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Health expenditure and GDP growth

Exploring Granger-causality in Sweden

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Abstract: Health care expenditure has been growing rapidly in OECD countries and is expected to continue rising. However, the interaction between health spending and economic growth is not straightforward. Using annual data between 1970 and 2013, this study examines the relationship between per capita health care expenditure and per capita GDP in Sweden. We perform Granger-causality analysis with two-year lag of the natural logarithm of GDP, revealing unidirectional causality from GDP to healthcare expenditure. This suggests that economic growth serves as an enabler of increased health expenditure. Following this, we specify a linear regression model in attempt to determine the effect of GDP growth on health expenditure. We find that GDP remains significant after controlling for population growth, as well as proxies for overall health and age structure. However, due to limitations in our study, we refrain from drawing definite conclusions from this model.

Keywords: healthcare expenditure, GDP, Granger-causality, Sweden

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Introduction

The share of spending allocated to health varies greatly across the world. For instance, the health expenditure per capita in high-income countries typically exceeds 3000 USD, while the average is less than 100 USD in low-income countries (Kea et al., 2011). Partly due to the availability of data, extensive research within health economics has been devoted to the determinants of health expenditure in OECD countries, where healthcare costs have increased and are expected to continue to rise (OECD, 2013). In previous studies, a positive relationship between GDP and health expenditure per capita has been observed consistently. Early examples include Kleiman (1974), who studied income and per capita health expenditure in 16 countries, as well as Newhouse (1977), who examined the relationship for a different set of 13 countries. While it has long been established that a relationship exists between health expenditure and GDP, the direction of causality is not straightforward. There are several theoretical explanations for causality in either direction and empirical studies have found varying results. This paper aims to determine the direction of causality between healthcare expenditure and GDP growth in the case of Sweden specifically.

The Swedish Healthcare System

The Swedish healthcare system is largely based on 21 county councils (Anell, 2012). These councils are responsible for the provision of services in the vast majority of hospitals and primary healthcare facilities. The care of the elderly population, on the other hand, generally falls under the responsibility of municipalities. Although privately-owned facilities exist, the system is dominated by publicly funded service providers. Primary care forms the basis of the healthcare system and acts as a gatekeeper to more specialized care. Hospital care is available at both county and regional level. Patients with need for highly specialized care are typically referred to regional hospitals which house more advanced equipment. Counties are further divided into six medical care regions to allow for cooperation in delivery of tertiary medical care. Health expenditure as a share of GDP reached 11.3% in 2013, compared to the OECD average of 8.9% (OECD, 2016). Government spending as a share of total health expenditure was 84% in the same year. Looking back, Swedish healthcare expenditure per capita has risen from under 2 500 USD to almost 7 000 USD in under 20 years

(1995 - 2014), as demonstrated in Figure 1. The rise in healthcare costs is, of course, a subject of much concern, and cost containment in the healthcare sector has received much attention. Subsidy levels on prescription drugs are agreed upon between city councils and the central government. The Dental and Pharmaceutical Benefits Agency prepares cost-effectiveness analyses and evaluations of health needs to support decision-making regarding pricing of prescription drugs and reimbursement. While many hospitals are reimbursed on a diagnosis-related group basis, the compensation scheme is commonly designed to result in smaller payments when a certain volume of activity has been attained (Glenngård, 2014). This should limit incentives to increase activity beyond that level. Likewise, primary care providers are financially responsible for prescription costs, serving as an incentive for cost containment with regard to pharmaceutical expenditure (Glenngård, 2014).

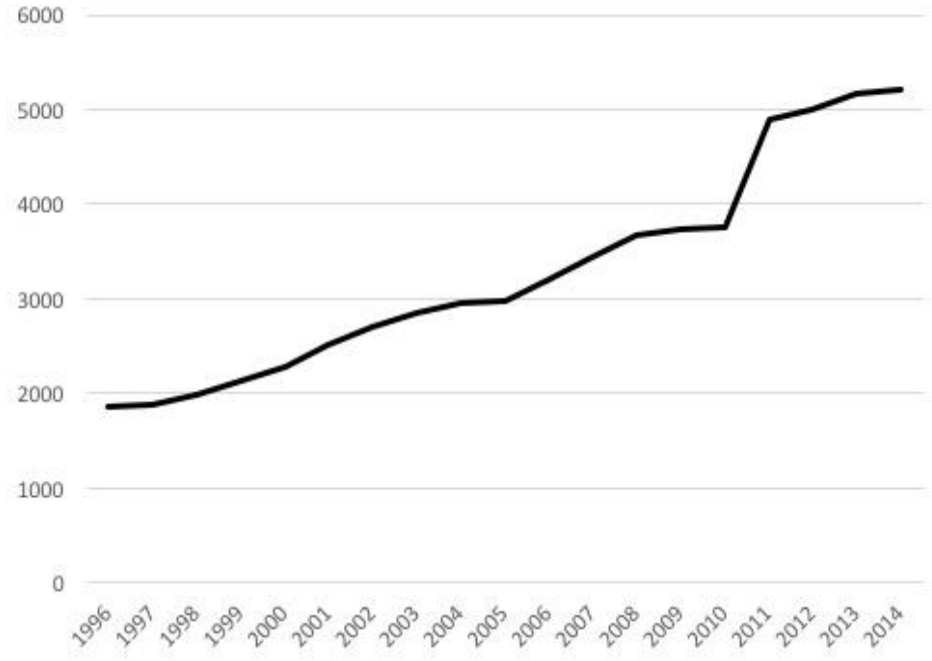

Figure 1: Healthcare Expenditure per capita (in current USD) in Sweden, from 1995 to 2014 (World Bank Database)

