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Understanding the effect of cancer incidence on labour productivity in the UK: An empirical approach with a health augmented production function

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Understanding the effect of cancer incidence on labour productivity in the UK: An empirical approach with a health augmented production function.

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Key words

Labour Productivity, Health Economics, Cancer, UK Productivity, Productivity Growth

Abstract

This study represents a new way of looking at health, by investigating the effect of aggregate cancer incidence rates on labour productivity, using a macroeconomic methodology. The health of the labour force is a key determinant of labour productivity, with poor health comes both physical and mental stresses that corrode the productive capacity of workers. Within this study, cancer was selected as an approximation of labour force health, given its ability to capture a range of lifestyle choices. Workers afflicted by cancer often face three choices: continue working, temporarily/permanently leave employment or retire early – all resulting in productivity loss. Moreover, the effect on productivity may not just be felt by the patient but also their family. This creates a negative externality, the result of which is additional productivity loss.

The study used an autoregressive distributed lag (ARDL) model to assess the impact of cancer rates in the short-run and long-run. The results were clear, with cancer rates having a significant short-run one year lagged effect on labour productivity. With a 10% short-run lagged increase in cancer rates, leading to a loss of -\$1711 in labour productivity per worker – using 2010 GDP per worker. In the long-run, the effect was positive suggesting cancer does not impact long-run economic growth.

This research offers a new insight into the mechanics of health within the environment of macroeconomics. With this study potentially unlocking a new avenue of productivity policy framework, aimed at health improvement rather than more traditional approaches involving training and technological advancement.

1. Introduction

In 2015, the United Nations created 17 sustainable development goals (SDGs) as part of the envisaged future of the world by 2030; detailed within the 2030 Agenda for Sustainable Development. Two goals bare themselves well to the nature of this report those being: Good Health and Wellbeing plus Decent Work and Economic Growth (UNGA, 2015). The productivity of labour has a direct connection to economic growth, with the economy able to expand output at a constant level of inputs. The Organisation for Economic Co-operation and Development (OECD) define productivity as a measurement of how efficiently production inputs such as physical and human capital are being used within the economy at a given state of output (OECD, 2021). Increased efficiency within the production function allows the economy to experience more economic growth given *ceteris paribus*, as the transition from input to output is turned into a more effective process. The idea of productivity fuelling economic growth was proposed by Adam Smith through his work on the division of labour, with specialisation leading to greater output (Adam Smith, 1776). Since then, many studies have looked further into the determinants of productivity, but perhaps one of the most unresearched areas is the effect of health.

The health of the labour force is a key determinant of productivity. Health is a component of human capital with the quality of labour being determined by the health and wellbeing of the labour force (Bleakley, 2010; Perkins *et al*, 2013). A worker that does not exhibit good health and wellbeing is likely to be unproductive, with poor health comes both physical and mental stresses that corrode the productive capacity of the workers it inflicts (Mitchell and Bates, 2011). Moreover, a worker that receives a diagnosis of a serious health condition may choose to temporarily or permanently leave the labour market, taking the experience and expertise they have collected (Bates *et al.*, 2018; Polachek and Tatsiramos, 2019). This loss of human capital and its effect on productivity is supported by Romer's endogenous growth theory which suggests that economic growth will be affected, as the knowledge held by these workers is removed from the labour force. (Doepke and Ziliotti, 2014; Romer, 1986). The effect of an adverse health event may also be felt by the wider family unit. Family and friends may have to adjust their employment arrangements, changing working hours or leaving the labour force – adding a negative externality effect on human capital and productivity loss (Veenstra *et al*, 2017; de Moor *et al*, 2017). Given this link between health and productivity, it is clear to see the possible relationship between health and economic growth – with health having both contractionary and expansionary effects on output.

