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Testing the growth effects of structural change

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Testing the growth effects of structural change

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Abstract

Against the backdrop of Baumol's model of 'unbalanced growth', a recent strand of literature has presented models that manage to reconcile structural change with Kaldor's 'stylized fact' of the relative constancy of per-capita GDP growth. Another strand of literature goes beyond this, arguing that the expenditure shifts toward Baumol's 'stagnant' sector stimulate rather than dampen long-term economic growth because of the human capital-accumulating nature of major 'stagnant' services (like health care and education). This paper tests the relationship between structural change and economic growth empirically by means of a Granger-causality analysis of a panel of 18 OECD countries.

1. Introduction

William J. Baumol's (1967) article 'Macroeconomics of unbalanced growth' is widely regarded as a major contribution to the literature on structural change. Whereas the first champions of the 'tertiarization hypothesis', Allan Fisher (1935) and Colin Clark (1940), traced back the gradual shift in production from the agricultural to the manufacturing sector and onwards to the services sector mainly to changes in demand, Baumol offers a supply-side explanation.¹ In a nutshell -a more thorough discussion will follow in the next section - the model's story goes like this. Productivity growth is higher in the 'progressive' (secondary) than in the 'nonprogressive' - or 'stagnant' - (tertiary) sector, but wages grow more or less the same in both sectors. Therefore, unit costs and also prices rise much faster in the tertiary sector than in the secondary. Demand for certain services, like health care and education for instance, is hardly price-elastic, hence consumers are willing to pay the higher prices. Therefore, even if the two sectors keep their proportion in terms of 'real' production, an ever higher share of total expenditures will be channeled into the services sector. This is called the 'cost disease' (or sometimes also 'Baumol's disease'). Also, since aggregate productivity growth is a weighted average of the sectoral productivity growth rates with the weights provided by the nominal value added shares, the aggregate productivity growth rate will decline over time as the sector with the low productivity growth receives an ever-increasing weight. Thus, Baumol's model of unbalanced growth predicts a tendency for per-capita output in mature economies to stagnate, even though technical progress (or exogenous productivity growth, respectively) is admitted for the 'progressive' sector.

The model's prediction of a decline in per-capita growth contradicts one of Kaldor's 'stylized facts', however. As is well known, Kaldor (1957) enumerates a number of "remarkable historical constancies revealed by recent empirical investigations" (Kaldor, 1957, p. 591), one of which is the rough constancy of per-capita output growth.² A recent literature aims at reconciling structural change – defined, for instance, in terms of employment shifts toward the 'stagnant sector' – with balanced (or constant) aggregate growth. The bulk of this literature, which includes Echevarria (1997), Laitner (2000), Caselli and Coleman (2001), Kongsamut et al. (2001), Meckl (2002), Foellmi and Zweimüller (2008), and Bonatti and Felice (2008) has relied on a demand-side explanation for structural change – namely on non-

¹ For a survey of the different 'tertiarization' theories, cf. Schettkat and Yocarini (2006).

 $^{^{2}}$ The other 'stylized facts' are that the capital stock per-capita grows at roughly the same rate as per-capita output, that the wage and profit shares in national income are nearly constant, and that the rate of return to capital is constant. These 'facts' imply that the capital/output ratio remains constant over time, whereas real wages will grow.

homothetic (hierarchical) utility functions consistent with Engel's law – to derive this result. Ngai and Pissarides (2007), and Acemoglu and Guerrieri (2008), however, follow Baumol in tracing back structural change to the supply side. Ngai and Pissarides (2007) show that when capital is added to Baumol's model as an additional factor of production, the economy can reach a balanced growth path in the aggregate under certain circumstances while still exhibiting supply-side driven structural change due to differences in (exogenous) total factor productivity growth across sectors and a low (below one) elasticity of substitution across final goods. In this case, a necessary and sufficient condition for balanced growth in the aggregate is a logarithmic intertemporal utility function, in other words, a unit intertemporal elasticity of substitution. Acemoglu and Guerrieri (2008) introduce differences in factor proportions and capital deepening in a Baumol-style two-sector model and show that capital deepening can cause structural change and unbalanced growth between the two sectors and at the same time constant consumption growth in the aggregate.

While these papers aim at reconciling Baumol's model – or at least structural change – with Kaldor's 'fact' of balanced aggregate GDP growth, a second strand of the literature goes a step further, arguing that the shift of expenditures and employment to the 'stagnant' services in fact *stimulates* long-term economic growth instead of dampening it. This argument builds on the Uzawa-Lucas model of endogenous economic growth. Uzawa (1965) and Lucas (1988) present models in which both physical capital and human capital enter the production function. Workers use only a fraction of their human capital in the production process and allocate the rest to an increase in human capital through education. The growth of human capital in the economy depends on the share of total time spent on education and a productivity parameter which measures the efficiency of education in increasing human capital. Long-term per-capita GDP growth does not depend on exogenous technological progress as in the Solow (1956, 1957) model of economic growth. Rather, growth is endogenous and depends on the fraction of human capital devoted to education.

Against the backdrop of Baumol's model, Pugno (2006) argues that the sectors that contribute most to human capital formation are exactly Baumol's 'stagnant services', namely education and health care.³ Pugno develops a 'health augmented' Uzawa-Lucas model and

³ Pioneering studies focused on the educational dimension of human capital, yet it has been known for long that human capital can also be accumulated by improving the health status of the population (cf. Schultz, 1961, Mushkin, 1962).

combines it with Baumol's model of unbalanced productivity growth.⁴ In the resulting synthetic model, the supply-side shift to the 'stagnant' – yet human capital accumulating – services endogenizes productivity growth. Although his model is able to perform different dynamics (depending on parameter constellations), Pugno (2006, p. 112) conjectures that "despite the possible adverse composition effect", the overall effect of structural change on aggregate productivity growth is likely to be positive. van Zon and Muysken (2005) follow a similar approach. They also augment the Uzawa-Lucas model with health and combine the resulting endogenous growth model with what they call 'Baumol's law', according to which employment shifts to the 'stagnant sectors'. In their model also, steady-state GDP growth may benefit from an increased employment in the education and health care sectors – i.e. from structural change – depending on the combination of the model's structural parameters. A noteworthy difference between the two models is that while Pugno remains true to Baumol's production function with factor labor only, van Zon and Muysken – more in line with the Uzawa-Lucas model – include physical capital also.

The articles discussed so far are theoretical in nature. Peneder (2003), Maudos et al. (2008), and Maroto-Sánchez and Cuadrado-Roura (2009) have attempted to determine the impact of tertiarization on economic growth empirically using 'shift-share analysis' as main tool. Shift-share analysis is an accounting technique which disaggregates productivity growth over a period of time into a 'within effect' (productivity growth within each industry), a 'static shift effect' (productivity growth due to employment shifts toward more productive industries), and a 'dynamic shift effect' (productivity growth due to employment shifts toward industries with higher productivity growth rates). Although not totally unambiguous, the results from this line of research tend to support Baumol's prediction that employment is shifting toward the stagnant sectors, thereby dampening overall productivity growth.

