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# The impact of supply-driven variation in time to death on the demand for health care

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## Abstract

Many high-income countries have successfully reduced hospital mortality in several acute health conditions. We test the hypothesis that variation in the supply of care directed to saving the life of individuals with a health shock may result in increasing the demand for health care as individuals are likely to contribute to the demand after surviving the health shock. We examined repeated cross-sections of individuals exposed to an AMI or a stroke over a time window of ten years in Denmark. Hospital survival probabilities in the interval 0-30 days from the shock are used as an indicator of the supply, while individual health care expenditure in the interval 31-365 days is used as an indicator of the demand. We find the demand is highly elastic to supply-driven variation in time to death. Results are robust to a placebo test on individuals exposed to the shock without entering time to death.

**JPL:** 110

**Keywords:** Health care demand, Hospital quality of care, Time to death

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## 1 Introduction

The rapid growth of the health care expenditure (HCE) is jeopardising the financial stability of the national health system in many high-income countries. A large body of studies have investigated the drivers of the increasing HCE by examining demand factors such as age, morbidity, and time to death (TTD) (Rodriguez Santana et al., 2020). Numerous evidence show that a large share of total health care consumption occurs in the last year of life, suggesting that TTD could be a key driver of HCE (Breyer and Lorenz, 2020; de Meijer et al., 2011; Howdon and Rice, 2018; Karlsson et al., 2016; Seshamani and Gray, 2004; van Baal and Wong, 2012; Wong et al., 2011). Most of the existing studies examine variation in TTD that stems from the characteristics of the demand for health care, namely variation in proximity to death due to age, morbidity, genetic endowment and random shocks (Breyer and Lorenz, 2020). However, variation in TTD is also produced by variation in the supply of care, for instance improvements in the quality of care and the introduction of new technologies and medical practices have made possible to reduce mortality from many health conditions over the past decades (Cutler and McClellan, 2001; OECD, 2017; Skinner et al., 2006). Supply-driven variation in TTD may have an important impact on HCE that adds to demand-driven variation and may point to a different direction. For instance, an emerging part of the demographic literature show that population increments in longevity are accompanied by a compression of morbidity, namely increments in life expectancy are followed by a relatively constant number of years lived with morbidity that are shifted forward to older age (Fries et al., 2011). Under such a scenario, increasing longevity may result in reducing HCE over time as the high demand for health care associated with the end-of-life is shifted forward in time (Breyer et al., 2015; Geue et al., 2014; van Baal and Wong, 2012). In contrast, technological progress directed to extending life expectancy after a health shock may result in increasing HCE if individuals do not fully recover and continue to contribute to the demand for health care after the shock (Gruenberg, 2005; Laudicella et al., 2013; Laudicella et al., 2018). In this case, supply-driven variation in TTD may have an effect on the demand of care that goes in the opposite direction to longevity and may become increasingly relevant for an aging population as the risk of a health shock increases with age. As technological progress continues to make considerable advances in medicine reducing mortality from several diseases, it is important to produce evidence on the impact that supply-driven variation in TTD may have on the demand for health care and HCE.

This paper studies the impact of supply-driven variation in TTD on the demand for health care of individuals exposed to a health shock. Our hypothesis is that variation in the supply of care directed to saving the life of individuals exposed to a health shock, namely postponing their TTD, may result in increasing the demand for health care as individuals are likely to contribute to the demand after surviving the health shock. We test our hypothesis by examining repeated cross-sections of individuals who experience a health shock due to an

acute myocardial infarction (AMI) or a stroke in different calendar years and who are treated according to the level of quality, technology and medical practice offered by their admission hospital at the time of the shock. Hospital risk-adjusted 30-day survival probabilities are used as an indicator of variation in TTD that stem from variation in the supply of care. Short-term mortality after an AMI or a stroke has declined dramatically over the past decades in many high-income countries thanks to improvements in the quality of care and the introduction of new technologies and medical practices (Cutler and McClellan, 2001; OECD, 2017; Skinner et al., 2006), thus individuals exposed to a health shock in late calendar years have better chances of surviving than individuals exposed to a similar shock in early calendar years. Moreover, improvements in quality of care occurs at different speed across different hospitals resulting in different trajectories in their risk-adjusted survival probabilities. Our identification strategy is based on variation in quality of care within hospitals over time after controlling for hospital time-invariant heterogeneity and individual heterogeneity.

A long-standing issue in the literature is that the effect of TTD on utilisation of care is likely to be endogenous due to circularity in the relationship between these two variables (Felder et al., 2010; Howdon and Rice, 2018; Kolodziejczyk, 2020), i.e. individuals may be able to postpone their TTD by using more resources and use more resources as they postpone their TTD. Hence, postponing TTD might be the *result* of using more resources to save the life of patients, rather than its *cause*. We address this issue by examining variation in TTD and variation in utilisation of care in two separate time intervals. We measure the former in the interval 0-30 days after the health shock and the latter in the interval 31-365 days. In our study framework, variation in utilisation of care occurs *after* variation in TTD and thus it cannot be its cause. However, endogeneity issues may originate from potential correlation between utilisation of care in these two separate time intervals due to unmeasured heterogeneity. For instance, an increment of resources allocated to some hospitals may result in increasing both their 30-day survival probabilities and the health services delivered to their patients in the year following the health shock. We test for this potential source of bias by repeating the analysis in a placebo subgroup of individuals who experience the health shock without entering TTD and find evidence supporting the validity of our identification strategy.

The study is based on a rich dataset on the population of residents in Denmark age 50+ exposed to a health shock due to an AMI or a stroke and using inpatient and outpatient hospital services between 2005 and 2014. The dataset includes very accurate information on individual's morbidity and DRG tariffs that are used to reimburse hospital services. The Danish National Health System (DNHS), which is free of charge at the point of use and offers a universal coverage to its population, provides an ideal setting to our study as variation in utilisation of care is not confounded by variation in ability to pay or access to health insurance.

We find evidence that the demand of health care is highly elastic to variation in supply-driven TTD. A one percent increment in hospital 30-day survival probabilities results in a 6.3% increment in individual HCE after an AMI and 5.4% after a stroke. Such a response is three times greater in a subgroup of patients who enter TTD in the medium term after the shock, while it is absent in the placebo. Our study suggests that the hospitals' success in saving the life of individuals suffering a health shock can be an important driver of the demand for health care. This may have important implications for policy makers in planning the targets and resources for the national health system. In particular, health policies aiming at reducing the mortality of high-risk health conditions by improving the quality of care should be accompanied by additional resources to address the demand of care of an increasing number of individuals surviving such conditions (OECD, 2010, 2017; OECD/WHO, 2019).