This study has been motivated by the need to: examine the possible relationship between labour force health and productivity, to recommend policies to satisfy both health and economic growth SDGs and to bridge the gap between branches of economics and health sciences. There is a gap in the literature regarding the empirical investigation of aggregate health on aggregate labour productivity. Existing studies of this nature are limited by their focus on life expectancy as an indicator of health (Swift, 2011; Ullah *et al.*, 2019), this presents many limitations with the current literature as life expectancy at birth may not be the optimum health measurement for productivity. Living longer may not mean productivity has increased, an individual may live to an old age but still be afflicted by health issues throughout their life that limit their productive capacity. For example, the mortality rate of cancer is decreasing (Cancer Research UK, 2021) but many individuals that are diagnosed with cancer, still undertake intensive treatment that temporarily reduces their productivity. As a result, a health

variable that captures lifestyle choices and involves a temporary to permanent contraction in productive capacity is needed. In this study health will be approximated by all cancer incidence rates. Given cancer's ability to capture a range of external influences such as stress, diet and physical activity that not only determine cancer risk but also the general health of the labour force (Khan *et al.*, 2010). Additionally, the associated rigorous treatment plans can temporarily and permanently reduce the productive capacity of a worker (Kamal *et al.*, 2017). This study will add to the range of literature on the effects of cancer, by using a new macroeconomic approach, with much of the existing literature looking at the effect of specific cancers on a small sample by focusing on labour market outcomes (Bates *et al.*, 2018; Polachek and Tatsiramos, 2019). Productivity will be approximated by GDP per worker – which is an acknowledged measurement of productivity (Hird *et al.*, 2019; Pessoa and Reenen, 2014; Guest, 2011). With subsequent production function inputs such as: capital formation, total education spend, and total health spend being derived in a per worker form.

The objectives of this study are to: examine the effect of cancer rates on labour productivity, assess if a negative relationship exists, and recommend policies to reduce cancer rates and expand labour productivity. To achieve this, the study will derive an augmented production function which includes cancer incidence rates as a health variable. The production function inputs will then be inserted into an autoregressive distributed lag (ARDL) model to estimate the short-run and long-run cointegration relationship between the augmented production function variables, to assess the impact of cancer rates on productivity from 1979 to 2010 in the UK; this will be a Macroeconomic approach. These results will be an aggregate approximation of the effect of cancer on productivity with all legal working ages employed, work type and risk factors being included within the labour force variable. Therefore, because of this aggregation of the labour force, the results gathered from this study will suffer from a cohort effect and will likely to be an upper bound measure of the effect of health. Based on the resulting empirical evidence this study will then make policy recommendations to satisfy both health and productivity objectives.

The proceeding sections of the study will comprise of a review of relevant literature to assess the current literary output on the topic and current economic theory, in order to draw conclusions on the research gap and assist in the formation of sign expectations. A rigorous methodology consisting of a detailed summary of the data, formulation of the theoretical approach and setting the framework for the empirical approach being used. An interpretation of the results, along with a comprehensive discussion, followed by recommendations and concluding remarks.

2. Literature Review

This section of the report will examine the existing literary output to find the current knowledge consensus within the topic, in addition to where this study fills gaps within the literature. The literature review will be divided into two parts. Firstly, a review of economic theories of growth and productivity determination – this is to relate contrasting economic schools of thought to our study to understand theoretical expectations of our model results. The second section will review the current knowledge on the effects of cancer specifically on labour market outcomes in the short-run and long-run, in addition to spill over effects for the whole family unit.

This will contribute to the achievement of this study's objectives, looking into possible effects cancer has on labour productivity.

2.1. Economic growth models and determination of productivity

The understanding of different economic growth models is important for the development of this study. Within our study, labour productivity will be approximated by GDP per worker. Therefore, it is crucial to understand the influences on GDP growth over time. The classical economist Adam Smith first proposed the influence of labour productivity on GDP (Smith, 1776). He explained how specialisation within the labour force can lead to a more efficient transaction from input to output through his pin factory analogy. Since then, the theories on both the determination of economic growth and the role of productivity have been expanded to encompass a range of methods.

