care expenditures			Pharmaceutical expenditures		
	1998	2004	Change (%)	1998	2004	Change (%)	1998	2004	Change (%)
Entire sample									
Zero spenders (%)	17.4	21.6		49.5	45.9		23.4	29.2	
Mean	84.3	107.8	28%	57.8	68.0	18%	17.7	29.8	69%
10th percentile	0.0	0.0	0%	0.0	0.0	0%	0.0	0.0	0%
25th percentile	1.2	1.0	-14%	0.0	0.0	0%	0.3	0.0	-100%
Median	9.1	12.5	38%	0.8	2.3	202%	3.3	3.5	5%
75th percentile	46.3	64.4	39%	15.5	19.5	26%	14.0	23.6	69%
90th percentile	153.6	201.4	31%	67.3	77.2	15%	49.1	80.3	64%
95th percentile	308.1	387.0	26%	201.3	217.0	8%	83.2	134.7	62%
Positive expenditures									
Mean	102.0	137.4	35%	114.5	125.3	10%	23.1	42.1	82%
10th percentile	1.3	1.9	46%	2.4	2.7	14%	0.8	1.1	37%
25th percentile	4.0	6.2	54%	5.1	5.8	13%	2.1	2.8	34%
Median	16.5	26.3	60%	15.1	16.7	10%	6.0	10.2	70%
75th percentile	61.6	92.0	49%	46.9	50.8	8%	22.5	42.6	90%
90th percentile	186.4	253.8	36%	199.0	192.9	-3%	61.7	106.0	72%
95th percentile	371.9	488.5	31%	463.5	482.2	4%	98.7	165.1	67%

Note: A sample of 165,000 is selected for each year from the full sickness fund population. IPF weights applied to correct for the sample selection caused by the linking process and oversampling of decedents and hospitalized.