## **1.2 Literature review**

A large body of studies examined the impact of TTD on the demand of care examining variation in TTD that stems from variation in demand characteristics, such as individual demographic, socioeconomic and health related characteristics (Breyer and Lorenz, 2020; de Meijer et al., 2011; Howdon and Rice, 2018; Karlsson et al., 2016; Seshamani and Gray, 2004; Shang and Goldman, 2008; Wong et al., 2011). Strong evidence shows that TTD and morbidity are key determinants of the demand of health care, whereas aging captures the effect of these factors when omitted from the analysis; hence aging has been labelled a red herring in this literature (Breyer and Lorenz, 2020; Felder et al., 2010; Werblow et al., 2007; Zweifel et al., 1999). More recent studies based on administrative hospital data reinforce the case for morbidity as one of the main drivers of utilization of care suggesting that TTD may capture the effect of unmeasured morbidity, although the effect of TTD remains statistically significant after controlling for measured morbidity in the regression (de Meijer et al., 2011; Howdon and Rice, 2018; Moore et al., 2017; Wong et al., 2011). The identification of the effect of TTD on the demand of care may be confounded by endogeneity from circularity in the relationship between these two variables, namely TTD may be influenced by utilisation of health care resources. Studies examining this issue suggest that the bias from the effect of utilisation of care on TTD is small as compared to the magnitude of the effect of TTD on utilisation, although the authors were not able to remove the bias completely by using instrumental variables approaches (Felder et al., 2010; Howdon and Rice, 2018; Kolodziejczyk, 2020). In particular, finding an instrument with a strong direct effect on TTD and no direct effect on utilisation of health care is quite a difficult task and poses a limitation on this type of estimation strategies.

Despite the great number of empirical investigations on the impact of TTD on HCE, only few examined the impact of variation in TTD that stem from variation in the supply of care. Laudicella et al. (2018) use a negative binomial model to examine the impact of variation in hospital mortality rates on emergency

readmissions in patients of the English National Health System. The authors find that improvements in hospital mortality rates can explain part of the growth in utilisation of emergency care over a period of ten years. A second study investigates the impact of technological progress on HCE by examining repeated cross-section of individuals exposed to a health shock over a ten-year window. By using an indirect approach, the authors capture unobservable technological progress from the residuals of a three-part regression model and decompose its effect on HCE in the part that is due to postponing TTD and the part that is due to increasing cost of resources. The present study contributes to this growing literature in several ways. First, we use a direct approach and measure supply-driven variation in TTD by using a direct indicator capturing hospital 30-day survival probabilities. Second, we address the potential issue of circularity in the relationship between TTD and utilisation of care by studying these two processes in two separate intervals of time and supporting our identification strategy with a placebo test. Finally, we examine the impact of supply-driven variation in TTD on the whole demand for inpatient and outpatient care in Denmark, thus extending the evidence on emergency admissions in England.

## **2 Methods**

### **2.1 A simple model of the demand for health care after a health shock**

The main theoretical model for the demand of health care is the human capital model proposed by Grossman, which describe the individual's optimal investment to maintain her health capital over the lifespan (Grossman, 2000). Unfortunately, such a model is unsuitable for describing the potential impact of postponing TTD that is driven by the supply of care and independent from individual's effort. Therefore, we sketch a simple model of the demand of health care to describe the hypothesis investigated in our empirical analysis. In formulating this model, we were directed by our specific application and institutional context characterised by free of charge access to acute care, universal health insurance coverage, and non-profit public providers.

We assume that individual demand for health care at time  $t$  is an increasing function of individual morbidity, age and decreasing function of expected TTD. The latter is a decreasing function of individual morbidity and age, and an increasing function of the supply of care. In turn, the supply of health care can be described as an increasing function of technological progress. The latter can be defined as the quality of care, the technology and medical practice available at time  $t$  in a hospital provider  $j$  following the broad definition of technological progress described in the literature (Chernew and Newhouse, 2011). Finally, we assume that technological progress increases with time at different speed for different hospitals as demonstrated by studies on variation in quality of care and diffusion of new technologies and medical practices (Skinner, 2011):

$$D_{it} = f(\text{Morbidity}_{it}, \text{Age}_{it}, E(\text{TTD}_{it}))$$

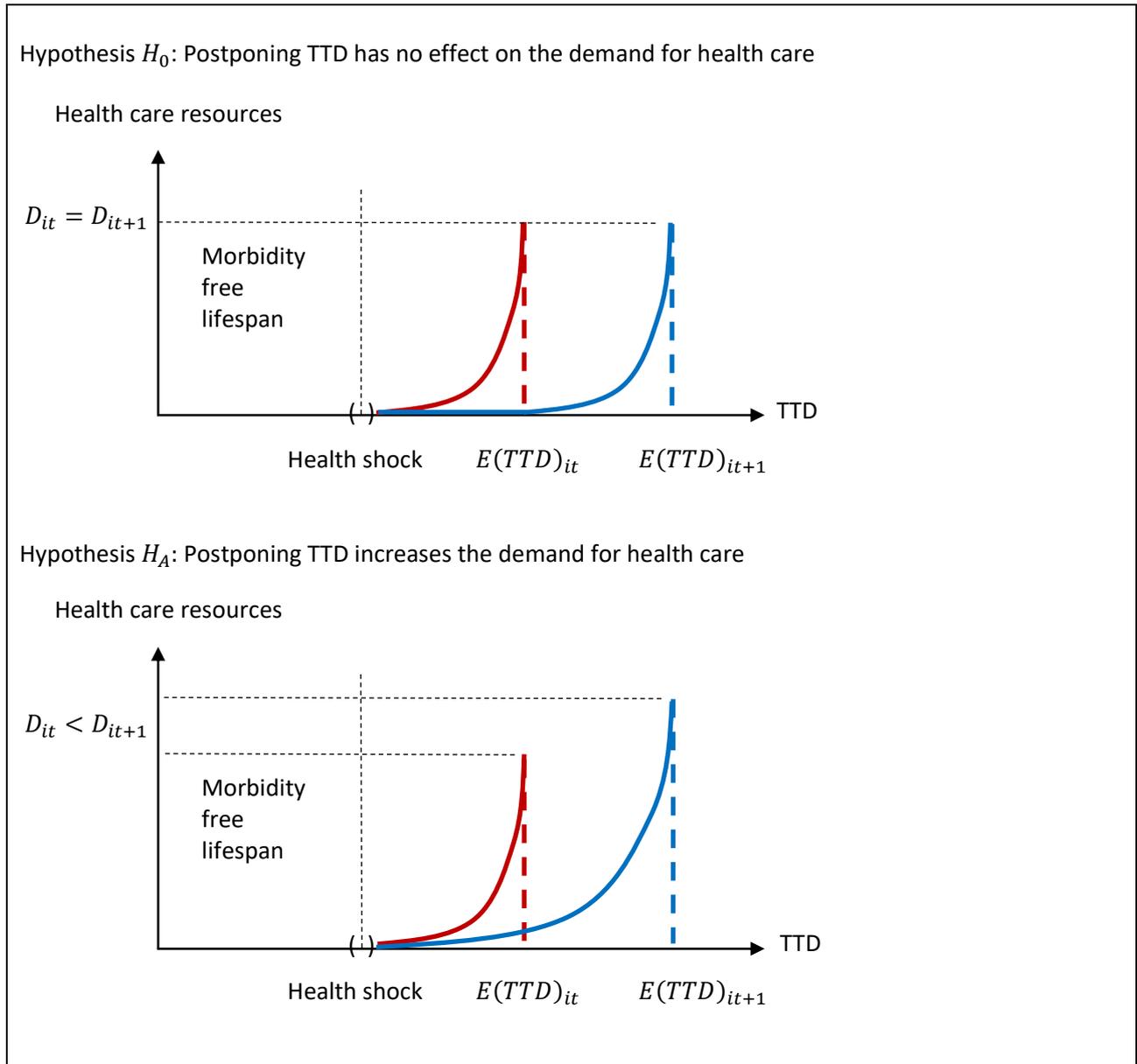
$$E(\text{TTD})_{it} = g(\text{Morbidity}_{it}, \text{Age}_{it}, S_{jt})$$