My aim in this paper it is to introduce an alternative test for the relationship between structural change and economic growth: the panel Granger-causality framework. As the articles discussed so far as well as international data compilations like the OECD's Health database amply document the perpetual shift of employment and expenditures into 'stagnant' sectors like health care and education, structural change as such will be taken for granted here. The question this paper will explore is whether past structural change had a negative effect on overall economic growth (as Baumol's model of unbalanced growth predicts), a positive

⁴ Pugno also assumes a hierarchical utility function – in other words a shift of demand to services as income increases –, but his results do not hinge on this assumption.

effect – as Pugno (2006) and van Zon and Muysken (2005) conjecture – or no effect at all as the aforementioned balanced growth models of structural change would imply.

The paper is organized as follows. The next section formally introduces Baumol's model of unbalanced growth which is the starting point of the debate over the growth effects of structural change. As Baumol's model predicts long-term stagnation, and human capital formation through education and health care has been proposed as an antidote for stagnation, section 3 briefly discusses the state of the empirical literature on the impact of human capital n formation on economic growth. Section 4 opens up the empirical part of the paper, introducing the data and methods to be used in the estimations, especially the methodology of (dynamic) panel Granger-causality tests. Section 5 presents the results of the causality analysis of a panel of 18 OECD countries, including results of robustness checks, and section 6 concludes.

2. Structural change as a cause of stagnation: Baumol's model of unbalanced growth revisited

Baumol presents a model in which the economy is divided into a 'progressive' and a 'nonprogressive' – or 'stagnant' – sector. His fundamental assumption then is that 'regular' growth in labor productivity can occur only in the 'progressive' sector.

It is important to understand what the term 'regular' here means. For Baumol, *regular* productivity growth is the result of technological innovation which manifests itself in new capital goods. Capital goods are also responsible for economies of scale, being another source of productivity growth. *Regular* productivity growth is thus defined to depend on certain physico-technological requirements. In the service industries, Baumol argues, and also in agriculture and construction, physical capital cannot be used as a substitute for labor on the same scale as in manufacturing. These industries he therefore relegates to the 'stagnant' sector. Baumol does not claim that increases in labor productivity are impossible in the 'stagnant' sector, only that this sector comprises "activities which, by their very nature, permit only sporadic increases in productivity" (Baumol, 1967, p. 416). In his model, Baumol abstracts from productivity increases in the 'stagnant' sector for simplicity.

Next, Baumol assumes that nominal wages in both sectors are related in the long run. He simplifies further and assumes that they are equal. His final assumption is that nominal wages (in both sectors) rise to the same extent as labor productivity in the 'progressive' sector. This implies that the price level in the 'progressive' sector stays constant, whereas it rises in the 'stagnant' sector in order to keep the level of real wages in line with the productivity level.

The workers, regardless in which sector they work, buy goods and services from both sectors so that their respective real wages converge.

Equations (1) and (2) describe the production functions of the two sectors. Labor is the only factor of production. Labor productivity in the 'stagnant' sector (1) stays constant, whereas it grows in the 'progressive' sector (2) at the constant rate r. Thus, output in the two sectors (Y_1 and Y_2) at time t is given by:

$$Y_{1t} = aL_{1t} \tag{1}$$

$$Y_{2t} = bL_{2t}e^{rt} \tag{2}$$

with L_1 and L_2 as quantities of labor employed in the two sectors and a and b as constants.

According to the aforementioned assumption, the nominal wage (in both sectors) is given by:

$$W_t = W e^{rt} \tag{3}$$

with *W* as an arbitrary starting value.

Equation (3) completes the model of unbalanced growth already. This simple model has a couple of interesting implications which Baumol draws out. First, the 'Cost Disease': equations (4) and (5) show that costs per unit of output in the 'stagnant' sector tend toward infinity while they stay constant in the 'progressive' sector.

$$C_{1} = W_{t}L_{1t} / Y_{1t} = We^{rt}L_{1t} / aL_{1t} = We^{rt} / a$$
(4)

$$C_2 = W_t L_{2t} / Y_{2t} = W e^{rt} L_{2t} / b L_{2t} e^{rt} = W / b$$
(5)

Relative costs also tend toward infinity ($C_1/C_2 = be^{rt}/a$). Under 'normal' circumstances – that is when prices rise in proportion to costs and when demand is price-elastic – the 'stagnant' sector will vanish. Baumol (1967, p. 421) mentions craftsmanship, fine restaurants, and theatres as examples of establishments that have either disappeared or retreated to luxury niches as a consequence of customers' unwillingness to tolerate the price increases that would have been necessary to cover rising costs.

Yet, parts of the 'stagnant' sector produce necessities for which the price elasticity is very low. Baumol calls attention to education and health care as prime examples. To show what happens in these industries as a consequence of unbalanced growth, Baumol assumes that the relation of real output of the two sectors remains unchanged as in (6):

$$(b/a) Y_1/Y_2 = L_1/L_2 e^{rt} = K$$
(6)

with K = const. If L (= L₁ + L₂) is the labor force, it follows:

$$L_1 = (L - L_1)Ke^{rt} \qquad \Longleftrightarrow \qquad L_1 = LKe^{rt} / (1 + Ke^{rt}) \tag{7}$$

and
$$L_2 = L - L_1 = L/(1 + Ke^{rt})$$
 (8).

From (7) and (8) we learn that, over the years $(t \to \infty)$, L_1 tends toward L, and L_2 tends toward zero. The model thus predicts structural change in terms of a perpetual shift of both expenditures and employment toward the 'stagnant services' sector.

Finally, it can be shown what happens to the GDP growth rate under these conditions. Let *I* be an index for real GDP which is calculated as a weighted average of the value added of the two sectors:

$$I = B_1 Y_1 + B_2 Y_2 = B_1 a L_1 + B_2 b L_2 e^{rt}$$
(9).

Then, if we insert (7) and (8) into (9) we get:

 $I = L(KB_1a + B_2b)e^{rt} / (1 + Ke^{rt}) = Re^{rt} / (1 + Ke^{rt})$ (10),

with
$$R = L(KB_1a + B_2b)$$
(11)

Applying the quotient rule leads to:

$$dI / dt = R[re^{rt}(1 + Ke^{rt}) - Kre^{2rt}] / (1 + Ke^{rt})^2$$

= $rRe^{rt} / (1 + Ke^{rt})^2$ (12).

We can calculate the growth rate of real GDP as:

$$(dI/dt)/I = r/(1 + Ke^{rt})$$
(13).