Theoretical Framework

The Health View

The concept of human capital was first presented by the economist and Nobel laureate Theodore W. Schultz (1961), but has since then been elaborated upon by numerous economists (Grossman 1972, Mirowsky 1998). The theory was developed as a reaction towards the oversimplification of factor labour which, together with capital, make up the two factors of productivity in a Cobb-Douglas production function. The term labour was thus replaced by human resource or human capital. This term was described as a stock of several interrelated factors including knowledge, competencies, health, culture, spirituality and humanity. These varying factors have been summarized and condensed into three: health capital, educational capital and capital humanitarian (Alexandru & Maria, 2012). Together, these constitute an individual's human capital stock. Considering human capital as a stock makes the concept of investments in stock, as well as the concepts of original stock and depreciation, relevant. An individual has, for any given moment, a baseline stock of human capital that the individual can make investments in (such as learning a new skill/competence through education) to increase this stock. However, as individuals lose their knowledge or competencies, the concept of a rate of depreciation of our human capital stock becomes significant. The human capital theory has had enormous impact globally, in fields ranging from public policy to individual firm management (Marimuthu et al., 2009).

When initially proposed, the human capital theory was primarily presented with respect to education as an explanation to varying income levels among individuals. It was, however, later expanded to include various other factors, including health. In 1972, the economist Michael Grossman presented a human capital model of the demand of health (Grossman, 1972, 2000). The model views health as a durable capital stock, with an inherited initial value, allowing investments and depreciation to occur. Grossman defined the output of this model as healthy time. Healthy time, in turn, is a necessary input for productivity. In the Grossman-model, health is both an input and output, and is thus effectively both demanded and produced by individuals. The output of health is desirable as individuals prefer being healthy to being sick, but is also demanded as an input or investment

commodity as it determines total amount of healthy time available. Healthy time available, in this context, is time not spent in an immobilized or ineffective state due to sickness. Available time will naturally affect utility as it can be spent on either market or nonmarket activities, depending on the individual's preference function (Grossman, 1972). Thus, according to the Grossman model, investments in human capital should lead to productivity growth. While this model was presented at the micro-level, it is relevant on the macro-level as well, seeing as investments in health and human capital represent important public policy decisions by governments worldwide, often with the hope of triggering growth (Oluwatobi et al., 2011; Baldwin et al., 2008). The Grossman model in its simplest form may be described as follows:

$$H_{t+1} = I_t - \delta_t H_t + H_t$$

where H_{t+1} is the next time period's health stock, H_t is this year's health stock, I_t is the investments made in health stock and δ_t is the depreciation rate of the current health stock. Grossman treats the depreciation rate as exogenous but dependent on age, implying that the health stock depreciates at a higher rate later in life. This H_t is then considered a vector of input in the variable TH_t, representing healthy time. Healthy time can be spent in productivity output or leisure time, as mentioned above. It may also be spent on further accumulation of human capital, for instance by receiving additional education or training. This results in a positive feedback loop between types of human capital (Mirowsky, 1998) and constitutes an indirect way in which increased health stock may increase productivity and growth.

Even before the Grossman model was conceptualized, Mushkin et al. had touched upon the implications on economic growth when regarding health as a human capital. Mushkin referred to this as the "health-led growth hypothesis" (Mushkin, 1962). In line with this, the World Health Organization's Commission on Macroeconomics and Health concluded in its report from December 2001 that "health is a creator and prerequisite of development" and that "extending the coverage of health services could save millions of lives, reduce poverty, spur economic development, and promote global security" (CMH, 2001). Health capital on a macro-level refers to the health of the population as well as the provision of healthcare services with respect to availability and quality.

In summary, the human capital theory suggests that investments in human capital and health capital enable and spur economic growth. This suggests that causality runs in the direction from healthcare expenditure to GDP growth. We refer to this as the Health View.

The Income View

There are indications that GDP should cause healthcare expenditure and not vice versa. First and foremost, without money available that can be spent on healthcare services, it is difficult to imagine an increase in health expenditure to occur. Economic growth, measured as GDP growth, enables higher tax revenue and increased potential for health investments. Also, on the individual level, increased income enables private expenditure on healthcare. This is sometimes collectively referred to as the Income View (Mehrara et al., 2011); income is a necessity for healthcare spending for governments and individuals alike.

Income-elasticity is the ratio of the percentage change in quantity demanded to the percentage change in income, and can take on either a positive or a negative value. For instance, an income-elasticity less than 0 indicates that higher income results in a decreased demand. These goods are often referred to as inferior goods. healthcare, however, has often been proposed to be a so-called luxury good based on some early empirical studies (Newhouse, 1977; Gerdtham & Jonsson, 2000). Luxury goods are goods characterized by income elasticities exceeding 1. For instance, an income increase of 20% would in such a case generate a demand increase exceeding 20%. The income elasticity of healthcare expenditure was debated extensively and studies reported conflicting results (Martín et al, 2011) until Thomas Getzen chose to examine individual and national causalities separately. Getzen concludes that "individual income elasticities are typically near zero, while national health expenditure elasticities are commonly greater than 1.0" (Getzen, 2000). In summary, if healthcare is a luxury good on the national level, the direction of causality should be from GDP to healthcare expenditure. Additionally, the coefficient estimate obtained from regressing expenditure on GDP should exceed one.

Previous Studies

Health capital and GDP

Knowles and Owen (1995) study the relationship between life expectancy as a proxy for health capital stock and per capita income, concluding that the relationship is stronger than that between educational human capital and per capita income. When examining the relationship between life expectancy and per capita GDP in OECD countries in the very long run, Swift (2011) observes a co-integrating relationship for all countries included. Further, the findings indicate that a 1% increase in life expectancy results in an average 5% increase in GDP per capita.