$$S_{jt} = h(\text{Tech}_{jt}) \text{ with: } \text{Tech}_t < \text{Tech}_{t+1} \text{ and } \text{Tech}_j \neq \text{Tech}_{-j}$$

Under this simple framework, technological progress is exogenously determined. For instance, technological progress in hospital  $j$  at time  $t$  may be determined by the hospital's preferences for new technologies and medical practices and its ability to deliver quality of care. The supply of care has an effect on TTD of individual  $i$  by providing the best treatment available in hospital  $j$  at time  $t$  according to the level of technological progress achieved by that particular hospital at that time. Although this simple framework is not generalisable to all cases, it captures the main characteristics of the demand and supply of care in a universal health care system for individuals exposed to a health shock, who are the subject of our empirical investigation.

We can now use the model above to describe our hypothesis. Figure 1 plots the relationship between TTD and the demand of care for an individual  $i$  after a health shock. If the health shock occurs in year  $t$ , she will receive the best care available at that time resulting in  $E(\text{TTD}_{it})$ , while if the same health shock occurs in year  $t+1$ , she will receive the best care available at time  $t+1$  resulting in  $E(\text{TTD}_{it+1})$ . Technological progress is higher in  $t+1$  resulting in  $E(\text{TTD}_{it+1}) > E(\text{TTD}_{it})$ . Under hypothesis  $H_0$ , supply-driven variation in TTD has no effect on the demand of care, namely utilisation of care due TTD is simply shifted forward in time and individual  $i$  consumes a similar total amount of resources whether she experience the health shock in year  $t$  or  $t+1$ , although she enjoys a longer life in  $t+1$ . In contrast under hypothesis  $H_a$ , supply-driven variation in TTD has a positive effect on the demand of care. This may occur if the health shock leaves a permanent mark on the individual prompting a higher demand of resources over a longer period of time after the health shock. It is worth noting that approaching TTD produces an exponential growth in the demand under both hypotheses, which is consistent with the predictions from studies on TTD. Also, it is important to notice that the amount of care devoted to save the life of the patient *during* the health shock, e.g. surgery during the first hospital admission following the shock, is excluded from the analysis so that variation in utilisation of care in Figure 1 does not include the cost of technological progress capable of postponing TTD.

**Figure 1.** Hypotheses



Note: TTD on the x-axis measures the time elapsed from a health shock occurring in calendar time  $t$  and in calendar time  $t+1$

## 2.2 Empirical model

In order to capture the effect of supply-driven variation in TTD on individual HCE, we use a two-stage estimation approach. In the first stage, we calculate an indicator of supply-driven variation in TTD. To this end, we estimate hospital-year risk-adjusted probabilities of surviving 30 days after a health shock in repeated cross-sections of individuals exposed to a health shock in different calendar years, i.e. our study population. Estimates are obtained from a linear probability model:

$$S_{i,0-30\text{ days}} = x_i\alpha + hospital_{jt} + \varepsilon_{ijt} \quad [1]$$

$S_{i,0-30\text{ days}}$  is the probability of surviving 30 days for the individual  $i$  exposed to a health shock in the year  $t$  and admitted in the hospital  $j$ .  $x_i$  includes control indicators for individual health, demographic, and socioeconomic characteristics that may affect individual survival probabilities.  $hospital_{jt}$  is a vector of hospital-year fixed effects capturing supply-driven variation in TTD across hospitals and over time. The linear probability model is robust to misspecification and allows for including a large number of covariates for individual characteristics and hospital-year effects. The model is consistent with our objective of estimating variation in 30-day survival aggregated at the hospital-year level after controlling for heterogeneity in case-mix at the individual level.

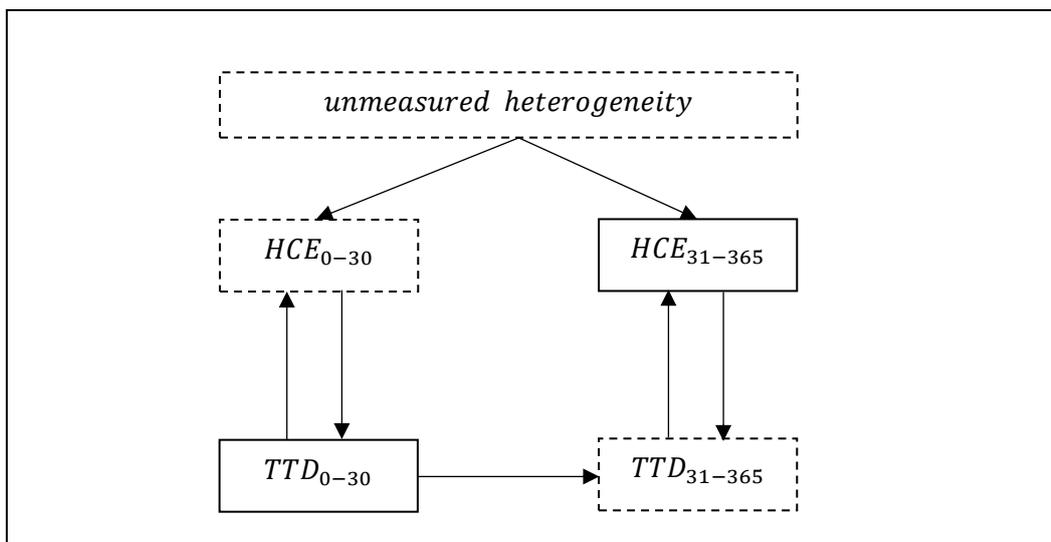
In the second stage, we estimate the elasticity of the demand for health care to supply-driven variation in TTD. To this end, we regress individual level HCE against hospital-year 30-day survival probabilities estimated from the first stage (Eq 1). The second stage model includes the same control indicators for individual heterogeneity used in the first stage to allow for variation in the demand that is due to differences in individual initial conditions at the time of the health shock. Also, it includes hospital fixed effects to control for time-invariant heterogeneity in the hospital of the first admission (i.e. the first stage hospital) that may influence the demand of care. For instance, hospital with a better performance in 30-day survival may offer more health services in the post-acute phase of care. The second stage model is estimated by using a simple log-linear regression that allows for skewness in the distribution of the individual HCE variable following a similar approach of other studies (Howdon and Rice, 2018):

$$\log(HCE_{i,31-365\text{ days}}) = x_i\beta + \gamma \log(\hat{S}_{jt,0-30\text{ days}}) + hospital_j + t + \omega_{ijt} \quad [2]$$