Table II Means of (selected) covariates

Covariate[b]	1998	2004	Change[a]
Age	38.75	39.56	0.81†
Deceased within 5 years (%)	4.70	4.60	-0.10
Mean TTD if deceased	31.24	30.04	-1.21†
Male (%)	46.34	45.51	-0.83†
Co-residence status: Living alone (%)	14.49	15.39	0.91†
Co-residence status: Couple alone (%)	26.00	25.11	-0.89†
Co-residence status: Other private household (%)	57.80	58.00	0.20
Co-residence status: Institutionalized (%)	1.71	1.49	-0.22†
Outpatient (policlinic) visit (%)	28.60	35.43	6.83†
Mean first policlinic visits if>0	1.52	1.60	0.08†
Inpatient admission: day care (%)	3.56	5.07	1.51†
Inpatient admission: overnight; %)	7.16	6.67	-0.50†
Mean length of stay if>0	9.08	7.08	-2.00†
Admission to university hospital (%)	10.24	10.71	0.46
Admission to other teaching hospital	20.48	29.61	9.13†
Admission to specialized hospital	1.07	0.92	-0.14
Admission to general hospital	72.35	63.67	-8.69†
Hospital procedure: PT(C)A (% inpatient stay)	1.07	1.77	0.70†
Hospital procedure: surgery eye (% inpatient stay)	5.91	7.47	1.55†
Hospital procedure: other bone and joint surgery(% inpatient stay)	9.34	9.55	0.22
Hospital procedure: other therapeutic/preventive procedure (% inpatient stay)	50.68	50.54	0.14
Hospital procedure: diagnostics	12.71	14.46	1.76†
Hospital procedure: no procedure (% inpatient stay)	26.65	24.62	-2.03†
Work disabled (%)	10.03	10.09	0.06
Cause-of-death: external (% decedents)	4.18	3.91	-0.27
Cause-of-death: neoplasm (% decedents)	27.60	28.73	1.13
Cause-of-death: endocrine, nutritional, metabolic disease (% decedents)	3.35	3.49	0.13
Cause-of-death: mental and behavioral disorder (% decedents)	3.03	4.92	1.89†
Cause-of-death: disease of the nervous system/sense organs (% decedents)	2.26	2.74	0.47*
Cause-of-death: cardiovascular disease (% decedents)	36.13	32.38	-3.75†
Cause-of-death: respiratory disease (% decedents)	9.95	10.30	0.35
Cause-of-death: digestive disease (% decedents)	3.68	4.01	0.34
Cause-of-death: disease of the genitourinary system (% decedents)	1.83	2.16	0.34
Cause-of-death: symptoms, signs and ill-defined conditions (% decedents)	5.31	4.22	-1.09†
Cause-of-death: else (% decedents)	2.66	3.13	0.47*
Hospital diagnosis: infectious disease (% inpatient stay)	1.39	1.24	-0.15
Hospital diagnosis: neoplasm (% inpatient stay)	6.95	7.41	0.46*
Hospital diagnosis: endocrine, nutritional, metabolic disease (% inpatient stay)	1.92	2.08	0.16
Hospital diagnosis: disease of the blood (forming organs) (% inpatient stay)	0.88	1.02	0.14*
Hospital diagnosis: mental and behavioral disorders (% inpatient stay)	1.6	1.23	0.07
Hospital diagnosis: disease nervous system/sense organs (% inpatient stay)	11.34	12.54	1.21†
Hospital diagnosis: cardiovascular disease(% inpatient stay)	12.97	13.17	0.21
Hospital diagnosis: respiratory disease (% inpatient stay)	9.71	7.98	-1.73†
Hospital diagnosis: disease digestive system (% inpatient stay)	9.55	10.39	0.84†
Hospital diagnosis: disease genitourinary system (% inpatient stay)	7.99	8.22	0.23
Hospital diagnosis: pregnancy, childbirth, contraception (% inpatient stay)	12.05	10.21	-1.84†
Hospital diagnosis: disease skin, subcutaneous tissue (% inpatient stay)	1.53	1.56	0.03
Hospital diagnosis: disease musculoskeletal system (% inpatient stay)	13.06	13.40	0.31
Hospital diagnosis: congenital abnormalities (% inpatient stay)	1.16	1.12	-0.04
Hospital diagnosis: conditions originating perinatal period (% inpatient stay)	2.32	1.66	-0.66†
Hospital diagnosis: injury and fractures (% inpatient stay)	7.23	7.00	-0.24
Hospital diagnosis: symptoms, signs, ill-defined conditions (% inpatient stay)	7.27	9.76	2.49†
Hospital diagnosis: not allocated and not disease related	6.79	9.49	2.69†

a. P-value of test of null of no change in mean of covariate. † $p<0.001$; ‡ $p<0.01$; * $p<0.05$

b. To save on space, diagnoses and hospital procedures are aggregated here. See appendix B for the complete lists used in the models

Table III Decomposition of changes in quantiles of log monthly expenditure between 1998 and 2004

	Observed values			Decomposition of change		
Quantile	1998	2004	Change	Change in covariates[a]	Change in conditional distribution[a]	Residual
Total acute health care expenditures						
0.20	0.45	0.00	-0.45	0.00[ns]	-0.45	0.00
0.30	1.16	1.12	-0.04	0.25	-0.28	-0.01
0.40	1.73	1.90	0.17	0.26	-0.09	0.00
0.50	2.31	2.60	0.29	0.26	0.03	0.00
0.60	2.94	3.26	0.32	0.24	0.08	0.00
0.70	3.56	3.87	0.32	0.20	0.12	0.00
0.80	4.18	4.51	0.33	0.15	0.17	0.01
0.90	5.04	5.31	0.27	0.07	0.20	0.00
Hospital and other secondary acute care expenditures						
0.50	0.56	1.18	0.62	1.20	-0.60	0.02
0.60	1.60	1.95	0.35	0.42	-0.08	0.01
0.70	2.40	2.66	0.26	0.33	-0.08	0.00
0.80	3.18	3.39	0.21	0.27	-0.06	0.00
0.90	4.22	4.36	0.14	0.13	0.01[ns]	0.00
Pharmaceutical expenditures						
0.30	0.55	0.34	-0.21	0.41	-0.55	-0.07
0.40	1.01	0.97	-0.04	0.15	-0.20	0.01
0.50	1.46	1.50	0.04	0.14	-0.09	-0.01
0.60	1.86	2.11	0.25	0.16	0.08	0.01
0.70	2.39	2.80	0.41	0.19	0.23	-0.01
0.80	3.09	3.60	0.51	0.15	0.36	0.00
0.90	3.91	4.40	0.49	0.11	0.38	0.01