It follows that over time $(t \to \infty)$, the GDP growth rate drops asymptotically to zero *ceteris* paribus.⁵

3. Human capital formation as an antidote? A brief review of the literature

Authors such as van Zon and Muysken (2005) and Pugno (2006), who argue that human capital formation could act as an antidote against the stagnationist consequences of structural change identified by Baumol's model, draw on Uzawa (1965) and Lucas (1988). In the Uzawa-Lucas model, endogenous human capital formation in the form of education replaces exogenous technological progress as driver of long-term per-capita growth. Against the backdrop of this model, however, Mankiw et al. (1992) vindicated exogenous growth, arguing that education is similar to investment in physical capital in that it can increase growth temporarily during the convergence phase toward steady state, but not thereafter.⁶ Subsequent empirical studies by Benhabib and Spiegel (1994), Bils and Klenow (2000), and Pritchett (2001) further put into perspective the importance of education for economic growth.

⁵ *Ceteris paribus* here especially means that *L* remains constant. If *L* grows at the rate *n*, then *n* must be added at the right hand side of (13). Long-run stagnation then occurs for per-capita GDP.

⁶ Bernanke and Gürkaynak (2001, p. 12) point out, however, that Mankiw et al.'s (1992) "basic estimation framework is broadly consistent with *any* growth model that admits a balanced growth path" and that it therefore cannot discriminate between theories of endogenous and exogenous growth.

However, the reason why these studies find insignificant or even negative coefficients for the education variable in growth regressions may be that the Barro and Lee (1993, 2001) dataset they use for educational attainment is on the one hand not completely satisfactory from a theoretical perspective (as it does not take account of differences in schooling quality, for instance) and is on the other hand vitiated by data anomalies (cf. Krueger and Lindahl, 2001, De la Fuente and Domenech, 2006). Cohen and Soto (2007) construct a new dataset for years of schooling which performs somewhat 'better' in terms of producing significantly positive coefficients. Their effort reduces rather than eliminates measurement errors, however, as Cohen and Soto admit.

More recently, growth models have been 'augmented' by the health dimension of human capital. Whereas doubts have emerged concerning the growth-enhancing role of education, the existing empirical evidence is quite favorable toward the idea of a growth-stimulating role of health capital formation. Macroeconomic growth studies with a focus on health such as Knowles and Owen (1995, 1997), Rivera and Currais (1999a, 1999b, 2003, 2004), Bhargava et al. (2001), Heshmati (2001), McDonald and Roberts (2002), Bloom et al. (2004), Jamison et al. (2005), and Weil (2007) have found a significantly positive influence of health capital formation on economic growth.⁷ However, health's positive effect on GDP seems to be strongest among poor countries. For rich countries, the existing empirical evidence on whether health capital formation stimulates GDP growth is mixed. While Heshmati (2001) and Rivera and Currais (1999a, 1999b, 2003, 2004) find a positive effect of health expenditure growth on productivity growth for OECD countries (or Spanish regions, respectively), Knowles and Owen (1995, 1997) as well as McDonald and Roberts (2002) reject the hypothesis that life expectancy is a statistically significant explanatory variable for productivity growth in high-income countries. Bhargava et al. (2001) even estimate a negative effect of the adult survival rate on per-capita GDP growth for several rich countries. Acemoglu and Johnson (2007) reach a similar conclusion.

The empirical evidence on the growth effects of human capital formation is mixed; and even if it wasn't, the studies discussed in this section would probably not be helpful in evaluating the growth effects of *structural change*. The reason is that all these studies model and estimate human capital-augmented aggregate production functions. In the estimation framework of an aggregate production function, the effects highlighted by Baumol's model of

 $^{^{7}}$ In these studies, health capital formation is proxied either by health care expenditure (growth) or by the increases in life expectancy and the adult survival rate (which measures the probability that a 15-year-old will reach the age of 60).

unbalanced growth – namely that the process of raising certain input levels (such as health and education) can imply structural change that is unfavorable for overall economic growth – might vanish in the estimates of total factor productivity growth. For example, if Baumol is right, then even if the health variable (e.g. life expectancy) has a positive sign in an estimated production function, a positive shock to health expenditure, even if it improves life expectancy, could lower total factor productivity growth to an extent that overcompensates the effect of the rise in health status on growth. The positive coefficient of the health variable would then be misleading. For such reasons, Temple (1999, p. 150) notes that "(t)he mention of structural change leads naturally to questioning the relevance of aggregate production functions". Another modeling framework is therefore required. The next section will apply the concept of Granger-causality to test the relationship between structural change and economic growth in a panel of OECD countries.

4. Methodology and data

Baumol's model of unbalanced growth predicts that structural change – defined as the shift of employment and expenditures toward 'stagnant' sectors like health care and education – will cause per-capita GDP growth to decline.⁸ Periods in which employment in and/or expenditures on health care and education increase strongly should therefore precede episodes with lower GDP growth. Otherwise, if van Zon and Muysken (2005) and Pugno (2006) were right, periods in which activity in health care and education rises are periods in which human capital is accumulated at a strong pace. Later periods should then show higher per-capita GDP growth. Finally, in models like those by Ngai and Pissarides (2007) and Acemoglu and Guerrieri (2008), structural change is compatible with balanced economic growth in the aggregate. Therefore, there should be no significant impact of structural change on per-capita GDP growth in later periods.

In order to test which of these hypotheses has an empirical grounding, we need a methodology that keeps track of the time-lags involved and that ascertains that causes, if we can identify them, precede effects. Both these aspects suggest choosing the methodology of Granger-causality testing for the empirical part of this paper. Granger's (1969) testable definition of causality has in the meantime become a standard analytical tool in applied

⁸ Baumol models unbalanced productivity – i.e. GDP per hour or per worker – growth. I follow Krueger and Lindahl (2001, p. 1118) in concentrating on GDP per-capita instead "because it reflects labor force participation decisions and because it has been the focus of much of the previous literature". Data availability considerations also play a role (see below).

econometrics. According to this definition, a stationary time series Y_t is said to 'cause' another stationary time series X_t if – under the assumption that all other information is irrelevant – the inclusion of past values of Y_t significantly reduces the predictive error variance of X_t . In econometric practice, whether Y_t Granger-causes X_t is typically tested by regressing X_t on its own lags and on lags of Y_t . If the lags of Y_t are found to be significantly different from zero, then the hypothesis that Y_t Granger-causes X_t cannot be rejected.

More recently, the notion of Granger-causality has found its way into panel econometrics. Around a dozen macroeconomic panel Granger-causality studies – mostly inquiring the causal nexus between certain variables and economic growth – have appeared in the literature since the beginning of the new millennium. These papers do not implement the methodology of panel Granger-causality testing identically, however. For instance, some do impose the restriction that the coefficients of the lagged X_t and Y_t variables must the same for all crosssection members while others don't. Another difference is that some papers use data in (log) levels while others use growth rates. This evidences that a thoroughly accepted way of implementing panel Granger-causality analysis has not yet emerged. Therefore, I will describe precisely how the method will be implemented here. Beforehand, it is apposite to take a look at the data, though.