$HCE_{i,31-365\text{ days}}$  measures individual level health care expenditure in the interval 31-365 days after the health shock, i.e. our indicator of the demand for health care after the shock.  $\hat{S}_{jt,0-30\text{ days}}$  is a vector of hospital-year 30-day survival probabilities estimated from the first stage (Eq. 1), i.e. our indicator of supply-driven variation in TTD. The coefficient  $\gamma$  identifies the elasticity of the demand to supply-driven variation in TTD in individual exposed to a health shock. Hence, the effect of TTD on the demand of care is identified from variation in the supply of care within hospital over time after controlling for individual heterogeneity and hospital time-invariant heterogeneity. TTD is likely to be endogenous to HCE, i.e. individuals may be able to postpone their TTD as a result of using more health care resources, rather than using more resources as a

result of postponing their TTD. Our strategy to address this issue is two folds: first we use predicted survival probabilities at the hospital-year level, rather than observed survival probabilities at the individual level. While the latter are more sensitive to the specific resources consumed by the individual, the former depend on the quality of care, technology, and medical practice in the admission hospital. Second, we measure variation in survival in the interval 0-30 days after the health shock and variation in utilisation of care in the interval 31-365 days after the health shock. Hence, variation in TTD captured by our model occurs before variation in utilisation of care, thus the latter cannot be the cause of the former. However, these two variables can be correlated due to unmeasured heterogeneity confounding our identification strategy. Figure 2 illustrates the potential relationships between the variables of interest:

**Figure 2. Relationship between time to death (TTD) and individual health care expenditure (HCE)**



Note: continuous margins indicate measured variables, while dashed margins indicate unmeasured variables. Arrows indicate the direction of the effect of each variable.

Variation in TTD in the interval 0-30 days after the shock has a direct effect on variation in TTD in the following interval 31-365 days, e.g. surviving in the interval 0-30 days improves the chance of surviving in the interval 31-365 days. The latter have a direct effect on utilisation of care in the interval 31-365 days as individuals have the option of consuming care if they survive up to that interval. Utilisation of care in the interval 31-365 days can also have a direct effect on TTD in the same time interval as consuming more resources may improve survival, but it cannot have a direct effect on TTD in the interval 0-30 days. However, unmeasured heterogeneity can potentially influence individual HCE in both the 0-30 and the 31-365 intervals, and thus TTD in the interval 0-30 days. For instance, hospitals offering higher quality of care could provide more health services after the health shock (time-invariant heterogeneity), or some hospitals may receive an increment

of resources that may affect both their survival rate and health services over time (time-varying heterogeneity). Although hospital fixed effects control for time-invariant heterogeneity in the second stage of our regression model, unmeasured heterogeneity could be correlated with hospital 30-day survival over time, hence confounding our identification strategy. We use a placebo to test this hypothesis and re-estimate our second stage model in a subsample of individuals who experience the health shock without entering TTD in the medium term after the health shock, i.e. in the first three years following the shock. Such individuals make a strong placebo as they are exposed to the same health shock and treated in the same hospitals-years as the rest of our study population (of which they are part of), but they do not experience TTD in the medium term following the health shock. Hence, we expect that variation in the demand of care in the placebo cannot be produced by marginal variation in supply-driven TTD, but it can be produced by unmeasured heterogeneity correlated with both hospital 30-day survival and demand of care over time.

### **3 Data and Institutional framework**

Our empirical application is based on individuals treated by the Danish National Health System (DNHS). The DNHS offers a universal coverage to residents in Denmark and access to primary and secondary care services free of charge at the point of demand. Secondary care, including elective and emergency outpatient and inpatient services, is provided by 21 large multi-service hospitals, which are non-profit public organisations serving a local population of 250,000 residents and managed by the five Danish Regions. Access to elective care is managed by General Practitioners (GPs), who are the gate keepers of the system, while emergency care is accessed by calling the Emergency Medical Coordination Centres, which sends an ambulance or direct the patients to the closest Emergency Department according to their assessment of the urgency of the call. Hospital services are reimbursed by the Danish Regions through a system of DRG tariffs centrally determined by the Department of Health on the basis of the average costs reported by hospitals every year. DRGs were initially introduced in 2003 as a tool to incentivize hospital productivity and in 2005 were officially adopted as a reimbursement system for hospital services.

We use data extracted from the Danish National Patient Register including hospital elective and emergency admissions and outpatient visits. The data include information on patient admission and discharge date, primary diagnosis and up to 20 secondary diagnosis reported using ICD-10 codes. Every hospital admission and outpatient visit attract a DRG tariff that we use to calculate the individual level HCE. All residents of Denmark are identified by a unique identification number that is used to follow them through the Danish National Patient Register and to link them to a number of other registers at the individual level. We linked data on date of death from the Register of Causes of Death, individual annual income from the Income

Statistics Register, and living alone status from the Central Person Register (see Thygesen et al. (2011) for an overview on Danish registries).

### *Study population*

Our study population includes individuals age 50+ experiencing an initial health shock in different calendar years over a ten-year window, from 2005 to 2014. A health shock is defined as experiencing an emergency admission to hospital for an AMI or a stroke. Both health conditions have gained significant increments in survival thanks to successful improvements in the quality of acute care over the period examined (OECD et al., 2017), hence they provide an excellent study population. Individuals are included in the study at the time of their initial health shock in a moving time window of five years, e.g. an individual enters the study in 2005 if she experiences a health shock in 2005 and no shocks in 2000-2004. We excluded individuals experiencing an emergency admission due to any health condition in the five-years to the health shock to reduce potential confounding effects from the supply of care in past periods. We follow each individual two years before and one year after her initial health shock and extract information on her HCE, date of death, health, and sociodemographic characteristics. Hence, our study population is constructed using data from 2000 to 2015 allowing for the identification of the initial health shock and the follow up period, although exposure to supply-driven variation in TTD is assessed between 2005 and 2014. The study population includes 32,193 individuals with a health shock due to AMI, and 40,465 due to stroke.