In addition to Smith's original ideas, this section will look at two of the main economic growth theories. The endogenous growth models suggest that growth is driven by internal systems – such as human capital and innovation (Romer, 1986; Schillirò, 2019; Perkins *et al*, 2013). The Exogenous Neoclassical growth models suggest that growth is fuelled by external systems – such as savings rate and exogenous technological progress (Solow, 1956; Perkins *et al*, 2013). Both theories have their roots within the Cobb-Douglas production function formed by Cobb and Douglas under their paper, “*A Theory of Production*” (Cobb and Douglas, 1927). Within this function variables for both physical and human capital were included, with these being multiplied by an exogenous total factor productivity (TFP) parameter. This function has been used to explain the foundation of economic growth determination with new research expanding on the role of both physical and human capital, in addition to adding new determinants. Given its foundation in modern economic theory, the Cobb-Douglas function will be used within this study. Additionally, the function will be expanded to consider the different determinants of human capital.

Within Romer's endogenous growth model he argues that knowledge is subject to increasing marginal productivity (Romer, 1986; Schillirò, 2019). This implies that there is a primary role of human capital in the expansion of economic growth and productivity, with any expansion in human capital having a greater marginal effect on productivity as knowledge, experience, and training increase. This is through the replacement of the traditional TFP parameter with a knowledge parameter, which is endogenously determined by the number of workers. This theory coincides with our study as we aim to look at the effect health has on labour productivity. If workers experience adverse health conditions, they are likely to be temporarily or permanently removed from the labour force, reducing human capital within the economy - foregoing the knowledge and experience of workers (Bates *et al.*, 2018). Knowledge is also a public good meaning that there is a spill over effect, with workers contributing knowledge to firms that may improve productivity – supporting the idea of increasing marginal productivity returns of knowledge (Romer, 1986). The idea of knowledge being a crucial element of the production function and productivity was supported by Adam Smith (Smith, 1776). His idea of the division of labour relied on workers gaining specific knowledge on their role, with this knowledge driving specialisation and productivity growth. However, Romer's theory has been criticised by Krugman who disputes the assumptions of the model (Krugman, 2013). Krugman suggests that empirically the model does not stand, as

many variables are unmeasurable – so the model cannot be proved. Whilst this may be correct the theory should not be disregarded as it still offers a new look into economic growth, which provides a new avenue of comparison to other models. Furthermore, this model also implies dual causality meaning that if a health variable was entered into the endogenous growth production function, there would have to be a bi-directional causation – something which may not exist.

Conversely, Neoclassical exogenous models present an alternative view on the determination of economic growth and productivity. One of the main exogenous growth models is the Solow model (Solow, 1956). Solow argues that the economy reaches a steady state of economic growth where given *ceteris paribus*, economic growth increases at the same rates as population growth (Perkins *et al.*, 2013). Moreover, the Solow model also relies on exogenous variables to drive economic growth – with savings and technological growth. The Solow model is limited by the assumption that productivity growth is exogenous, in the form of total factor productivity (TFP), representing technology – independent of the model (Ramanayake, 2019). This ignores the crucial role of knowledge within the production function something that is shown within the endogenous growth models such as Romer’s with knowledge being modelled within the production decisions replacing TFP (technology) (Perkins *et al.*, 2013; Romer, 1986). Additionally, the theory is also heavily focused on the capital side of the production function. With capital accumulation determined by an array of strict exogenous variables, with less emphasis on the role of human capital and its composition.