Note: Results are derived from the decomposition method defined by equation (2). The total change is derived from the left hand side of (2), the contribution of changes in covariates from the first term on the right-hand side and the contribution of change in the conditional distribution from the second term on the RHS. Bootstrap standard errors were calculated. The decomposition results (5^{th} and 6^{th} column) were significant at 5% or less, with the exception of those indicated by superscript ns.

Table IV Decomposition of changes in quantiles of log monthly positive expenditures between 1998 and 2004

	Observed values			Decomposition of change		
Quantile	1998	2004	Change	Change in covariates	Change in conditional distribution	Residual
Total acute health care expenditures						
0.10	0.84	1.08	0.24	0.11	0.12	0.01
0.20	1.40	1.69	0.29	0.20	0.09	0.00
0.30	1.85	2.26	0.41	0.25	0.15	0.01
0.40	2.34	2.80	0.46	0.28	0.18	0.00
0.50	2.86	3.31	0.45	0.27	0.18	0.00
0.60	3.37	3.79	0.42	0.25	0.17	0.00
0.70	3.87	4.27	0.40	0.22	0.18	0.00
0.80	4.44	4.82	0.38	0.20	0.19	-0.01
0.90	5.23	5.54	0.31	0.11	0.19	0.01
Hospital and other secondary acute care expenditures						
0.10	1.22	1.31	0.09	0.09	0.02^{ns}	-0.02
0.20	1.57	1.71	0.14	0.16	-0.01^{ns}	-0.01
0.30	1.99	2.11	0.12	0.16	-0.04	0.00
0.40	2.38	2.49	0.11	0.17	-0.06	0.00
0.50	2.78	2.87	0.09	0.18	-0.08	-0.01
0.60	3.17	3.26	0.09	0.15	-0.05	-0.01
0.70	3.62	3.70	0.08	0.12	-0.03	-0.01
0.80	4.21	4.25	0.04	0.05	-0.01^{ns}	0.00
0.90	5.30	5.27	-0.03	-0.08	0.05	0.00
Pharmaceutical expenditures						
0.10	0.60	0.75	0.15	0.04	0.11	0.00
0.20	0.95	1.14	0.19	0.07	0.12	0.00
0.30	1.30	1.54	0.24	0.12	0.12	0.00
0.40	1.61	1.95	0.34	0.16	0.18	0.00
0.50	1.94	2.42	0.48	0.20	0.27	0.01
0.60	2.35	2.94	0.59	0.25	0.35	-0.01
0.70	2.86	3.50	0.64	0.24	0.41	-0.01
0.80	3.46	4.06	0.60	0.20	0.40	0.00
0.90	4.14	4.67	0.53	0.14	0.40	-0.01

Note: As for Table III.