### *Dependent variable*

Our dependent variable is the individual HCE for hospital inpatient and outpatient services accessed by individuals in the interval 31-365 days after the health shock. HCE is measured by a system of DRG tariffs that the Danish Regions pay to hospitals on the basis of the services delivered to their patients every year. The DRG tariffs are updated every year to allow for changes in the cost of services; updates are based on the national average cost of each service calculated from the hospitals' cost returns. The DRG payment system was introduced in Denmark in 2003.

### *Control variables*

We use a large basket of indicators capturing individual health and sociodemographic characteristics to control for individual heterogeneity in the analysis. Health indicators include: age, gender, primary diagnosis and total secondary diagnoses, Charlson index measuring individual mortality risk, comorbidities for acute and chronic conditions affecting the mortality risk, including congestive heart failure, peripheral vascular disease, cerebrovascular disease, dementia, chronic obstructive pulmonary disease, rheumatoid disease,

peptic ulcer, liver disease, diabetes, and renal disease, cancer. Health indicators are calculated from hospital records at the time of the health shock and two years before the shock. Socioeconomic indicators include: individual income, living alone status, and migrant status. Individuals not living alone might receive informal care reducing utilisation of other types of care (de Meijer et al., 2011). Finally, we included indicators for individual HCE that was generated one and two years before the health shock to sharpen our controls for heterogeneity in the demand for health care.

### *Descriptive statistics*

Table 1 shows descriptive statistics for our study population of individuals exposed to a health shock from AMI and stroke between 2005 and 2014. About 10% of individuals exposed to an AMI and 16% exposed to a stroke die within one year from the health shock, while the share of patients who survive 3 years after the health shock is 83% for AMI and 75% for stroke. While all individuals exposed to the shock are included in our statistical analysis, the subgroup surviving three years after the shock is going to be used in our placebo test. With respect to socioeconomic characteristics, individuals with an AMI earn on average 28,620 euros per year, while individuals with a stroke earn 26,330 euros; migrants account for 6% of AMI and 4% of strokes; finally, 34% of individuals with AMI and 42% with a Stroke live alone and thus may not have easy access to informal care. In terms of demographic characteristics and morbidity, individuals in our study population are on average 68-70 years old, women are more prevalent in the stroke subgroup (44%) than in the AMI (31%), the most prevalent morbidities are diabetes, chronic obstructive pulmonary disease and cancer.

Figure 3 shows the individual HCE before, during and after the health shock. HCE reaches its peak in the interval 0-30 days after the health shock as this period includes the surgery and initial post-surgical treatment provided during the first hospital admission. We exclude expenditure produced in this period from the statistical analysis due to its potential endogeneity with TTD as argued in the method section. Individual HCE maintains high levels in the period of 31-365 days after the health shock both for AMI (9,679 Euros) and for stroke (8,020 Euros) as it is produced over a longer interval of time than the former.

Finally, Table 2 reports hospitals 30-day survival probabilities in patients admitted for an AMI or a stroke between 2005 and 2014. Over this period of time, the average hospital survival improved by 0.0317 probability points for AMI and 0.0223 probability points for stroke, which correspond to an improvement of 3.4% for the former and 2.5% for the latter.

## **4 Results**

Figure 4 shows the variation in hospital 30-day survival probabilities after adjusting for differences in hospitals' case-mix. Estimates are obtained from the first stage regression model described in the method

section and centred at the global mean. Each of the dots plotted in Figure 3 can be interpreted as the performance of a hospital in saving the life of its patients as compared with the mean survival probability in the first 30 days after the health shock. Most of Hospital 30-day survival probabilities range between +0.10 and -0.10 probability points with respect to the global mean and show an average positive increment over the years examined in this study, which is consistent with the positive time trend reported in Table 2. This is the result of improvements in the quality of hospital care for patients with AMI and strokes that have been reported elsewhere (OECD et al., 2017).

Table 2 reports estimated coefficients from the second stage regression model. Model 1 includes case-mix adjusted hospital survival probabilities estimated from the first stage, while Model 2 includes unadjusted hospital survival probabilities from the observed data as a sensitivity analysis. A one percent variation in the 30-day hospital survival results in a 6.3% increment in individual HCE in the interval 31-365 days from the health shock for AMI and a 5.4% increment for stroke. The response of HCE to supply-driven variations in TTD is highly elastic, suggesting that individuals who are able to postpone their TTD due to variation in supply of care contribute positively to the demand for health care in the medium term after the health shock producing an increment of HCE over this period. This supports our hypothesis on the effect of supply-driven variation in TTD on the demand of care and also provides evidence on the magnitude of the effect. It is worth noting that the increment estimated in Table 2 is the average effect in the whole population exposed to the health shock, including the subgroup of individuals whose TTD is effectively postponed as a result of marginal variations in the supply of care and the rest of the population whose TTD is unaffected, namely individuals who would have survived the health shock even if they were treated in the poorest quality hospital and individuals who would have died even if they were treated in the highest quality hospital. The identification of individual profiles whose TTD effectively responds to marginal variations in the supply would require data on specific quality of care, new technologies and medical practices adopted by hospitals over time, which unfortunately we do not have, and an assessment of their impact on health outcomes, which goes beyond the scope of the present study. However, a reasonable guess is that such a subgroup of the population includes individuals who enter TTD in the medium term after the health shock, since they share a high mortality risk that is likely to respond to marginal variation in the supply. Table 3 reports the results of the analysis in a subgroup of our study population entering TTD within three years from the health shock. In this subgroup, the response of individual HCE to supply driven variation in TTD is about three times greater than in the whole population both for AMI and stroke, with an elasticity of 20.1% and 13.4% respectively. Finally, Table 4 reports the result of our placebo test. We repeated the analysis in a subgroup of the population whose TTD is not expected to be affected by marginal variation in the supply, namely individuals who are likely to survive the health shock due to their low mortality risk, rather than due to variation in the

supply of care. To this end, we selected individuals who are alive three years after the health shock. Table 4 show no statistically significant effect of supply-driven variation in TTD in our placebo, also the effect is close to zero in magnitude. We interpret the result of the placebo test as evidence that unmeasured heterogeneity is not confounding our identification strategy as discussed in the method section.

The same basket of demographic and socioeconomic indicators is used to control for observable heterogeneity in the demand for health care in all model specifications reported in Table 2-4. In general, control indicators affect HCE in the expected direction, although they are likely to be correlated making difficult to assess their effect in isolation. For instance, the 15 morbidity indicators capture the mortality risk that is also captured by the Charlson index and by the indicator for the total number of diagnoses.