Determinants of Health Expenditure

In order to characterize the relationship between GDP and healthcare expenditure, a thorough understanding of other factors affecting healthcare expenditure is required. The earliest study on determinants of healthcare expenditure, based on cross-sectional data from 13 OECD countries, was carried out by Newhouse (1977). The study identified GDP as a key predictor of healthcare expenditure. Moreover, a number of studies have investigated the effect of an ageing population on healthcare expenditure. For instance, Gerdtham (1993) analysed cross-sectional data from Sweden between 1970 and 1985, arriving at the conclusion that only 13% of the increase in healthcare expenditure could be accounted for by demographic changes. However, Blomqvist and Carter (1997), looking at 24 OECD countries between 1960 and 1991, identify the number of people above the age of 65 as a main driver of rising healthcare costs. The effect of technology has also received considerable attention as a possible determinant of healthcare expenditure. A study by Newhouse (1992) used cross-sectional data on medical care expenditure in the US between 1960 and 1980, revealing a strong positive relationship between healthcare expenditure and medical technologies. Nevertheless, several issues pertaining to methodology in earlier studies have been highlighted, including not taking the possibility of non-stationarity in health and income data into account (Kea et al., 2011).

Empirical Evidence on Sweden

Erdil and Yetkiner (2009) employed panel data covering 75 low to high-income countries to test the direction of Granger-causality between GDP and health expenditure. A significant simultaneous causation between the two variables was found in 46 countries. In the case of unidirectional causality, causality from GDP to health expenditure was more common for low and middle-income countries, while the opposite was true for high-income countries. In Sweden specifically, Erdil and Yetkiner conclude that the direction of causality runs from real per capita health expenditure to real per capita GDP. Analysing data from 1960 to 1987, Devlin and Hansen (2001) reach the same conclusion for Sweden. However, a more recent study including data between 1975 and 2011 found the direction of causality to be from GDP to health expenditure in the case of Sweden (Sghari & Hammami, 2013).

Research Gap and Aim

Determining what influences healthcare expenditure is of great importance in order to make informed health policy decisions, and much research has been devoted to this purpose. The majority of empirical work published has included several countries in panel data analysis (Erdil & Yetkiner 2009; Blomqvist & Carter 1997; Xu et al. 2011). This provides statistical strength by including more observations. However, the regressions performed are on the aggregate level and thus, do not necessarily reflect the actual relationship in Sweden. Choosing a specific country for analysis allows for a regression model to be specified, should unidirectional causality be indicated. Such a regression model can confirm that the coefficient estimate significantly differs from zero and attempt to determine the magnitude of dependence. In addition to this, conflicting results have been found with regard to the direction of causality between GDP and health expenditure in the specific case of Sweden, warranting further study. Thus the aim of this study is two-fold:

- To examine the relationship and direction of Granger-causality between GDP growth and healthcare spending in Sweden between 1970 and 2013.

and,

- To determine the magnitude of this effect in the case of unidirectional causality.

Method

Variable list

Table 1 provides an overview of the variables included in the analysis. All data is for Sweden and for the range of years 1970 to 2013, with the exception of Sickness Benefits only available between 1999 and 2013.

Table 1: Variable Description

Variable Abbreviation	Variable Description	Source
LogGDP	Log Gross Domestic Product PPP per capita in USD at current prices	Organisation for Economic Co-operation and Development (OECD)
LogHCE	Log variable Healthcare Expenditure PPP per capita in USD at current prices	Organisation for Economic Co-operation and Development (OECD)
LogGDPlag1	Lagged 1 year LogGDP PPP per capita	Organisation for Economic Co-operation and Development (OECD)
LogGDPlag2	Lagged 2 years LogGDP PPP per capita	Organisation for Economic Co-operation and Development (OECD)
Sickness Benefits	Average days of sickness benefits, sickness compensation or activity compensation, per insured person (age between 16 and 64) and year	Swedish Social Insurance Agency's Database
Pop Growth (%)	% Population Growth	Organisation for Economic Co-operation and Development (OECD)
Elderly Pop	% of Population >65 years of Total Population	Organisation for Economic Co-operation and Development (OECD)

For all statistical analysis, a significance level of 5% was used.

Test for Unit Root - Dickey Fuller Test

Augmented Dickey-Fuller tests (ADF-tests) were performed to determine whether our variables followed a unit-root process. Unit-roots and unit-root tests have been a topic under discussion among economists for a long time (Cochrane, 1991; Nelson, 1982). Trend lines can either have a unit root or be trend stationary (indicated by a unit root of 0). This is expressed in two separate trend functions:

$$(1) \qquad y_t = \mu + y_{t-1} + u_t$$

$$(2) \qquad y_t = \beta_t \times y_{t-1} + u_t$$

where μ and β_t are constants defining the functions and u_t is the "noise" variable assumed to be independently identically distributed. Function (1) is non-stationary and function (2) is trend-stationary. The random walk of the variable u_t will cause drifts in the first function but not the second, as the second will return to its original trend over the time series, and is thus characterized by trend stationarity.

Despite much debate on the importance of unit roots (Cochrane, 1991; Nelson, 1982), it was tested for in this study as it determines which statistical methods and analysis can be performed. For instance, non-stationary trend lines must be tested for co-integration prior to a Granger-causality analysis or an OLS (Ordinary Least Squares) regression. Non-stationary trend lines may also give rise to "spurious regressions", meaning that regression between two variables results in a high R^2 despite the absence of a true relationship.

The null hypothesis in an ADF-test is that the trend series contains a unit root, while the alternative hypothesis is that the variable was generated by a stationary process, and so, is trend stationary.