Since this paper aims at testing the growth effects of structural change, and tertiarization is predominantly a rich-country phenomenon, I will concentrate on OECD economies. Data on the dependent variable, which is real per-capita GDP, are readily available from the OECD's National Accounts website back to 1970.⁹ Structural change can be measured either in terms of employment shifts or in terms of expenditure shifts toward the 'stagnant' services sectors – or human-capital accumulating services sectors, depending on the perspective – education and health care.

Employment data for the two-digit level of the *International Standard Industrial Classification* (ISIC) can be retrieved from the OECD's Structural Analysis (STAN) database.¹⁰ Unfortunately, only five out of 26 OECD countries for which there are data in the 2008 edition of the STAN database report employment data for the two-digit industries 'education' (ISIC 80) and 'health and social work' (ISIC 85) that go back to 1970. For 12 countries on the other hand, the time series start later than 1990. Since it is standard in empirical growth studies to transform annual data into five-year average growth rates in order to eliminate the cyclical component, and since the application of the panel Granger-causality

⁹ Cf. http://www.oecd.org/std/national-accounts and http://stats.oecd.org/wbos/index.aspx.

¹⁰ Cf. http://www.oecd.org/sti/stan and http://stats.oecd.org/wbos/index.aspx.

method involves estimating models with lags, a loss of observations from the 1970s and 1980s cannot be accepted. Therefore, measuring structural change on the basis of employment data is not an option for the purpose of this paper. I will resort to expenditure data instead.

Most research involving health care expenditure data is based on the OECD's Health database (OECD, 2007). This source will be used for the present study also. Data on public expenditure on education are available from the World Bank's educational statistics database (EdStats).¹¹ This database contains time series for the share of public education expenditure in GDP which, for most OECD countries, cover the period 1970 to 2005. By multiplying these shares with nominal GDP, time series for nominal public education expenditures can be obtained. Unfortunately, data on private education expenditure back to 1970 (in levels or as a share of GDP) are not available from the EdStats database. This is probably not very harmful, however. The OECD (2008) has recently published the split between public and private education expenditure for its member states in 2000 and 2005. Table B3.1 of the OECD study shows that next to all continental European OECD countries had public shares in total education expenditure above 90 percent both in 2000 and in 2005. All non-European countries and the U.K., on the other hand, had much lower shares - with South Korea at the bottom end (58.9 percent in 2005). However, if the split between public and private education expenditure remains stable over time, then public expenditure growth can still serve as a proxy for total expenditure growth in dynamic analysis. Low but stable public expenditure shares can be observed in South Korea and in the U.S. (In the U.S., the share has remained at 67.3 percent between 2000 and 2005.) There are two countries, however, where a marked shift from public toward private education expenditure has occurred between 2000 and 2005 (and probably already earlier). These two countries are the U.K., where the public share fell by 5.2 percentage points (PP) to 80.0 percent in 2005, and Canada, with a 4.4 PP drop to 75.5 percent.¹² For these two countries at least, public education expenditure growth will probably understate the true human capital formation. I will control for a possible bias due to the inclusion of the U.K. and Canada in a jackknifing exercise which is part of the robustness analysis to be presented in the next section.

Data on public education expenditure with a frequency of at least five years and a starting point in 1970 are available for 20 OECD countries from the World Bank database. Health expenditure data for the same period are available for 21 countries from the OECD's Health database. The data overlap for 18 countries which will therefore be included in the empirical

¹¹ Cf. http://www.worldbank.org/education/edstats.

¹² The public education expenditure share also declined in Japan (by 2.2 PP) and Australia (by 1.9 PP).

analysis of this paper. These countries are *Australia*, *Austria*, *Canada*, *Denmark*, *Finland*, *France*, *Iceland*, *Ireland*, *Japan*, *Luxembourg*, *Netherlands*, *New Zealand*, *Norway*, *Spain*, *Sweden*, *Switzerland*, the *United Kingdom*, and the *United States*. All nominal data will be deflated by the GDP deflator in order to eliminate the upward trend in the time series that is due to purely monetary factors, or general inflation, respectively. Afterwards, real expenditures will be divided by population numbers to obtain per-capita values. Finally, all level data will be transformed into five-year average annual growth rates (geometrical means) in order to eliminate the cyclical component.¹³ From the data covering the period 1970-2005 it is possible to construct a balanced panel with 18 cross-sections and a time dimension of seven five-year-average growth rates. The panel thus consists of 126 observations for each variable¹⁴ (which is a clear improvement over the earlier empirical literature).¹⁵

As the Granger-causality tests require the data to be stationary, the resulting time series of growth rates will be tested for the presence of unit roots, applying a battery of now standard panel unit root tests. When these tests fail to detect unit roots, the panel estimation models can be set up, for which the restriction of identical coefficients of the lagged X_{it} , Y_{it} and Z_{it} variables will be imposed. Thus, I will estimate a time-stationary VAR model adapted to a panel context (as in Holtz-Eakin et al., 1988) of the form:

$$X_{it} = \alpha_0 + \sum_{l=1}^m \alpha_l X_{it-l} + \sum_{l=1}^m \beta_l Y_{it-l} + \sum_{l=1}^m \chi_l Z_{it-l} + \mu_i + u_{it}$$
(1).

 X_{it} denotes the five-year averages of the growth rates of per-capita GDP, and Y_{it} stands for the five-year averages of the growth rates of per-capita investment in human capital (education and health care expenditures taken together). Contrary to Pugno (2006), van Zon and Muysken (2005) also include investment in physical capital in their model. This variable will

¹³ An alternative to using five-year-average real growth rates would be to use the change in the share of expenditures on health and education in GDP over five years. But if we want to explain GDP growth with this variable, the fact that GDP appears as the denominator of the share will introduce a negative bias in the estimated coefficients (cf. also Cohen and Soto, 2007). Therefore, I opt for the five-year-average growth rates.

¹⁴ There are no data on public education expenditure for Canada and Luxembourg for the most recent five-year interval, so we lose two observations for this variable.

¹⁵ Most of the studies discussed in section 3 use cross-sectional data. Some, however, also use panel data. Heshmati's (2001) data, for instance, cover the period 1970-1992. He calculates four five-year averages and one three-year average, and he has 22 OECD countries in his panel. His database thus consists of 110 observations. Rivera and Currais (2004) also use a panel approach with five five-year averages over the period 1973-1993. For the 17 Spanish regions they look at, this yields 85 observations.

be tested also and included as Z_{it} .¹⁶ As a robustness test, education and health care expenditures will also be tested separately; in this case Y_{it} stands for the former and Z_{it} for the latter variable. N countries (indexed by i) are observed over T periods (indexed by t). I allow for country-specific effects μ_i . The disturbances u_{it} are assumed to be independently distributed across countries with a zero mean. They may display heteroscedasticity across time and countries, though.