## **5 Discussion**

This study examines the response of the demand for health care to supply-driven variation in TTD. We studied repeated cross-sections of individuals exposed to a health shock from AMI and strokes in different calendar years and receiving their treatment according to the quality of care in the hospital of admission at that time of the shock. Hospital 30-day survival probabilities are used as an indicator of the variation in TTD that stem from the supply of care, while individual HCE for inpatient and outpatient care in the interval 31-365 days after the shock is used as an indicator of the demand for health care. We find that the demand is highly elastic to variation in supply-driven TTD with a one percentage increment in Hospital 30-day survival probabilities resulting in a 6.3% increment in individual HCE after an AMI and 5.4% after a stroke. We find that the demand response is about three times higher in the subgroup of the study population who enter TTD in the medium term after the shock as these individuals are likely to be directly affected by marginal variation in the supply. In contrast, we find no demand response in the placebo subgroup of individuals who experience the health shock, but do not enter TTD in the medium term after the shock. The latter are low risk patients whose TTD is not expected to be affected by marginal variation in the supply. The outcomes of the subgroup analysis mitigate potential concerns over unmeasured heterogeneity that might be correlated with both TTD and HCE over time and support our identification strategy based on hospital fixed effects and a large number of control indicators for heterogeneity in the demand for health care. We avoid potential circularity in the identification of the effect of TTD on HCE by studying TTD and HCE in two separate intervals of time. This is a long-standing issue that has not been fully addressed by instrumental variables approaches (Felder et al., 2010; Howdon and Rice, 2018; Kolodziejczyk, 2020). Our estimation strategy excludes the HCE produced in the short term after the health shock from the indicator of the demand for health care. The latter usually includes surgical and post-surgical treatment that are driven by variation in the supply of care, e.g. quality of care, new technologies and medical practice adopted by the admission hospital, rather than by variation in

the demand of care. In this respect, our estimation strategy is consistent with our objective of capturing variation in the demand of health care. Finally, our results are consistent with previous findings that the reduction in hospital mortality rates achieved over a period of ten years explains part of the increment in emergency admissions in the English NHS (Laudicella et al., 2018).

Hospital 30-day survival from AMI and strokes are a widely used indicators to measure multidimensional aspects of the quality of hospital care (Krumholz and Normand, 2008; Spertus et al., 2003). This includes the adoption of the most effective technologies and medical practices that go under the label of technological progress (Chernew and Newhouse, 2011), but also other aspects of quality of care such as the overall management and organisation of the hospital services and the employment of appropriate mix of nurses and doctors with the appropriate level of experience. Although these are all factors that may affect supply-driven variation in TTD, technological progress is usually considered the main driver of the dramatic improvement in life expectancy in many acute conditions over time, including cardiovascular diseases examined in this study (Skinner et al., 2006). Unfortunately, we are unable to identify the contribution of specific supply characteristics to hospital 30-day survival probabilities due to lack of data on specific technologies, medical practice and organisation of services adopted by the hospitals examined in this study.

The source of the variation in TTD is one of the main novelties of this study. While most of existing investigations examine variations in TTD driven by characteristics of the demand after fixing the supply of care, the present study examine variations in TTD driven by the supply of care after fixing the demand. Our predictions are consistent with previous studies showing an exponential growth in HCE as individuals approach their TTD (Breyer and Lorenz, 2020). However, our conclusions on the impact of postponing TTD are different. In particular, variation in TTD that is prompted by longevity is considered to have a reducing effect on HCE as individuals enjoy a longer lifespan free of morbidity and shift their TTD forward to future periods. Our study shows that supply-driven variation in TTD points to the opposite direction prompting an increment in the demand of care in the year following the health shock. This source of variation might be especially relevant in an aging population as the risk of a health shock increases with age. Combining the effect of longevity and supply-driven variation in TTD suggest that part of the reduction in the demand of care produced by the former may be offset by the additional demand created by the latter. It is possible that supply-driven variation in TTD affects only a relatively small subgroup of the population using acute hospital services, while increasing longevity is a widespread phenomenon affecting the population as a whole, hence the latter is likely to prevail over the former. Further studies should be devoted to assessing the relative impact of these two sources of variation on the demand of health care and HCE.

This study has relevant policy implications. In the past decades, many high-income countries have made remarkable progress in reducing the mortality of acute and chronic health conditions, such as cardiovascular diseases, pulmonary diseases and cancer (OECD, 2017). A wave of new health policies have contribute to leading this success by setting quality targets and monitoring systems, resources and incentives that supported improvement in the quality of care and adoption of more effective technologies and medical practice (OECD/WHO, 2019). Our evidence suggests that the demand for health care is highly elastic to variation in short term survival from acute conditions, such as AMI and strokes. Therefore, health policies aiming at reducing hospital mortality rates for patients with acute conditions should be accompanied by additional resources to address the increment in the demand that they may generate.

## 6 Ethics statement

The analysis uses previously collected and pseudonomised individual level data on access to health services. Ethics approval to conduct the study has been granted by Statistics Denmark (DST) and the University Research and Ethics Commission.

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**Table 1.** Descriptive statistics on the study population of individuals exposed to a health shock from an AMI or a Stroke between 2005 and 2014.

	Observations	AMI Mean	S.D.	Observations	Stroke Mean	S.D.
<i>Individual Health Care Expenditure (Euros)</i>						
0-30 days after shock	32,193	10170.89	9551.42	40,465	11060.96	11761.07
31-365 days after shock	32,193	9679.86	14958.29	40,465	8020.95	20266.94
366-730 days after shock	32,193	3271.80	8631.12	40,465	3160.90	8989.16
731-1095 days after shock	32,193	2958.51	8806.35	40,465	2762.79	8243.55
1-365 days before shock	32,193	1099.52	3765.10	40,465	1077.72	3650.34
366-730 days before shock	32,193	840.43	3135.24	40,465	818.96	2863.86
<i>Time to Death</i>						
share dying within 1 year from the shock	32,193	10.47%		40,465	16.03%	
share dying within 3 years from the shock	32,193	16.79%		40,465	24.33%	
<i>Socioeconomic characteristics</i>						
living alone	32,193	34.11%		40,465	41.93%	
migrant	32,193	5.66%		40,465	3.91%	
income (x1,000 Euros)	32,193	28.62	81.21	40,465	26.33	31.07
<i>Demographic characteristics</i>						
female	32,193	31.26%		40,465	44.90%	
age	32,193	68.07	10.73	40,465	70.31	10.82
<i>Comorbidities</i>						
total diagnoses	32,193	1.87	1.13	40,465	2.12	1.22
Charlson index	32,193	1.45	0.85	40,465	1.35	0.84
AMI	32,193	100.00%		40,465	1.05%	
congestive heart failure	32,193	12.02%		40,465	1.41%	
peripheral vascular disease	32,193	3.11%		40,465	2.51%	
cerebrovascular disease	32,193	2.17%		40,465	100.00%	
dementia	32,193	0.61%		40,465	1.93%	
chronic obstructive pulmonary dis.	32,193	3.87%		40,465	3.03%	
rheumatoid disease	32,193	1.27%		40,465	1.23%	
peptic ulcer	32,193	0.42%		40,465	0.34%	
liver disease (mild)	32,193	0.18%		40,465	0.25%	
liver disease (severe)	32,193	0.02%		40,465	0.04%	
diabetes	32,193	9.06%		40,465	7.74%	
diabetes complications	32,193	1.64%		40,465	2.60%	
renal disease	32,193	1.22%		40,465	0.66%	
cancer	32,193	3.39%		40,465	3.98%	
metastatic cancer	32,193	0.22%		40,465	0.49%	

**Table 2.** Hospitals average 30-day survival probabilities

	AMI	Strokes
2005	0.9274	0.8975
2006	0.9343	0.8958
2007	0.9351	0.9089
2008	0.9493	0.8995
2009	0.9381	0.9052
2010	0.9468	0.9068
2011	0.9473	0.91
2012	0.9543	0.917
2013	0.9555	0.9277
2014	0.9591	0.9198
Increment from 2005 to 2014	0.0317	0.0223

**Table 3.** Effect of supply-driven variation in time to death on individual health care expenditure in repeated cross-sections of individuals exposed to a health shock between 2005 and 2014.