Lag-order Selection Statistics

Pre-estimative lag order selection analysis was performed to determine appropriate lag levels. The statistical tests included the final prediction error (FPE), Akaike's information criterion (AIC), Schwarz's Bayesian information criterion (SBIC), and the Hannan and Quinn information criterion

(HQIC). These selection criteria are described extensively in the literature (Ivanov & Kilian, 2005). Previous literature suggests that the selection of lag order should be made based on these tests complemented by the theoretically presumed relationship between the variables (Ivanov & Kilian, 2005). Although some argue that healthcare expenditure can take up to six years to respond to other macro-variables (Getzen, 1990), there is a trade-off between lag-length and statistical power.

Granger-Causality Analysis

Causality in both directions gives rise to an endogeneity problem, ultimately resulting in inconsistent OLS estimates. However, even if bidirectional causality is the case, one may expect the feedback from GDP to health expenditure (and vice versa) to occur with a lag. In this context, a Granger-causality test (Granger, 1969) allows for the determination of direction of causality between the chosen variables representing economic growth and healthcare expenditure.

A variable, X_t is said to Granger-cause another variable, Y_t, if it can improve prediction of future values of Y_t. The null hypothesis is the following:

$$H_0\text{: ``}X_t \text{ does not Granger-cause } Y_t\text{''}$$

In this study, this translates to:

$$H_0\text{: ``}LogHCE_t \text{ does not Granger-cause } LogGDP_t\text{''}$$

To test this, y_t is first regressed on y_{t-i} and x_{t-j}(unrestricted model). Subsequently, y_t is regressed on y_{t-i} (restricted model). F-statistics are then used to see whether x_{t-j} enhances prediction of y_t :

$$\mathrm{F} = (\mathrm{N} - \mathrm{k})(\mathrm{ESS}_r - \mathrm{ESS}_{ur})/(\mathrm{q})(\mathrm{ESS}_r)$$

where N refers to the number of observations, k is the number of estimated parameters in the unrestricted regression, q is the number of parameter restrictions and ESS_r and ESS_{ur} are the sums of squared residuals in the restricted and unrestricted regression, respectively.

If this is the case (i.e. if the resulting F statistic exceeds the critical value), the null hypothesis is rejected, allowing one to conclude that X_t does indeed Granger-cause Y_t. This must be tested in both directions by also testing the null hypothesis that "Y_t does not Granger-cause X_t". That is,

H_0: "$LogGDP_t$ does not Granger-cause $LogHCE_t$ "

Linear Regression Model

Following the Granger test, we sought to determine the size of the coefficient of GDP per capita using an Ordinary Least Square-regression (OLS), and to test whether this coefficient is significantly different from zero. The initial model was formulated as follows:

$$LogHCE = \beta_0 + \beta_1 \times LogGDP + u$$

with u representing the error term. Other combinations were tested by adding lagged variables of GDP (1 year and 2 years).

Control Variables

Naturally, there may be other variables affecting GDP and or HCE. The original model was complemented by adding variables resulting in the following augmented model:

$$LogHCE = \beta_0 + \beta_1 \times LogGDP + \beta_2 \times LogGDPlag1 + \beta_3 \times LogGDPlag2 + \beta_4 \times SicknessBenefits + \beta_5 \times PopGrowth + \beta_6 \times ElderlyPop + u$$

Sickness Benefits: Apart from Granlund (2010), who finds that public healthcare expenditure has a negligible effect on sickness absence, there are few studies on the effect of healthcare expenditure on sickness absence or vice versa in Sweden. Theoretically, one may expect that individuals receiving sickness benefits consume more healthcare. At the same time, the absence of these individuals in the job market may influence GDP. Thus, sickness absence was introduced as a control variable in the model.

Elderly population: Earlier studies, most notably Blomqvist and Carter (1997), identified the number of people above the age of 65 as the main determinant of increasing healthcare costs. While later studies (Leu 1986; Hitiris & Posnett 1992) find that population age structure is not a significant determinant, we reason that an elderly population generally consumes more healthcare and should therefore be controlled for.

LogGDPlag: This follows from the Granger-causality test, suggesting that a GDP variable with lag 1 and 2 can help predict future values of healthcare expenditure per capita.

Population Growth: A growing population may lead to higher demand and necessitate increased healthcare expenditure. For instance, this may include healthcare costs pertaining to pregnancy and childbirth.

Heteroscedasticity

Before considering a regression model, the variables must be tested for heteroscedasticity. Recall that OLS requires the assumption that the variance of the error term is constant (homoscedasticity). This can be tested by the Breusch-Pagan / Cook-Weisberg test for heteroscedasticity (Stata, UCLA) with the null-hypothesis that the variance is homogeneous.

Multicollinearity

Testing for multicollinearity is also relevant as it can make the coefficient estimate of the regression model unstable. This results in inflated standard errors of coefficients. Multicollinearity occurs when two or more variables are near perfect linear combinations of each other. It can be tested for in Stata with the variance inflator factor (Stata, UCLA).

Results

The output from the Granger-Causality Test is presented in Table 2. A Prob>chi2 below 0.05 indicates significance and, consequently, rejection of the null-hypothesis of no Granger-causality between the variables. The null hypothesis for the top table (with lag of 1 year) cannot be rejected. However, one of the two null hypotheses in the bottom table can be rejected, indicating that *LogGDP* Granger-causes *LogHCE* with a lag of two years.