Estimating equation (1) with pooled OLS presents an endogeneity problem since if the dummy variables (country-specific effects) affect GDP growth in one period they presumably affected them in the previous period also (cf. Nickell, 1981). The first step in the direction of correcting this endogeneity problem in dynamic panels is to take the first difference of all variables and to thereby eliminate the individual effects. Still, if health and education expenditure rise and decline with GDP (which is probable¹⁷), the explanatory variables are not exogenous to GDP, which will result in a correlation between the right hand side variables and the error term. Arellano and Bond (1991) have shown that the best way around this problem is to use lags of the dependent variable from at least two periods earlier (in levels) as well as lags of the right-hand side variables as instruments in a Generalized Method of Moments (GMM) estimator. Arellano and Bover (1995) and Blundell and Bond (1998) suggest to difference the instruments instead of the regressors in order to make them exogenous to the fixed effects. This leads from the 'difference' GMM to the 'system' GMM estimator, which is a joint estimation of the equation in levels and in first differences (cf. Roodman, 2009). The next section will present results using the OLS, the one-step system GMM, and the two-step system GMM estimators. Based on these results, a conclusion on causality will be reached by running Wald tests on the coefficients of the lagged Y_t's and Z_t's to check whether they are jointly statistically different from zero.¹⁸ A number of robustness tests will consolidate the empirical analysis.

5. Empirical results

A reasonable first step in empirical analysis is a visual inspection of the data. Figures 1 and 2 show the histograms of the five-year average growth rates of real per-capita GDP and real

¹⁶ The data source for real per-capita gross fixed capital formation is the same as for real per-capita GDP, namely the OECD's National Accounts website.

¹⁷ For instance, Hartwig (2008) reviews the literature on health care expenditure (HCE) determinants and concludes that GDP – or national income, respectively – is *the only* robust explanatory variable for HCE that this literature has been able to uncover.

¹⁸ Podrecca and Carmeci (2001) follow a similar approach to estimation and causality testing.

per-capita expenditure on health and education for my sample of 18 OECD countries.¹⁹ Obviously, health and education expenditure has outgrown GDP. The median growth rate of real per-capita expenditure on 'stagnant services' is 3.0 percent, whereas the median growth rate of real per-capita GDP is only 2.1 percent.

Both variables exhibit large outliers. Ireland's per-capita real GDP grew by an amazing 8¹/₂ percent per year on average over the period 1995-2000. On the other hand, Luxembourg's per-capita real expenditure on health and education shrank each year by almost 5 percent on average over the period 1980-85. Outliers like these strengthen the case for carrying out the jackknifing exercise proposed in the previous section as a robustness test.

< Insert Figures 1 and 2 around here >

As was also mentioned in previous section, Granger-causality tests require stationary time series. Unfortunately, the available panel unit root tests are mainly designed for panels where both the time dimension and the cross section dimension are relatively large.²⁰ In panels such as mine with a time dimension of only 7 observations, the analysis can proceed only under restrictive assumptions like, for instance, dynamic homogeneity. This has to be kept in mind when interpreting the results of panel unit root tests reported in Table 1.²¹ As the table shows, the tests reject the null hypothesis of non-stationarity for both variables. For what they are worth, these test results at least do not speak against proceeding to the Granger-causality tests.

< Insert Table 1 around here >

Since Granger-causality test results are sensitive to the choice of lag length m in the timestationary VAR model given by equation (1), it is important to specify the lag structure appropriately. In a first step, I follow Miyakoshi and Tsukuda (2004) and Atukeren (2007) in estimating equation (1) with OLS and basing the choice of the optimal lag length on the Schwarz Information Criterion (SIC). Table 2 shows that – based on this criterion – the optimal lag length is one.

¹⁹ Figure 2 shows growth rates for the sum of total health care expenditure (HCE) and public education expenditure (PEE) (deflated by the GDP deflator and divided by population numbers).

²⁰ For a detailed account of panel unit root tests, cf. Breitung and Pesaran (2008).

²¹ Table 1 reports results for the panel unit root tests available in EViews. The estimations for this paper were done either with EViews (v. 6) or with Stata (v. 9).

< Insert Table 2 around here >

Table 3 shows the results for estimating the VAR model (1) with OLS, with the one-step system GMM estimator and with the two-step system GMM estimator.²² The OLS specification includes country-specific fixed effects (random effects were rejected by the Hausman test for correlated random effects). The GMM specifications include period-specific effects. Lags of the dependent variable from at least two periods earlier as well as lags of the per-capita health and education expenditure growth variable serve as GMM-style instruments.²³ For the two-step estimator, the small sample correction proposed by Windmeijer (2005) is implemented.

< Insert Table 3 around here >

The bottom of the table reports specification test results for the GMM estimations. The Sargan test is a test on whether the instruments are uncorrelated with the error term u_{it} (which they must be in order to be valid instruments). Table 3 shows that the null hypothesis is accepted (models (2) and (4)). While in the standard instrument matrix each instrumenting variable generates one column for each time period and lag available to that time period, Roodman (2009) proposes to 'collapse' the instrument set into a single column to limit the instrument count. This option is available in Stata (v. 9) and has been used here as a robustness check (models (3) and (5)). Collapsing the set of instruments raises the probability level for the Sargan test. For the two-step estimation, another test – the Hansen test – is available.²⁴ This test also accepts the over-identifying restrictions.

The Arellano-Bond test, however, rejects the null hypothesis of no second-order autocorrelation in the disturbances of the first differenced equation in three out of four estimations at the 10 percent level. This is unfortunate since second-order autocorrelation in the differenced residuals is in conflict with the estimator's consistency requirements (cf. Arellano and Bond, 1991). To solve this problem – and also to scrutinize the robustness of the

²² The one-step estimator uses the identity matrix as a weighting matrix. The two-step estimator weighs the instruments asymptotically efficient using one-step estimates.

²³ Roodman's 'xtabond2' command was used in Stata (v. 9) for the GMM estimations; and Roodman's (2009) examples geared my handling of the syntax.

²⁴ The Sargan statistic, which is the minimized value of the one-step GMM criterion function, is not robust to heteroskedasticity or autocorrelation. The Hansen statistic (which is the minimized value of the two-step GMM criterion function) is robust.

results – another lag of the explanatory variables will be added. Table 4 shows that all tests now accept the specification. Therefore – and despite the test results reported in Table 2 –, I will use two lags of the right-hand side variables from now on.

< Insert Table 4 around here >

In all estimations except model (4), the coefficients of the health and public education expenditure growth variable are negative. Most of the time, however, the coefficients are not statistically different from zero both individually and jointly. Only in model (8), the Wald test rejects the null hypothesis that the coefficients of the lagged expenditure growth variables are jointly equal to zero at the 10 percent level. In this case we might conclude that real per-capita expenditure growth on health and education Granger-causes real per-capita GDP growth with a negative sign. Otherwise, the estimations yield no evidence in favor of Granger-causality.