VARIABLES	AMI		Stroke	
	Model 1	Model 2	Model 1	Model 2
risk-adjusted 30-day survival probability in the hospital of admission after the shock (log)	6.3495*** (10.6713)		5.3778*** (6.9033)	
unadjusted 30-day survival probability in the hospital of admission after the shock (log)		5.1465*** (8.9656)		3.9345*** (4.4086)
living alone	-0.4174*** (-10.0100)	-0.4164*** (-10.0156)	-0.4730*** (-9.0338)	-0.4717*** (-8.9546)
migrant	0.1936*** (3.9243)	0.1910*** (3.9023)	0.1853*** (2.9786)	0.1840*** (2.9437)
2nd quintile of income distribution	1.0536*** (18.7637)	1.0476*** (18.6890)	1.1647*** (15.0830)	1.1604*** (15.1014)
3rd quintile	1.1803*** (19.3650)	1.1742*** (19.2062)	1.4388*** (15.1600)	1.4338*** (15.1850)
4th quintile	1.0897*** (22.0200)	1.0828*** (21.9938)	1.4240*** (15.1101)	1.4185*** (15.0662)
top quintile of income distribution	1.0050*** (18.8232)	0.9984*** (18.7787)	1.4985*** (12.4788)	1.4930*** (12.4747)
female	0.0136 (0.4476)	0.0130 (0.4256)	0.0927** (2.0666)	0.0912** (2.0338)
age	0.3926*** (13.2494)	0.3917*** (13.2534)	0.1763*** (7.2442)	0.1755*** (7.2408)
age squared	-0.0031*** (-13.7963)	-0.0031*** (-13.7995)	-0.0015*** (-8.4365)	-0.0015*** (-8.4361)
Charlson index	-0.2704 (-1.0153)	-0.2728 (-1.0292)	0.4010** (2.6793)	0.3885** (2.5995)
total diagnosis	0.0081 (0.4596)	0.0097 (0.5481)	0.1712*** (8.0810)	0.1696*** (7.8272)
Health Care Expenditure 1 year before the shock (log)	0.0486*** (10.3595)	0.0486*** (10.3407)	0.1023*** (12.7115)	0.1024*** (12.7723)
Health Care Expenditure 2 years before the shock (log)	0.0328*** (7.8405)	0.0328*** (7.8630)	0.0482*** (9.0363)	0.0482*** (9.0289)
15 comorbidities indicators	Yes	Yes	Yes	Yes
Calendar year dummies	Yes	Yes	Yes	Yes
Hospital fixed effects	Yes	Yes	Yes	Yes
Constant	-5.1022*** (-4.5551)	-5.1773*** (-4.6468)	0.6449 (0.8543)	0.1889 (0.2588)
Observations	32,193	32,193	40,465	40,465
R-squared	0.1231	0.1227	0.1113	0.1107

Notes: estimates from log-linear regression models of individual health care expenditure in the interval 31-365 days after the health shock; \*\*\* p<0.01, \*\* p<0.05, \* p<0.1; cluster robust standard error in parentheses.

**Table 4.** Heterogeneity analysis in a subgroup of individuals who enter time to death in the medium term after a health shock. Effect of supply-driven variation in time to death on individual health care expenditure.