Table 2: Granger-causality tests between LogGDP and LogHealthcare Expenditure

Granger causality Wald tests				
Equation	Excluded	chi2	df	Prob>chi2
LogGDP	LogHCE	1.3029	1	0.254
LogGDP	ALL	1.3029	1	0.254
LogHCE	LogGDP	.99344	1	0.319
LogHCE	ALL	.99344	1	0.319

Granger causality Wald tests				
Equation	Excluded	chi2	df	Prob>chi2
LogGDP	LogHCE	1.8423	2	0.398
LogGDP	ALL	1.8423	2	0.398
LogHCE	LogGDP	9.1482	2	0.010
LogHCE	ALL	9.1482	2	0.010

Top table with Granger test with 1year lag; bottom table with 2 years lag.
df (degrees of freedom) denotes lag length.

Table A2 (see appendices) presents data from the ADF-test of unit roots. For both variables, the MacKinnon approximate p-value was less than 0.05, leading to rejection of the null hypothesis that variables are not stationary. Thus, both of the variables are henceforth regarded as stationary. Lag-order selection statistics can be found in Table A3 in appendices. All order-selection criteria for both variables of interest, *LogGDP* and *LogHCE*, showed a suitable lagged variable of one year. For reasons discussed previously regarding the selection of lag-orders, Granger-causality analysis was performed twice, once with lag 1 and once with lag 2. This choice was confirmed by Lagrange-Multiplier (LM) tests (see Table A5 in Appendices) of the residuals, which showed that the included lags were sufficient to avoid serial autocorrelation.

OLS regression outputs in Table 3 show the result of regressing *LogHCE* on several combinations of GDP variables. Lagged GDP variables only become significant when *LogGDP* (lag of 0 years) is excluded. In that case, however, both *LogGDPlag1* and *LogGDPlag2* become significant. R^2 is consistently high, with the lowest being 0.986. It is possible that this is due to inclusion of a large number of variables relative to the number of observations, especially when *Sickness Benefits* is included. The magnitude of coefficients generally varies between 0.8 and 1.7 for significant variables. Tests for heteroscedasticity and multicollinearity can be found in Tables A6 and A10 in the Appendices.

Table 3: OLS regressions with dependent variable *LogHCE*

VARIABLES	(1) LogHCE	(2) LogHCE	(3) LogHCE	(4) LogHCE	(5) LogHCE	(6) LogHCE	(7) LogHCE
LogGDP	1.246***	0.987**	1.197***	0.797*			
	(0.0178)	(0.414)	(0.275)	(0.433)			
lag_GDP1		0.242		0.780	1.200***		1.713***
		(0.402)		(0.654)	(0.0188)		(0.426)
lag_GDP2			0.0305	-0.351		1.155***	-0.509
			(0.259)	(0.411)		(0.0220)	(0.414)
Constant	-5.031***	-4.844***	-4.836***	-4.817***	-4.511***	-4.006***	-4.573***
	(0.175)	(0.223)	(0.262)	(0.261)	(0.184)	(0.215)	(0.231)
Observations	44	43	42	42	43	42	42
R-squared	0.992	0.991	0.990	0.991	0.990	0.986	0.990

Standard errors in brackets
*** p<0.01, ** p<0.05, * p<0.1

Explanatory variables consist of log(GDP per capita) current year, lagged 1 year, and lagged 2 years.

Table 4 shows the linear regression output from the model including control variables. Model 1 includes *Sickness Benefits*, while the variable is excluded in model 2. Tests for heteroscedasticity and multicollinearity were performed and can be found in the Tables A7-A9 and A11 in the Appendices. As heteroscedasticity was indicated, robust standard errors are presented in Table 4.

Table 4: OLS regression with control variables

VARIABLES	(1) LogHCE	(2) LogHCE
LogGDP	0.804**	0.993***
	(0.257)	(0.296)
LogGDPlag1	0.465	0.676*
	(0.382)	(0.343)
LogGDPlag2	-0.151	-0.524*
	(0.333)	(0.309)
Pop Growth (%)	-0.0458	0.0188
	(0.173)	(0.0461)
Elderly Pop	0.354***	0.0510**
	(0.0788)	(0.0201)
Sickness Benefits	0.0236***	
	(0.00585)	
Constant	-10.79***	-4.927**
	(2.596)	(0.300)
Observations	41	14
R-squared	0.987	0.993

Robust standard errors in brackets
*** p<0.01, ** p<0.05, * p<0.1

Discussion

Findings

Our results indicate a unidirectional Granger-causality from GDP to HCE, providing support for the Income View in the case of Sweden. In other words, our results suggest that GDP growth serves as an enabler of increased health spending. Moreover, we find that both *LogHCE* and *LogGDP* are stationary for the time period studied. This combination of results allowed us to perform an OLS regression, revealing that the coefficient estimate of *LogGDP* was positive and differed significantly from zero. We note that *LogGDPlag1* and *LogGDPlag2* are only significant in the absence of *LogGDP* (Table 3). In the augmented model (Table 4), demographic structure (measured as elderly population, *ElderlyPop*), as well as general health in the working-age population (measured through *Sickness Benefits*) were significant. Concerning demographic structure, we interpret this to be due to increased healthcare consumption in older age groups as found by Gerdtham (1993). Regarding the effect of *Sickness Benefits,* we are careful to draw definite conclusions as the augmented model is based on fewer observations (14 as opposed to 41), although our results indicate that increased *Sickness Benefits*, reflecting a decrease in health of the working-age population, correlate with increased healthcare expenditure. According to our model, an increase of one percentage point in GDP per capita leads to a 0.993% increase in healthcare expenditure, *ceteris paribus*. Inclusion of the variable *Sickness Benefits* reduces this effect to 0.804%.