Table V Stability tests the effect of covariates on the probability of positive expenditure and conditional mean of (log) positive expenditure between 1998 and 2004

	Hospital and other secondary care				Pharmaceuticals			
Effect on	Pr(lnHCE>0)		E[HCE|HCE>0]		Pr(HCE>0)		E[HCE|lnHCE>0]	
Group of determinants	Test statistic[a]	Change in effect (b_{04}-b_{98})	Test statistic[a]	Change in effect (b_{04}-b_{98})	Test statistic[a]	Change in effect (b_{04}-b_{98})	Test statistic[a]	Change in effect (b_{04}-b_{98})
Age and sex	394†	+	8.84†	+	1236†	b.	24.25†	b.
35-56 years	149†	+	6.71†	+	207†	+	10.99†	+
>65 years	256†	+	4.45†	+	302†	+	11.83†	+
Deceased	9‡	–	0.09	not significant	5*	–	0.05	not significant
Co-residence status	10*	f1.	0.22	not significant	10*	f2.	2.51	not significant
Work disabled	7	+	2.67	not significant	86†	+	30.24†	+
Cause-of-Death	10	+	0.68	not significant	9	not significant	1.70	not significant
Nr of first policlinic visits	NA	NA	68.93†	+	NA	NA	97.97†	+
Nr of day care admissions	NA	NA	6.70‡	–	NA	NA	3.11*	g.
Nr of overnight admissions	NA	NA	7.58†	c.	NA	NA	1.22	not significant
Length of stay	NA	NA	1.59	not significant	NA	NA	7.68†	+
Type of hospital	NA	NA	0.61	not significant	NA	NA	NA	NA
Hospital procedures	NA	NA	2.30†	d.	NA	NA	NA	NA
Hospital diagnosis	NA	NA	3.38†	e1.	NA	NA	3.59†	e2.

Pr(HCE>0) modelled by probit; E[lnHCE/HCE>0] by OLS

a A model with a full set of time interactions is used to test the joint significance of a change in the effect of a (group of) covariate(s) on expenditures over time. Null of change in effect (group of) covariate(s) on the probability to use (χ^2-statistic) or conditional mean expenditures (F-statistic); † p<0.001; ‡ p<0.01; * p<0.05. A positive (negative) change indicates higher (lower) conditional treatment costs associated with the covariate, ceteris paribus

b Average treatment costs by age increased, except for females aged 15-44 which is due to the removal of oral contraceptives

c Effect of one overnight admission fell but remained positive; the effect of more than one overnight admission became more positive

d Increase in average treatment costs for: surgery male genital organs, obstetric surgery; Reduction in average treatment costs for: surgery urinary ways and bladder, PT(C)A, diagnostic endoscopy lower gastrointestinal, other diagnostic procedures

e1 Increase in average treatment costs for: infectious disease, colorectal cancer, other malignant neoplasm, mental and behavioral disorders, heart failure, acute respiratory infections, asthma and COPD, rheumatoid arthritis; Reduction in average treatment costs for: diseases of the skin and subcutaneous tissue, conditions originating in the perinatal period, other not allocated and not disease related.

e2 Increase in average treatment costs for: osteoarthritis, dorsopathy, conditions originating in the perinatal period; Reduction in average treatment cost for: mental and behavioral disorder, eye and ear disorders, asthma and COPD, other respiratory disease, digestive disease, pregnancy childbirth and contraception, sympthoms signs and ill-defined conditions, other not allocated and not disease related.

f1 The effect of being institutionalized (ref = individuals living alone) on the probability to use pharmaceuticals decreased, e.g. became more negative.

f2 The additional probability to use pharmaceuticals for couples alone compared to individuals living alone decreased over time but remained positive.

g Positive effect of having one day care admission fell, positive effect of having more than one day care admission increased.