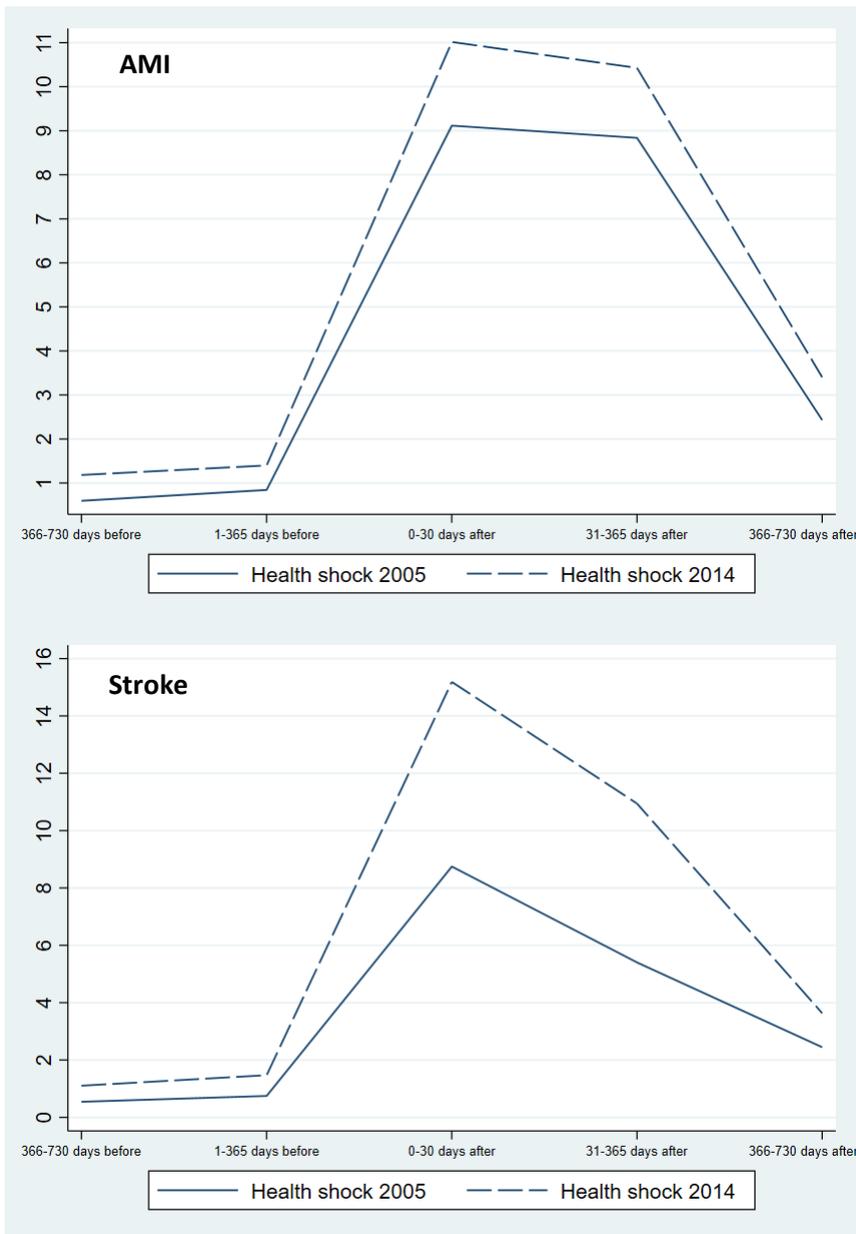
VARIABLES	AMI		Stroke	
	Model 1	Model 2	Model 1	Model 2
risk-adjusted 30-day survival probability in the hospital of admission after the shock (log)	20.1166*** (8.5728)		13.4149*** (7.8654)	
unadjusted 30-day survival probability in the hospital of admission after the shock (log)		16.7190*** (7.8222)		10.3213*** (5.6754)
living alone	-0.9022*** (-7.4612)	-0.9040*** (-7.4859)	-0.3789*** (-2.8984)	-0.3739*** (-2.8322)
migrant	0.7194*** (3.0514)	0.7191*** (3.0336)	0.4765** (2.3177)	0.4583** (2.2242)
2nd quintile of income distribution	2.4761*** (16.7598)	2.4644*** (16.4241)	2.2003*** (21.7619)	2.1933*** (21.8396)
3rd quintile	2.9395*** (21.4982)	2.9249*** (21.0711)	2.8543*** (18.5193)	2.8414*** (18.4628)
4th quintile	2.5704*** (16.6439)	2.5523*** (16.6655)	2.6037*** (17.3684)	2.5971*** (17.1194)
top quintile of income distribution	2.6092*** (10.2544)	2.5928*** (10.3203)	2.9356*** (20.1011)	2.9200*** (19.9123)
female	-0.0528 (-0.4582)	-0.0535 (-0.4619)	-0.3809*** (-3.0868)	-0.3884*** (-3.1551)
age	0.4891*** (7.9017)	0.4869*** (7.8782)	0.5965*** (11.5848)	0.5959*** (11.5623)
age squared	-0.0034*** (-8.0227)	-0.0034*** (-7.9631)	-0.0041*** (-11.7952)	-0.0041*** (-11.7504)
Charlson index	-1.2983*** (-25.0490)	-1.2808*** (-24.4210)	0.0835 (1.2647)	0.0872 (1.3251)
total diagnosis	-0.0140 (-0.2654)	-0.0110 (-0.2098)	0.3280*** (9.0769)	0.3260*** (8.9069)
Health Care Expenditure 1 year before the shock (log)	0.0611*** (2.7422)	0.0618*** (2.7713)	0.0690*** (5.2551)	0.0698*** (5.3105)
Health Care Expenditure 2 years before the shock (log)	0.0736*** (4.0949)	0.0735*** (4.0953)	0.0364*** (2.7640)	0.0361*** (2.7728)
15 comorbidities indicators	Yes	Yes	Yes	Yes
Calendar year dummies	Yes	Yes	Yes	Yes
Hospital fixed effects	Yes	Yes	Yes	Yes
Constant	-10.7145*** (-4.7673)	-10.9876*** (-4.8786)	-16.9591*** (-8.3192)	-18.0647*** (-8.8250)
Observations	5,352	5,352	9,728	9,728
R-squared	0.1814	0.1804	0.1668	0.1649

**Table 5.** Placebo test in a subgroup of individuals who do not enter time to death in the medium term after a health shock. Effect of supply-driven variation in time to death on individual health care expenditure.

VARIABLES	AMI		Stroke	
	Model 1	Model 2	Model 1	Model 2
risk-adjusted 30-day survival probability in the hospital of admission after the shock (log)	0.2737 (0.4179)		-0.4477 (-0.4746)	
unadjusted 30-day survival probability in the hospital of admission after the shock (log)		0.0626 (0.1093)		-1.2206 (-1.1823)
living alone	-0.0902*** (-3.2600)	-0.0902*** (-3.2645)	-0.2174*** (-5.2758)	-0.2176*** (-5.2662)
migrant	-0.0108 (-0.2075)	-0.0108 (-0.2069)	-0.0904 (-1.0802)	-0.0898 (-1.0732)
2nd quintile of income distribution	0.0249 (0.6175)	0.0246 (0.6060)	-0.0367 (-0.6764)	-0.0364 (-0.6743)
3rd quintile	0.0514 (1.4969)	0.0512 (1.4836)	0.0307 (0.5559)	0.0315 (0.5728)
4th quintile	0.0553* (1.7622)	0.0550* (1.7451)	0.0875* (1.7746)	0.0880* (1.7997)
top quintile of income distribution	-0.0136 (-0.3553)	-0.0139 (-0.3637)	0.1292 (1.3991)	0.1301 (1.4210)
female	-0.0903*** (-3.3372)	-0.0903*** (-3.3380)	0.0834* (1.8081)	0.0829* (1.7978)
age	0.2579*** (10.4078)	0.2579*** (10.4215)	-0.0587* (-1.7998)	-0.0586* (-1.7991)
age squared	-0.0020*** (-10.6576)	-0.0020*** (-10.6713)	0.0002 (0.6611)	0.0002 (0.6609)
Charlson index	0.0207 (0.4617)	0.0208 (0.4625)	0.2806*** (2.8182)	0.2787*** (2.8295)
total diagnosis	0.0392*** (3.1630)	0.0394*** (3.1937)	0.1024*** (4.4977)	0.1027*** (4.4833)
Health Care Expenditure 1 year before the shock (log)	0.0452*** (13.9292)	0.0452*** (13.9043)	0.1085*** (13.3727)	0.1084*** (13.3969)
Health Care Expenditure 2 years before the shock (log)	0.0154*** (4.6605)	0.0154*** (4.6547)	0.0467*** (9.0215)	0.0467*** (8.9777)
15 comorbidities indicators	Yes	Yes	Yes	Yes
Calendar year dummies	Yes	Yes	Yes	Yes
Hospital fixed effects	Yes	Yes	Yes	Yes
Constant	-0.5347 (-0.6921)	-0.5499 (-0.7136)	9.7835*** (9.8267)	9.5927*** (10.0837)
Observations	26,841	26,841	30,737	30,737
R-squared	0.0782	0.0782	0.0991	0.0992

Notes: estimates from log-linear regression models of individual health care expenditure in the interval 31-365 days after the health shock; \*\*\* p<0.01, \*\* p<0.05, \* p<0.1; cluster robust standard error in parentheses.

**Figure 3.** Individual health care expenditure before and after a health shock in 2005 and 2014



**Figure 4.** Risk-adjusted 30-day survival probabilities in the hospital of admission