Granger-causality analysis as a method brings with it a number of caveats. Granger (1969) states that perhaps a more appropriate term than causality would be "temporally related". Zellner (1994) criticizes Granger-causality tests that are based on two variables for being derived in a "theoretical vacuum", as relevant variables may have been omitted. It is important to keep in mind that while Granger-causality indicates that causality between the variables may exist, is *not equivalent to causality in the strict sense*. Our results merely indicate that values of *LogGDP c*an help predict future values of *LogHCE*. This is by no means sufficient to conclude that this represents "real" causality. Moreover, the absence of Granger-causality from healthcare expenditure to GDP is not a convincing argument against health investment altogether, as our model is not sufficient to fully characterize the relationship. There may still exist a feedback relationship from healthcare expenditure to GDP.

Even if this is not the case, the issue of omitted variable bias remains. As the possible determinants of health expenditure as well as the factors influencing GDP growth are numerous, it is highly likely that we have failed to control for all relevant variables in the OLS regression. Therefore, we are disinclined to draw definite conclusions from our model.

Comparison with previous studies

Our results concerning the direction of causality are consistent with the findings of Sghari and Hammami (2013). However, they found that the variables were non-stationary, which is not in agreement with our results. Another difference is the use of of only 1 lag, whereas in our study only the lag order of 2 generated significant Granger-causality. Reasons for differences in findings may relate to the use of a different dataset or the fact that Sghari and Hammami used a modified version of the Granger-causality, namely the Toda and Yamanaka test.

The direction of causality indicated in our study contradicts the findings of Erdil and Yetkiner and Devlin and Hansen. These papers both concluded that the direction of causality runs from HCE to GDP. Erdil and Yetkiner studied a total of 75 countries and found that bidirectional Granger-causality was most prevalent. In the cases of unidirectional causality, the direction of causality often differed between the income groups of the countries. Sweden, being a high-income country, showed evidence of causality from per capita health expenditure to per capita GDP. Erdil and Yetkiner report unit roots for most original series of GDP and HCE, however, they do not report country-specific unit roots tests, making it difficult to compare our results. To account for unit roots, Erdil and Yetkiner mad use of the first difference of their time series. Furthermore, Erdil and Yetkiner were limited to the years between 1990 and 2000, as data was lacking for many countries. The lag length was selected at an aggregate level. All Granger-causality analysis for high-income countries applied a lag length of 1 year for GDP and 3 years for HCE. This was based on the Akaike Information Criteria (AIC), also used in this study. The 3 year lag and and 10-year time frame means that this study could effectively only examine the relationship between GDP and HCE during a seven-year period, which we believe accounts for the differences in our results.

Devlin and Hansen found bidirectional causality in 2 out of the 20 OECD countries studied, whereas Erdil and Yetkiner found a bidirectional relationship in 12 out of the 24 high-income

countries, despite significant overlap in countries included. However, Devlin and Hansen studied an earlier but longer period between 1960 and 1987. They also studied first differences of the natural logarithm of the variables of interest, motivated by the assumption of non-stationary series. However, they do not test for unit roots or stationarity. Lastly, Devlin and Hansen used a lag length of 4, a relatively long lag length which may have compromised the power of the statistical analysis.

Strengths and Limitations

Strengths of this study include the time period studied which is considerably longer than many previous studies. This is attributed to the choice of a country-specific approach. Furthermore, our primary data source, OECD database, is a reliable source for data. However, the focus on Sweden naturally meant that fewer observations were included compared to panel data analyses, making it more difficult to generalize the findings. As data was not available as far back as the 1970s for a number of variables, there was a trade-off between inclusion of relevant variables in the OLS model and number of observations included.

Not controlling for changes in disease patterns throughout this period may have resulted in omitted variable bias. The prevalence of diseases associated with affluence should increase as GDP increases, but are also particularly costly to treat as they are chronic in nature – thereby promoting growth of healthcare expenditure. The absence of a variable capturing technological change within healthcare as a driver of costs may similarly be problematic, as evidence from a study on HCE in the United States suggested that technological advances partly explained increases in healthcare expenditure (Okunabe, 2002). Other human capital inputs, specifically education, should indirectly affect health and economic growth as indicated by economic theory (Alexandru & Maria, 2012). Gerdtham and Jonsson (2000) argue that more and "new" regressors should be tested in models attempting to explain rises in healthcare expenditure. Suggested variables include government budget deficits or tax subsidy levels of private health insurance. Another attempt to include a "new" explanatory variable was based on Baumol's model of 'unbalanced growth' (Baumol, 1967) and tested by Hartwig (Hartwig, 2008). This study indicated that the cost rises in HCE are partly due to wage increases in excess of productivity growth (Hartwig, 2008).

Test of multicollinearity (see Appendices Tables 14 & 15) indicated the presence of multicollinearity in certain models. High multicollinearity was expected between the variables of *LogGDP*, *LogGDPlag1*, and *LogGDPlag2* in model 1. However, multicollinearity between variables in model 2 (Table 4) is worth noting. The presence of multicollinearity may, of course, render the model less effective as additional variables have less added explanatory value. In addition to this, it is worth noting that the functional form of the model has not been tested. Although a linear relationship between GDP and HCE was hypothesized, as it is the most common relationship explored in the literature, there may of course exist a different relationship between these variables.

Finally, since the coefficient estimate of the impact of *LogGDP* on *LogHCE* varies depending on which control variables are included, and since the 95 % confidence interval indicated we could not establish that coefficient estimate differed from 1, we refrain from drawing any conclusions on the income-elasticity of health on the national level for Sweden. We can thus not establish whether health is a luxury good in the present study.