Romer’s endogenous growth model is the most transferable to this study, with the theory supporting the idea of the influence of knowledge on productivity. Cancer patients are often removed from the labour force temporarily or permanently which in turn removes the knowledge of these workers, something which both Romer and Smith would argue decreases productivity and reduces economic growth (Romer, 1986; Smith, 1776). Furthermore, Romer’s idea of the increasing marginal returns to productivity adds further weight to the need to investigate the impact of health on productivity, with this study possibly unlocking a new improvement pathway – maximising the increasing marginal productivity returns property in the form of knowledge retention and preservation of its spill over effect. However, both theories can be applied to our study. The implementation of a health variable into either of these theories would allow the effect of labour force health to be seen – extending their current explanatory power. On the other hand, a health variable may not be able to enter Romer’s model if bi-directional causality is not present – as this would violate the model’s endogeneity principle.

2.2. Productivity implications of cancer diagnosis

A cancer diagnosis is a life changing event that influences the behaviour of an individual. It takes a toll on both the physical side through extensive and regular treatment, and the mental side through the knowledge of a diagnosis and the stresses it causes (Morrow *et al.*, 2002). This raises the question as to what effect a diagnosis has on labour market participation. An individual that is removed from the labour force will result in a loss of productivity as the individual’s knowledge and expertise is forgone. Romer’s endogenous growth model suggests that because of this human capital and therefore knowledge loss, productivity will be affected (Doepke

and Ziliotti, 2014; Romer, 1986). If after investigation of the literature this is proved to be correct, this will be symbolic of a contraction in labour supply. This section of the literature review will focus on three areas: the short-run effects, long-run effects, and the negative externality effect.

Many studies have revealed a link between labour market participation and cancer diagnosis – with participation decreasing after a cancer diagnosis in the short-run (0-3 years) (Bates *et al.*, 2018; Menhnert, 2011; Polachek and Tatsiramos, 2019; Barnay *et al.*, 2019). One study states that of those with a cancer diagnosis, 46% are not participating in the labour force (Bates *et al.*, 2018). Within this study, healthy individuals had three times the probability of full-time employment compared to participants with a cancer diagnosis. However, was a study on Australian individuals with a cancer diagnosis aged 25-64, capturing only those of labour participation age. Whilst this study does not shed light on the productivity lost by the decrease in labour force participation among individuals with cancer, Romer's model would infer that the experience and knowledge that these individuals held has now temporarily or permanently been removed from the labour market pool of talent; suggesting that per worker productivity decreases (Romer, 1986). An additional study (Mehnert, 2011) supports this level of labour participation removal and offers an average time scale of return. On average there was a sizable short-run labour market removal of 60% six months post-diagnosis. Followed by a decrease to 38% at 12 months post-diagnosis, and at 24 months post-diagnosis only 11% are not participating. The additional time element founded by Mehnart builds on the previous study conducted by Bates *et al.*, suggesting that labour productivity will fall more significantly in the short-run but will slowly recover as individuals re-join the labour force over the months following a diagnosis. Conversely, an additional study has taken a more conservative view on the scale of labour force removal (Polachek and Tatsiramos, 2019). From the first year of diagnosis to the third year, the number of diagnosed women in the labour market with a permanent contract decreased from 98.33% to 95.26% and decreased from 91.63% to 88.60% for those with a temporary contract. This disputes the finding of both Bates *et al.*, and Mehnart, suggesting that a cancer diagnosis does not significantly affect the labour market participation of individuals. However, Polachek and Tatsiramos only cover a sample of women where cancer is a proportion of the adverse health conditions tested, therefore creating complexity when applying the findings to the context of our study. The other studies also have their limitations, firstly, Mehnart uses results from a sample that predominantly consists of women with a breast cancer diagnosis (Mehnert, 2011). This limits the ability of the study to draw an aggregate interpretation on the effect cancer has on labour market participation as different cancer may require different severity levels of treatment. Secondly, Bates *et al.*, conducted a study with a sample of only Australian residents (Bates *et al.*, 2018). Our study looks at the UK and therefore conditions in Australia may not be reflected within the UK – for example annual levels of ultraviolet radiation (World Health Organisation, 2017; Met Office, 2022). Which would make Australians more susceptible to skin cancer which may require less treatment than other variants of cancer. Applying the example literature to our study allows the formation of sign expectations, based on the findings of Bates *et al.*, Mehnart and Barnay *et al.*, it is expected that in the short-run an increase in cancer rates will decrease productivity (Bates *et al.*, 2018; Mehnert, 2011; Barnay *et al.*, 2019). This is something that is supported by Romer's endogenous growth model, with this short-run labour removal being symbolic of knowledge removal – which Romer argues will negatively affect productivity rates (Romer, 1986). Whilst these studies allow for the creation of sign expectations in the short-run, the long-run case also needs to be considered.