Appendix A Description of covariates

Variable	Description	Data source[a]
Dependent variables	The dependent variables comprise spending on acute health care services covered by the basic benefit package.	Vektis
log acute HCE	Logarithm of average monthly spending on hospital and other secondary acute care (excluding paramedical care), pharmaceuticals, obstetrics and maternity care, transport and devices. Spending is first corrected for inflation and Baumol's disease, then logarithmically transformed.	
log hospital spending	Logarithm of average monthly spending on hospital and other secondary acute care (excluding paramedical care).	
log pharmaceutical spending	Logarithm of average monthly spending on outpatient pharmaceuticals	
Age and sex	Dummy categories: females 0-14 (reference category), females 15-24, females 25-34. females 35-44, females 45-54, females 55-64, females 65-74. females 75-84, females 85+, males 0-14, males 15-24, males 25-34. males 35-44, males 45-54, males 55-64, males 65-74. males 75-84, males 85+.	GBA
Coresidence status	Co-residence status on January 1st; dummy categories: living alone (reference category), couple alone, other private household, institutionalized.	GBA
Work disabled	Indictor: individual received work disability benefits	Vektis
Number of first policlinic visits	Defined as the number of different policlinic specialists an individual visited during the year; dummy categories: none (reference category, 1 first policlinic visit, 2 first policlinic visits, 3 or more policlinic visits	Vektis
Number of day care admissions	Number of day care admissions during the year; dummy categories: none (reference category), 1day care admission, 2 or more day care admissions	LMR
Number of overnight admissions	Number of overnight admissions during the year; dummy categories: none (reference category), 1 overnight admission, 2 or more overnight admissions	LMR
Length of stay	Number of inpatient admission days and its square	LMR
Hospital diagnosis	*Inpatient* hospital diagnosis. 39 indicators; coded according to the International Classification of Disease 10th version (ICD-10). See appendix C for an overview of the selected diagnoses	LMR
Hospital procedures[b]	Restricted to primary procedures during *inpatient* hospital stays. 47 indicators. See appendix B for an overview of the selected procedures.	LMR
Type of hospital[b]	Indicators for type of hospital individual was admitted to for inpatient hospital stay: general hospital, university hospital, other teaching hospital, specialized hospital	LMR
Deceased	Indicator: deceased within 5 years after the measurement year	DO
TTD	TTD in months (set at a maximum of 60 for survivors) and its square	DO
Cause-of-Death	10 dummies; measured by the ICD-10. External cause of death (reference category); neoplasm; endocrine, nutritional or metabolic disease; mental and behavioral disorder; disease of the nervous system or sense organs, cardiovascular disease; respiratory disease; digestive disease; disease of the genitourinary system; symptoms signs and ill-defined conditions, else.	DO

a. sickness fund records (Vektis); hospital registry (LMR), municipality register (GBA), national death registry (DO)

b. not included in pharmaceutical expenditure model

Appendix B Percentage of inpatients by procedures

	1998	2004	Change[a]
Neurosurgery	2.62	2.97	0.35‡
Surgery endocrine glands	0.21	0.19	-0.02
Surgery lense and eye	5.91	7.47	1.55†
Surgery ear	2.85	2.38	-0.46†
Surgery nose and sinuses	1.67	1.45	-0.22*
Surgery airways, tonsils and adenoid	4.70	3.66	-1.04†
Surgery heart and thoracic vessels	1.39	1.46	0.06
Surgery other vessels	1.14	1.37	0.23‡
Other surgery arteries	0.97	0.88	-0.08
Surgery spline, bone marrow, lymphatic system	0.41	0.36	-0.05
Surgery mouth	1.03	1.03	0.00
Surgery stomach and esophagus	0.44	0.57	0.13*
Surgery colon and intestines	0.98	1.17	0.19*
Surgery appendix	0.77	0.68	-0.09
Surgery rectum and anus	1.03	1.06	0.03
Surgery gall bladder, bile ducts, liver, pancreas	1.53	1.75	0.22*
Surgery abdominal hernia	2.06	2.02	-0.04
Other surgery abdominal wall, peritoneum	0.50	0.48	0.02
Surgery kidneys	0.28	0.30	0.02
Surgery urinary ways and bladder	1.22	1.43	0.21*
Surgery male genital organs	2.49	2.42	-0.07
Surgery female genital organs incl. curettage	4.69	4.59	-0.09
Obstetric surgery	5.14	4.81	-0.33
Surgery facial bones	0.25	0.22	-0.03
Surgery for fractures and luxations	1.88	1.74	-0.14
Other bone and joint surgery	9.34	9.55	0.22
Surgery soft tissue	1.77	1.78	0.01
Other surgery skeletal and muscular system	0.30	0.24	-0.06
Surgery mamma	2.05	1.82	-0.23*
Surgery skin	2.38	2.35	-0.03
PT(C)A	1.07	1.77	0.70†
Nonsurgical procedures obstetrics	1.62	1.57	-0.05
Nonsurgical procedures musculoskeletal system	0.48	0.52	0.04
Radiotherapy	0.18	0.13	-0.05
Chemotherapy	0.36	0.40	0.04
Other therapeutic or preventive procedures	3.67	5.09	1.42†
Biopsy	2.46	2.45	-0.01
Diagnostic endoscopy respiratory tract	0.84	0.75	-0.09
Diagnostic endoscopy upper gastrointestinal	0.90	1.25	0.35†
Diagnostic endoscopy lower gastrointestinal	1.61	3.13	1.52†
Diagnostic endoscopy urogenital tract	0.54	0.74	0.20‡
Diagnostic laparoscopy	0.69	0.46	-0.23†
Diagnostic arthroscopy	1.01	0.76	-0.25†
Other and unspecified diagnostic endoscopy	0.02	0.04	0.01
Diagnostic radiology	1.94	1.64	-0.30‡
Other diagnostic procedures	3.14	3.97	0.83†
No procedure	26.65	24.62	-2.03†