Conclusion

We set out to determine the direction and magnitude of causality between GDP and healthcare expenditure in Sweden between the years of 1970 and 2013. The study was largely based on the human capital theory (Health View) and the notion that healthcare expenditure is determined by income (Income View), which suggest opposing directions of causality. Naturally, one can imagine a third scenario – that is, that the two variables are interdependent. To test direction of causality, a Granger-causality analysis was applied, followed by OLS regressions with and without control variables. Our findings indicate the presence of unidirectional causality from per capita GDP to per capita HCE, supporting the Income View in the case of Sweden. However, this is not consistent with two previous studies (Erdil and Yetkiner, 1999; Devlin & Hansen, 2001). An OLS regression of HCE on GDP revealed that the coefficient estimate was positive and differed significantly from zero. This was true even when control variables were introduced into the model. The absence of a Granger-causality from healthcare expenditure to GDP is not necessarily an argument against the health-led growth hypothesis (Mushkin, 1962), as there may be more complicated unobservable causality at play. In summary, we concur with Gerdtham and Jonsson in that more and "new" regressors should be tested in models attempting to explain rises in healthcare expenditure.

Despite our findings being in favour of the Income View, other studies have found evidence supporting the Health View (Knowles & Owen, 1995; Swift, 2011). Considering this, we suggest a more specific model to test for direction of causation, perhaps by identifying intermediate variables in this causation and testing these. We also believe that the issue of varying results with respect to stationarity of time series warrants further research and insight into the implications of unit roots for analysis. Jewell et al. (2003) has pioneered this field suggesting that GDP and HCE are stationary around structural breaks. They did, however, not find a structural break in the case of Sweden, supporting our findings of stationary series.

Summary

Using Swedish data on the country-level between 1970 and 2013, this study investigates the relationship between healthcare expenditure and GDP. We summarize our methods and findings as follows:

1) By considering human capital theory and developmental economics, we found theoretical suggestions of both directions of causation between healthcare expenditure and GDP.

2) We review the literature available with regard to direction of causality in Sweden and find that conflicting findings are reported.

3) We perform Granger-causality analysis which indicates that *LogGDP* Granger-causes *LogHCE* with a lag order of 2.

4) Further, we perform OLS regressions with the dependent variable *LogHCE*. We include different combinations of *LogGDP* and lagged variables of *LogGDP* (1 year and 2 years). We find that *LogGDP* significantly affects *LogHCE* in Sweden during the time period studied. The coefficients are positive, suggesting that GDP growth predicts a rise in HCE.

5) To test the strength of the regression model mentioned above, we include control variables that could independently, or in combination, affect HCE. Out of these, elderly population as % of total population and sickness benefits among the working-age population are statistically significant. We note that *LogGDP* remains statistically significant upon inclusion of these variables.

In conclusion, we find that *LogGDP* Granger-causes *LogHCE* in Sweden between 1970 and 2013. However, we identify the need to develop more sophisticated methods to better identify the intermediates through which GDP and HCE may affect each other.

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Appendices

Descriptive Statistics

Table A1: Descriptive Statistics of Variables

Variable	Obs	Mean	Std. Dev.	Min	Max
LogGDP	44	9.817554	.6609816	8.483139	10.71591
LogHCE	44	7.203471	.8272173	5.407789	8.497818
Elderly Pop	44	16.93784	1.3129	13.66947	19.90842
Pop growth (%)	43	.4148872	.2646542	.0452718	.9378556
Sickness Benefits	15	36.26769	6.068011	26.8285	43.2

Unit-root analysis

Table A2: Augmented Dickey-Fuller tests for unit roots.

Dickey-Fuller test for unit root — Number of obs = 43

---------- Interpolated Dickey-Fuller ---------

	Test Statistic	1% Critical Value	5% Critical Value	10% Critical Value
Z(t)	-4.330	-3.628	-2.950	-2.608

MacKinnon approximate p-value for Z(t) = 0.0004

Log(GDP)

Dickey-Fuller test for unit root — Number of obs = 43

---------- Interpolated Dickey-Fuller ---------

	Test Statistic	1% Critical Value	5% Critical Value	10% Critical Value
Z(t)	-3.312	-3.628	-2.950	-2.608

MacKinnon approximate p-value for Z(t) = 0.0144

Log(Healthcare Expenditure)

Lag-order Selection Statistics

Table A3: Lag-order Selection Statistics

Selection-order criteria
Sample: 1975 - 2013 Number of obs = 39

lag	LL	LR	df	p	FPE	AIC	HQIC	SBIC
0	-30.0202				.287341	1.59078	1.60609	1.63344
1	84.5473	229.14*	1	0.000	.000849*	-4.2332*	-4.20259*	-4.14789*
2	85.1641	1.2335	1	0.267	.000866	-4.21354	-4.16763	-4.08558
3	86.5344	2.7407	1	0.098	.00085	-4.23253	-4.17132	-4.06191
4	86.64	.21116	1	0.646	.000891	-4.18667	-4.11014	-3.97339
5	87.9112	2.5425	1	0.111	.00088	-4.20058	-4.10875	-3.94464

Endogenous: LogGDP
Exogenous: _cons

Selection-order criteria
Sample: 1975 - 2013 Number of obs = 39

lag	LL	LR	df	p	FPE	AIC	HQIC	SBIC
0	-37.4143				.419832	1.96996	1.98527	2.01262
1	63.515	201.86*	1	0.000	.002498*	-3.15461*	-3.124*	-3.0693*
2	63.5632	.09645	1	0.756	.002623	-3.1058	-3.05989	-2.97784
3	63.6011	.07578	1	0.783	.002757	-3.05647	-2.99525	-2.88584
4	64.8479	2.4936	1	0.114	.002724	-3.06912	-2.9926	-2.85585
5	64.8857	.07561	1	0.783	.002865	-3.01978	-2.92795	-2.76385

Endogenous: LogHCE
Exogenous: _cons

a * indicates opitmal lag-order for that specific selection statistics.