In the long-run (3+ years) diagnosed individuals face several options when assessing their labour market participation; continue working, reduce hours, leave employment or retirement (Bradley and Bednarek, 2002; De Boer *et al.*, 2020). By looking at studies that provide knowledge in this area, conclusions can be drawn on the sign expectations of the effect of increased cancer rates in the long-run. One study looks at the change in employment levels five years post-diagnosis (Bradley and Bednarek, 2002). Bradley and Bednarek found that of those employed before a cancer diagnosis 67% were employed 5-7 years later. Of those no longer employed 54% retired, 24% were disabled, 13% had other reasons. Suggesting, that there is a sustained labour force exit in the long-run among cancer survivors, with 54% choosing to retire. This is the second most populous employment outcome, after remaining in work. The survivors at this age are typically the most experienced in the labour market and an earlier exit could see productivity decrease in the long-run, as these workers can be seen to have the largest human capital contribution due to their expertise. However, the effect in the long-run may be small, these workers may have been close to the retirement decision prior to the cancer diagnosis and therefore the illness may have only triggered the inevitable choice – implying that the loss of productivity may only be small and limited to the short-run. Another study supports this loss of labour force participation in the long-run (De Boer *et al.*, 2020). De Boer *et al* reports that after six or more years post-diagnosis only 65% of cancer survivors remained in employment. This study does not offer the same detail of labour force outcomes as Bradley and Bednarek, therefore, it is hard comparing the two in terms other than employment levels – but both figures are similar. From these studies we gain an understanding about the long-run effect on employment and can conclude that the long-run effect may be insignificant, as survivors with cancer accelerate their retirement plans rather than leaving the work force spontaneously. Therefore, the sign expectations on the long-run coefficients are unclear. This study by taking a macroeconomic approach will add to these findings, providing clarity in terms of the long-run effect on labour productivity – where the existing studies have been too micro-specific to do so.

It is also important to note that a cancer diagnosis does not just affect the individual it afflicts but, possibly the whole family unit. This is a research gap within the literature with few studies looking into the effect beyond the diagnosed individual. If a link to family productivity and employment outcome does exist, then there would be a negative externality effect of lost productivity as a result of cancer diagnosis. Two studies support the existence of a negative externality effect in terms of collective loss of productivity (Veenstra *et al.*, 2017; de Moor *et al.*, 2017). One study looks at the effect that a breast cancer diagnosis has on the long-run economic and employment outcomes of women's partners (Veenstra *et al.*, 2017). This study reported that out of 517 participants 32% reduced their hours as a result of diagnosis and a further 51% were reported to be worried about job security in order to keep up with health insurance payments. By looking at these findings from a productivity perspective, it could be seen that productivity falls temporarily as these individuals are being removed from the labour force. This could be because of a loss of human capital. The knowledge and experience of these individuals have now been foregone as they exit the labour force – with their former job duties being given to either new, unexperienced hires or adding to the workload of existing employees. Another study further adds to our understanding of cancer's effect on the family unit (de Moor *et al.*, 2017). This is a study on informal cancer caregiving, where individuals close to someone with a cancer diagnosis assist in the

care of that person – where care is not their employment nature. Of these informal carers 38.9% made changes to their work schedule, workload or employment hours. This finding supports the result of Veenstra *et al.* and adds to the argument that the effect of a cancer diagnosis is not felt solely by the individual with cancer but by those close to this person. This suggests that there exists a negative externality effect, with cancer affecting the productivity of both the cancer patient and their close circle. These studies do have their limitations, both studies have collected their data from individuals from the United States. This makes comparing these results to a UK scenario problematic, as the financial burden of health insurance is not prevalent – the reported figures of employment adjustment in both studies may be higher in the UK as family members feel comfortable leaving employment because of the safety net of national healthcare. Therefore, when applying these studies to the UK we can view these figures as conservative – with the UK possibly having a larger negative externality effect on productivity loss than seen in the US.