† $p<0.001$; ‡ $p<0.01$; * $p<0.05$

Appendix C Percentage of inpatients by diagnoses

	1998	2004	Change[a]
Infectious disease	1.39	1.24	-0.15
Colorectal cancer	0.52	0.58	0.06
Lung cancer	0.50	0.52	0.01
Breast cancer	0.72	0.63	-0.09
Prostate cancer	0.22	0.22	0.00
Other malignant neoplasm	2.39	2.60	0.21
Benign neoplasm	2.73	3.05	0.31*
Diabetes mellitus	0.70	0.73	0.03
Other endocrine, nutritional, metabolic disease	1.23	1.36	0.13
Disease of the blood (forming organs)	0.88	1.02	0.14*
Mental and behavioral disorders	1.6	1.23	0.07
Multiple sclerosis	0.25	0.23	-0.02
Epilepsy	0.48	0.33	-0.15‡
Eye disorders	5.91	7.48	1.57†
Ear disorders	2.82	2.30	-0.53†
Other disease nervous system/sense organs	1.96	2.30	0.33‡
Coronary heart disease (CHD)	4.52	4.05	-0.47‡
Heart failure	1.35	1.27	-0.08
Stroke	1.60	1.83	0.23*
Other cardiovascular disease	6.19	6.84	0.65†
Acute respiratory infections	1.93	1.75	-0.18
Asthma and COPD	1.64	1.22	-0.42†
Other respiratory disease	6.46	5.28	-1.18†
Disease of the digestive system	9.55	10.39	0.84†
Disease of the genitourinary system	7.99	8.22	0.23
Pregnancy, childbirth and contraception	12.05	10.21	-1.84†
Disease of the skin and subcutaneous tissue	1.53	1.56	0.03
Rheumatoid arthritis	0.25	0.35	0.10*
Osteoarthritis	1.85	2.51	0.66†
Dorsopathy	3.03	2.70	-0.33*
Other disease of the musculoskeletal system	8.21	8.15	-0.06
Congenital abnormalities	1.16	1.12	-0.04
Conditions originating in the perinatal period	2.32	1.66	-0.66†
Hip fracture	1.01	0.91	-0.09
Other fracture	1.42	1.49	0.07
Injury	4.92	4.73	-0.20
Symptoms, signs and ill-defined conditions	7.27	9.76	2.49†
Cancer not allocated	0.42	0.43	0.01
Other not allocated and not disease related	6.79	9.49	2.69†

† $p<0.001$; ‡ $p<0.01$; * $p<0.05$

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