Vector Autoregression (VAR)

Table A4: Vector Autoregression (VAR)

Sample: 1972 - 2013 Number of obs = 42
Log likelihood = 167.1065 AIC = -7.481264
FPE = 1.94e-06 HQIC = -7.329615
Det(Sigma_ml) = 1.20e-06 SBIC = -7.067533

Equation	Parms	RMSE	R-sq	chi2	P>chi2
LogGDP	5	.027966	0.9981	22017.69	0.0000
LogHCE	5	.046718	0.9965	12013.25	0.0000

	Coef.	Std. Err.	z	P>|z|	[95% Conf.	Interval]
LogGDP						
LogGDP						
L1.	1.145148	.1593534	7.19	0.000	.8328206	1.457475
L2.	-.2695767	.1525759	-1.77	0.077	-.56862	.0294666
LogHCE						
L1.	-.0240222	.0913908	-0.26	0.793	-.2031448	.1551004
L2.	.1021579	.0955644	1.07	0.285	-.0851449	.2894607
_cons	.7042155	.3316039	2.12	0.034	.0542838	1.354147
LogHCE						
LogGDP						
L1.	.7986342	.2662057	3.00	0.003	.2768806	1.320388
L2.	-.7114261	.2548836	-2.79	0.005	-1.210989	-.2118634
LogHCE						
L1.	.8279548	.1526716	5.42	0.000	.528724	1.127186
L2.	.0851486	.1596438	0.53	0.594	-.2277476	.3980448
_cons	-.1912518	.5539565	-0.35	0.730	-1.276987	.8944829

Vector Autoregression is the output upon which the Granger-causality analysis is performed.

Test for Auto-Correlation

Table A5: Lagrange-multiplier test of Autocorrelation

Lagrange-multiplier test

lag	chi2	df	Prob > chi2
1	**6.2359**	**4**	**0.18221**
2	**5.6797**	**4**	**0.22438**

HO: no autocorrelation at lag order

Table 9 shows test for autocorrelation of the Vector Autoregression. The presence of autocorrelation may indicate that a greater number of lags is needed . Since the P-values exceed .05 at 5% level the null hypothesis of no autocorrelation cannot be rejected, suggesting an adequately selected lag order. The LM-test is chosen over a DW-test since the VAR includes lagged dependent variables.

Tests for Heteroskedasticity

Table A6: Breusch-Pagan test for heteroscedasticity.

```
Breusch-Pagan / Cook-Weisberg test for heteroskedasticity
         Ho: Constant variance
         Variables: fitted values of LogHCE

         chi2(1)      =     1.65
         Prob > chi2  =   0.1995
```

Prob > chi2 = 0.1995 signifying that the null-hypothesis that the variance is homogenous cannot be rejected.

Table A7: Breusch-Pagan test for heteroscedasticity excluding the Sickness Benefits model.

```
Breusch-Pagan / Cook-Weisberg test for heteroskedasticity
         Ho: Constant variance
         Variables: fitted values of LogHCE

         chi2(1)      =     0.32
         Prob > chi2  =   0.5715
```

Table A8: Breusch-Pagan test for heteroscedasticity including the Sickness Benefits model

```
Breusch-Pagan / Cook-Weisberg test for heteroskedasticity
         Ho: Constant variance
         Variables: fitted values of LogHCE

         chi2(1)      =     4.12
         Prob > chi2  =   0.0425
```

.

When including the Sickness Benefit variable the Breusch-Pagan test seems to indicate heteroscedasticity. However this test is very sensitive to model assumptions (Stata, UCLA) which is why we choose to complement with the Cameron & Trivedi test below.

Table A9: White's test for heteroscedasticity with null-hypothesis that variance is homogenous.

```
Cameron & Trivedi's decomposition of IM-test
```

Source	chi2	df	p
Heteroskedasticity	14.00	13	0.3738
Skewness	8.20	6	0.2239
Kurtosis	0.17	1	0.6815
Total	22.37	20	0.3209

As evident from Table 13. The null hypothesis of homoscedasticity cannot be rejected as p-values exced critical value of 0.05.

Test for Multicollinearity

Table A10: Variance Inflation Factors

Variable	VIF	1/VIF
LogGDPlag1	1225.08	0.000816
LogGDPlag2	511.82	0.001954
LogGDP	503.16	0.001987
Mean VIF	746.68	

High VIF values indicates collinearity (Stata, UCLA). Unsurprisingly, values of GDP are co-linear with lagged values of itself. The primary concern of multicollinearity is that standard errors in the regression model become inflated (Stata, UCLA).

Table A11: Variance Inflation Factors

Variable	VIF	1/VIF
POPTOTAGRWTH	45.59	0.021935
LogGDPlag1	45.01	0.022218
LogGDPlag2	43.82	0.022821
ELDLYPOP	31.62	0.031629
LogGDP	26.09	0.038330
OHALSOTAL	18.62	0.053697
Mean VIF	35.12	

OHALSOTAL refers to the Sickness Benefit variable

Stata Commands

summarize LogGDP LogHCE

dfuller LogGDP
dfuller LogHCE

vecrank LogGDP LogHCE, trend(constant) max

varsoc LogGDP, maxlag (5)
varsoc LogHCE, maxlag (5)

varlmar

var LogGDP, LogHCE, lags (1/2)

vargranger

regress LogHCE LogGDP

regress LogHCE LogGDP LogGDPlag1 LogGDPlag2 OHALSOTAL ELDLYPOP POPTOTAGRWTH

estat hettest

estat imtest

vif