How do these results relate to the controversy over the growth effects of structural change? According to Baumol's model, structural change leads to employment and expenditure shifts toward 'stagnant services' like health care and education, which in turn reduces per-capita GDP growth. Although the expenditure shifts predicted by Baumol's model have indeed occurred - adequate employment data is unfortunately lacking for the majority of OECD countries - the foreseen effect on economic growth is dubious. Only model (8) – the one-step system GMM estimation with a collapsed set of instruments and two lags of the explanatory variables - yields some evidence in favor of the stagnationist view. The other estimations find no Granger-causality running from real per-capita expenditure growth on health and education toward real per-capita GDP growth, which is more in line with models à la Ngai and Pissarides (2007) and Acemoglu and Guerrieri (2008) in which structural change is compatible with balanced economic growth in the aggregate. van Zon and Muysken's (2005) and Pugno's (2006) conjecture, on the other hand, that the expenditure shift toward 'stagnant services' fosters human capital formation and thereby raises economic growth endogenously finds no support in the data. This conjecture can be translated into a requirement of significantly positive Granger-causality running from the expenditure variable to GDP growth. No evidence for positive Granger-causality was found.

In order to strengthen confidence in the results, a number of robustness tests will be carried out. First, the expenditure variable will be split into growth in real per-capita expenditure on health and on education, respectively. These two expenditure categories have been summed so far because Baumol's model suggests that they are highly collinear (in which case insignificant coefficients would have ensued). This can now be tested. In the remainder, I will rely on models with two lags and a collapsed set of instruments because these have performed best in previous tests.

Table 5 does not yield evidence for strong collinearity between the expenditure variables since in the one-step estimation the coefficients for health expenditure growth are statistically significant. In all estimations, the health expenditure growth coefficients have negative signs, which is incompatible with the predictions of the health capital augmented endogenous growth model. The coefficients for public education expenditure growth are thoroughly insignificant, as are the coefficients for the health variable in the case of the two-step estimation. These results bolster balanced growth models of structural change. Overall, the robustness test corroborates the results reported in Table 4.

< Insert Table 5 around here >

As a second robustness test, I will add physical capital to models (8) and (10). Although physical capital is absent from both Baumol's (1967) and Pugno's (2006) models, it is present in the Uzawa-Lucas model of endogenous growth as well as in van Zon and Muysken's (2005) health capital augmented version of it. Of course, physical capital is also present in the balanced growth models of structural change by Ngai and Pissarides (2007) and Acemoglu and Guerrieri (2008).

Adding physical capital as an explanatory variable is also apposite in order to meet one of the assumptions behind the Granger-causality methodology which some might regard as problematic, namely the assumption that all information not included in the stationary VAR model is irrelevant. Since investment in physical capital is usually recognized as an important driver of economic growth, its inclusion among the list of explanatory variables helps to justify this assumption.²⁵ Cohen and Soto (2007) also use physical capital formation as a control variable in order to test the robustness of their findings on the effects of human capital formation on economic growth.

Table 6 shows that when real per-capita gross fixed investment growth is added as an explanatory variable, the lags of real per-capita health and public education expenditure growth lose their joint significance even in the one-step estimation. They keep their negative

²⁵ In fact, Sturm and de Haan (2005) find that when robust estimation techniques are used, only physical capital formation and a couple of regional dummies (not relevant for my sample of OECD countries) remain significant out of a long list of possible determinants of economic growth.

sign, though, in most of the cases. Real per-capita gross fixed investment growth Grangercauses real per-capita GDP growth with a negative sign in the (OLS and) one-step estimation(s).²⁶ It does not do so in the two-step estimation, which seems suspicious, though. Collapsing the set of instruments produced a non-positive definite weighting matrix; therefore the full set of instruments was used. Still, the Sargan and Hansen test results look strange. Hence, not too much emphasis will be put on this estimation here.

< Insert Table 6 around here >

Overall, the estimation results do not yield much evidence in favor of either the endogenous growth view or the stagnationist view of the growth effects of structural change. The expenditure shift toward 'stagnant services' that we've witnessed over the last 40 years has neither Granger-caused economic growth to rise nor to decline – at least not if the impact of physical capital formation is accounted for. This finding supports the idea recently formalized by Ngai and Pissarides (2007) and Acemoglu and Guerrieri (2008) that structural change is compatible with balanced economic growth in the aggregate.

Still, there is the possibility that the results are driven by outliers. To check this, I will perform a jackknifing exercise which consists in re-estimating model (15) dropping each of the 18 countries in turn. Table 7 shows that there is indeed one outlier which has a large impact on the overall result. This outlier is Japan. If Japan is dropped from the sample, then the lags of real per-capita health and public education expenditure growth re-emerge as statistically significant explanatory variables for real per-capita GDP growth even in the presence of real per-capita gross fixed investment growth. The coefficients for both lags are negative and significant (both jointly and individually) at the 5 percent level.

Of particular interest in this context is the significance of the second lag. It can be argued that in the medium run – for instance, over a five-year period – the finding of negative Granger-causality running from investment in physical or human capital to GDP is compatible with the augmented Solow model of economic growth. In that model, a positive (negative) shock to investment leads to an instantaneous rise (drop) in economic growth followed by declining (rising) GDP growth rates over subsequent years as the economy moves back to steady state. Significantly negative coefficients for the first lags of the investment variables within a Granger-causality testing framework are therefore in line with

²⁶ The negative sign can be interpreted as yielding evidence in favor of exogenous rather than endogenous growth theories (see below).

standard exogenous growth theory (cf. Hartwig, 2009, and already Vanhoudt, 1998). Over a longer-term horizon (ten years), however, the finding of negative Granger-causality is not compatible with the augmented Solow model because according to that model, changes in investment rates in physical or human capital can have no growth effects whatsoever in the long run. Therefore, if we disregard Japan, the empirical evidence is more favorable toward the idea behind Baumol's model of unbalanced growth that structural change causes long-term economic growth to decline. – What makes Japan so different in this respect seems to be an interesting question for further research.

< Insert Table 7 around here >

6. Conclusion

The impact of structural change on economic growth is contested in the literature. Baumol (1967) famously introduced a model in which structural change - defined as the shift of employment and expenditures toward 'stagnant services' like health care and education leads to a decline in per-capita GDP growth and eventually to stagnation. This, however, contradicts one of Kaldor's 'stylized facts' according to which the growth rate of per-capita GDP is relatively stable over time. Therefore, a string of recent literature has aimed at reconciling structural change with balanced economic growth in the aggregate. Two approaches can be distinguished in this literature: a demand-side approach relying on nonhomothetic utility functions (Echevarria, 1997, Laitner, 2000, Caselli and Coleman, 2001, Kongsamut et al., 2001, Foellmi and Zweimüller, 2008) and – arguably more in line with Baumol's contribution – a supply-side approach which overrides Baumol's stagnationist outlook by introducing physical capital to the model (Ngai and Pissarides, 2007, Acemoglu and Guerrieri, 2008). Another line of literature turns Baumol's approach, so to speak, upside down, arguing that the expenditure shift toward 'stagnant services' stimulates rather than dampens long-term economic growth because of the human capital-accumulating nature of major 'stagnant services' like health care and education (van Zon and Muysken, 2005, Pugno, 2006).

So far, this literature has been mostly theoretical. The present paper follows Acemoglu and Guerrieri's (2008) call to estimate structural change models, applying a relatively new empirical methodology – the method of panel Granger-causality testing – to OECD data. The findings can be summarized as follows. First, no evidence was found in support of van Zon and Muysken's (2005) and Pugno's (2006) conjecture that the shift toward 'stagnant' – yet

human capital-accumulating – services like health care and education raises economic growth endogenously. Whether structural change is compatible with balanced economic growth in the aggregate or whether it leads to long-term stagnation is less evident from the data at hand. At first sight it appears that the shift in real expenditure toward health care and education that has occurred in OECD countries since the beginning of the 1970s had no significant effect on – in other words, did not Granger-cause – real GDP growth when physical capital growth is used as a control variable. At a second glance, however, it turns out that this bottom line is highly dependent upon the inclusion of Japan in the sample of countries. If Japan is excluded, there is a statistically significant and long-lasting negative impact of this expenditure shift (or of structural change, respectively) on economic growth. This finding is more in line with the predictions of Baumol's model of unbalanced growth than with models of balanced economic growth in the aggregate. What makes Japan so special in this respect seems to be an interesting question for further research.

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Figure 1: *Histogram of real per-capita GDP growth rates of 18 OECD countries (five-year averages, 1970-*2005)



Figure 2: Histogram of real per-capita health and public education expenditure growth rates of 18 OECD countries (five-year averages, 1970-2005)

	Gl	DPRPC		HCPEERPC			
H_0 : Unit root in level	Stat.	Prob.	Obs.	Stat.	Prob.	Obs.	
Levin, Lin & Chu t*	-12.223	0.000	108	-14.066	0.000	106	
Im, Pesaran & Shin W-stat	-4.583	0.000	108	-4.637	0.000	106	
ADF – Fisher Chi-square	93.910	0.000	108	92.918	0.000	106	
PP – Fisher Chi-square	109.230	0.000	108	126.493	0.000	106	

Table 1: Panel unit root test results (18 OECD countries, 1970-2005)

GDPRPC = real per-capita GDP growth rates (five-year averages), HCPEERPC = real per-capita health care plus public education expenditure growth rates (five-year averages)

Note: Individual intercepts are included as exogenous variables in the test equations. For the first three tests listed in the table, maximum lags are automatically selected based on the Schwarz Information Criterion. The remaining test uses the Bartlett kernel for the Newey-West bandwidth selection. The probabilities for the Fisher tests are computed using an asymptotic Chi-square distribution. The other tests assume asymptotic normality.

Table 2 : Optimal lag length for equation (1)						
Lag	1	2	3			
SIC	4.014	4.076	4.186			

SIC = Schwarz Information Criterion

	(1)	(2) (3)		(4)	(5)		
	OLS	On	e-step	Two-step			
		system	m GMM	system GMM			
		Full set of instruments	Collapsed set of instruments	Full set of instruments	Collapsed set of instruments		
GDPRPC(-1)	-0.222**	0.298***	0.142	0.330*	0.114		
	(0.104)	(0.101)	(0.120)	(0.180)	(0.239)		
HCPEERPC(-1)	-0.043	-0.048 -0.081		0.026	-0.060		
	(0.055)	(0.063)	(0.061)	(0.092)	(0.069)		
Number of obs.	108	108	108 108		108		
Sargan test	_	0.313	0.757	0.313	0.757		
(p-level)							
Hansen test	_	_	_	0.999	0.443		
(p-level)		0.02.5					
AB test	-	0.036	0.049	0.089	0.125		
(p-ievel)							

Table 3: Estimation results for equation (1), one lag

GDPRPC = real per-capita GDP growth rates (five-year averages), HCPEERPC = real per-capita health care plus public education expenditure growth rates (five-year averages)

Standard errors are in parenthesis. *, ** and *** denote significance at the 10, 5 and 1 percent levels,

respectively. Estimates for constant terms not shown. AB test = Arellano-Bond test for AR(2) in first differences.

	(6)	(7)	(8)	(9)	(10)	
	OLS	On	ie-step	Two-step		
		syste	m GMM	system GMM		
		Full set of instruments	Collapsed set of instruments	Full set of instruments	Collapsed set of instruments	
GDPRPC(-1)	-0.311***	0.261***	0.341***	0.436	0.383***	
	(0.117)	(0.101)	(0.132)	(0.326)	(0.116)	
GDPRPC(-2)	0.140	0.268**	0.383***	0.468	0.379	
	(0.125)	(0.114)	(0.149)	(0.462)	(0.496)	
HCPEERPC(-1)	-0.095	-0.116*	-0.116* -0.183**		-0.210*	
	(0.073)	(0.069)	(0.081)	(0.150)	(0.118)	
HCPEERPC(-2)	-0.052	-0.025 -0.069		-0.134	-0.038	
	(0.056)	(0.060)	(0.069)	(0.104)	(0.109)	
Number of obs.	90	90	90	90	90	
Wald test	0.332	0.243	0.076	0.330	0.191	
(p-level)		0.106	0.047	0.106	0.047	
Sargan test	_	0.196	0.847	0.196	0.847	
Hansen test	_	_	_	0.999	0.432	
(p-level)				0.777	0.132	
AB test	_	0.233	0.317	0.718	0.700	
(p-level)						

GDPRPC = real per-capita GDP growth rates (five-year averages), HCPEERPC = real per-capita health care plus public education expenditure growth rates (five-year averages)

Standard errors are in parenthesis. *, ** and *** denote significance at the 10, 5 and 1 percent levels,

respectively. Estimates for constant terms not shown. AB test = Arellano-Bond test for AR(2) in first differences.

	(11)	(12)	(13)
	OLS	One-step system	Two-step system
		GMM	GMM
GDPRPC(-1)	-0.318**	0.362***	0.480**
	(0.124)	(0.133)	(0.208)
GDPRPC(-2)	0.207	0.480***	0.730
	(0.131)	(0.154)	(0.466)
HCERPC(-1)	-0.038	-0.164*	-0.141
	(0.084)	(0.086)	(0.123)
HCERPC(-2)	-0.140 **	-0.176**	-0.197
	(0.068)	(0.078)	(0.206)
PEERPC(-1)	-0.032	-0.024	-0.107
	(0.052)	(0.053)	(0.112)
PEERPC(-2)	0.060	0.053	0.030
	(0.048)	(0.051)	(0.078)
Number of obs.	90	90	90
Wald test HCERPC	0.122	0.031	0.520
Wald test PEERPC	0.308	0.370	0.275
(p-level) Sargan test	_	0.839	0.839
(p-level) Hansen test	_	_	0.783
(p-level) AB test (p-level)	_	0.451	0.895

 Table 5: Robustness test – Splitting the expenditure variable

GDPRPC = real per-capita GDP growth rates (five-year averages), HCERPC = real per-capita health care expenditure growth rates (five-year averages), PEERPC = real per-capita public education expenditure growth rates (five-year averages)

Standard errors are in parenthesis. *, ** and *** denote significance at the 10, 5 and 1 percent levels,

respectively. Estimates for constant terms not shown. AB test = Arellano-Bond test for AR(2) in first differences.

	(14)	(15)	(16)
	OLS	One-step system	Two-step system
		GMM	GMM
GDPRPC(-1)	0.143	0.669***	0.759*
	(0.195)	(0.180)	(0.390)
GDPRPC(-2)	0.003	0.104	-0.242
	(0.186)	(0.190)	(0.466)
HCPEERPC(-1)	-0.031	-0.098	0.034
	(0.075)	(0.074)	(0.115)
HCPEERPC(-2)	-0.064	-0.062	-0.013
	(0.055)	(0.061)	(0.151)
GFIRPC(-1)	-0.197***	-0.159**	-0.238
	(0.071)	(0.066)	(0.176)
GFIRPC(-2)	-0.022	0.019	0.059
	(0.048)	(0.056)	(0.191)
Number of obs.	90	90	90
Wald test HCPEERPC	0.494	0.329	0.923
(p-level) Wald test GFIRPC	0.022	0.016	0.166
(p-level) Sargan test	_	0.163	0.000
(p-ievel) Hansen test	_	_	0.999
(p-level) AB test (p-level)	-	0.299	0.501

Table 6: Robustness test – Adding physical capital

GDPRPC = real per-capita GDP growth rates (five-year averages), HCPEERPC = real per-capita health care plus public education expenditure growth rates (five-year averages), GFIRPC = real per-capita gross fixed investment growth rates (five-year averages)

Standard errors are in parenthesis. *, ** and *** denote significance at the 10, 5 and 1 percent levels, respectively. Estimates for constant terms not shown. AB test = Arellano-Bond test for AR(2) in first differences.

GDPRPC									
	Australia	Austria	Canada	Denmark	Finland	France	Iceland	Ireland	Japan
GDPRPC(-1)	0.681*** (0.182)	0.690^{***} (0.184)	0.692*** (0.185)	0.687*** (0.186)	0.628*** (0.180)	0.660*** (0.185)	0.958^{***} (0.188)	0.300* (0.179)	0.589*** (0.175)
GDPRPC(-2)	0.092 (0.192)	0.126 (0.196)	$\begin{array}{c} 0.111 \\ (0.195) \end{array}$	0.093 (0.192)	$\begin{array}{c} 0.070 \\ (0.188) \end{array}$	0.101 (0.194)	$0.125 \\ (0.203)$	-0.181 (0.165)	0.197 (0.182)
HCPEERPC(-1)	-0.087 (0.075)	-0.100 (0.077)	-0.089 (0.077)	-0.084 (0.075)	-0.101 (0.075)	-0.102 (0.077)	-0.021 (0.074)	-0.078 (0.064)	-0.179^{**} (0.075)
HCPEERPC(-2)	-0.084 (0.063)	-0.057 (0.064)	-0.076 (0.066)	-0.068 (0.061)	-0.040 (0.062)	-0.068 (0.063)	-0.021 (0.058)	-0.024 (0.057)	-0.124** (0.062)
GFIRPC(-1)	-0.163^{**} (0.067)	-0.165^{**} (0.067)	-0.169** (0.067)	-0.156^{**} (0.068)	-0.125^{*} (0.065)	-0.157** (0.067)	-0.301^{***} (0.067)	-0.106* (0.064)	-0.114^{*} (0.065)
GFIRPC(-2)	0.023 (0.057)	0.012 (0.057)	0.017 (0.057)	0.022 (0.056)	0.044 (0.055)	0.020 (0.057)	-0.027 (0.060)	0.067 (0.050)	0.040 (0.055)
Number of obs.	85	85	85	85	85	85	85	85	85
Wald test HCPEERPC (p-level)	0.279	0.370	0.362	0.370	0.388	0.311	0.920	0.472	0.024
Wald test GFIRPC (p-level)	0.013	0.018	0.013	0.020	0.030	0.020	0.000	0.016	0.049
	Luxembourg	Netherlands	New Zealand	Norway	Spain	Sweden	Switzerland	U.K.	U.S.
GDPRPC(-1)	0.471** (0.198)	0.682^{***} (0.184)	0.620*** (0.177)	0.735*** (0.185)	0.692*** (0.178)	0.676*** (0.188)	0.667^{***} (0.182)	0.687*** (0.184)	0.709*** (0.185)
GDPRPC(-2)	$0.076 \\ (0.208)$	$0.130 \\ (0.196)$	$ \begin{array}{c} 0.033 \\ (0.188) \end{array} $	$\begin{array}{c} 0.186 \\ (0.202) \end{array}$	$\begin{array}{c} 0.230 \\ (0.200) \end{array}$	0.057 (0.196)	$0.089 \\ (0.190)$	$\begin{array}{c} 0.103 \\ (0.195) \end{array}$	0.086 (0.193)
HCPEERPC(-1)	-0.048 (0.077)	-0.083 (0.075)	-0.120 (0.084)	-0.132* (0.076)	-0.130* (0.077)	-0.119 (0.078)	-0.099 (0.075)	-0.090 (0.077)	-0.082 (0.076)
HCPEERPC(-2)	-0.084 (0.065)	-0.063 (0.061)	-0.070 (0.068)	-0.045 (0.061)	-0.076 (0.062)	-0.058 (0.064)	-0.058 (0.062)	-0.061 (0.063)	-0.056 (0.063)
GFIRPC(-1)	-0.128* (0.070)	-0.177^{***} (0.067)	-0.121* (0.068)	-0.152^{**} (0.066)	-0.158** (0.066)	-0.161** (0.070)	-0.160^{**} (0.068)	-0.165^{**} (0.068)	-0.184^{***} (0.068)
GFIRPC(-2)	$0.008 \\ (0.059)$	-0.003 (0.058)	$0.008 \\ (0.058)$	$0.006 \\ (0.056)$	$0.007 \\ (0.056)$	$0.030 \\ (0.059)$	0.022 (0.057)	$0.019 \\ (0.058)$	0.014 (0.058)
Number of obs.	85	85	85	85	85	85	85	85	85
Wald test HCPEERPC (p-level)	0.419	0.404	0.293	0.212	0.175	0.260	0.352	0.405	0.468
Wald test GFIRPC	0.103	0.013	0.148	0.040	0.024	0.014	0.016	0.015	0.007

Table 7: Robustness test – Cross-national stability of parameters, One-step system GMM estimator: excluded countries

GDPRPC = real per-capita GDP growth rates (five-year averages), HCPEERPC = real per-capita health care plus public education expenditure growth rates (five-year averages), GFIRPC = real per-capita gross fixed investment growth rates (five-year averages) Standard errors are in parenthesis. *, ** and *** denote significance at the 10, 5 and 1 percent level, respectively. Estimates for constant terms not shown.