Overall, based on this wide supply of literature we can assume in the short-run there should be a negative effect on productivity given an increase in cancer rates. In the long-run the expectation is unclear with a large proportion of employees taking a retirement, something which may have been inevitable whether a diagnosis occurred or not. This uncertainty over the long-run effect is a literature gap that this study seeks to add clarity to. It is also suggested that a negative externality effect exists on productivity as those around a cancer patient will often take time off/ leave work, to support the patient. Much of the literature above is case specific looking at certain cancers and its effect on a limited number of individuals that may or may not be within the UK. This leaves the possibility that the samples used may not be a representation of the whole UK population – which may explain the inconclusive results on the long-run effect of cancer rates. To correct for this, the study will build on this from a macro time-series perspective, offering a new avenue of comparison into cancer's effect on labour productivity. The short-run and long-run effects will be reported, with the studies mentioned above providing insight into possible explanations of results. The following section will comprise of the methodology where the theoretic and empirical framework of the study will be explained.

3. Methodology

This section of the study focuses on the data and the empirical framework being used within the model. The first section will define the variables and the sources they have been collected from, so that a foundation of understanding is built. Further to this, the first section will contain descriptive statistics on the model variables. Following this, the second section will use graphical visualisation to establish the trend within the study period 1979-2010. Proceeding this will be sections where the production function is derived into the study context with the theoretical model being established, in addition to the formation of the autoregressive distributed lag model.

3.1. About the of data

Secondary data has been used within this study, collected from a variety of reputable data sources. A detailed description of all the variables used within the study can be found in *Appendix 1* – the nation of study is the United Kingdom. Three data sources were used to collect the five variables used with these being: The World

Bank, OECD, and Cancer Research UK. GDP, capital formation and education spend were collected from the World Bank's open data bank (World Bank, 2022). The World Bank's data library contains an array of macroeconomic data, with much of the data from the national statistical institute of the country – in our case the Office of National Statistics (ONS). Therefore, inaccuracies within the data are rare. Annual labour force and health spend were collected from the OECD's databank (OECD, 2022). Similarly to the World Bank, the OECD statistical database contains a large array of data from national statistical institutes. Lastly, cancer incidence rates were gathered from a Cancer Research UK open data sheet, that consists of observed European age standardised UK cancer rates from 1979 (Cancer Research UK, 2017). This data compiled by Cancer Research UK was based on information from (Smittenaar *et al*, 2016) which collects information from: England's National Cancer Registration and Analysis Service (NCRAS) from 1979, Scotland's Information Services Division (ISD) from 1979, and from Wales the Welsh Cancer Intelligence Surveillance Unit (WCISU) from 1979. Data from Northern Ireland was collected from the Northern Ireland Cancer Registry (NICR) from 1993. From 1979 to 1993 the Great Britain incidence data was scaled to the UK level to correct for this. This data set has been referred to by many published studies (Kim *et al*, 2018; Seesaghur *et al*, 2021; Liposits *et al*, 2022). The time frame of the study is limited by the availability of historic annual data on cancer incidence rates and the existence of recent yearly data, therefore the time frame of the study will be between 1979 and 2010. The variables used and their representation can be found below in *table 1*. These variables are all in their logarithm form so that a log-log function form can be used within the interpretation of the ARDL model results.

Table 1: Description of model variables

Variable	Description
Ly	Logarithm of GDP per Worker
Lk	Logarithm of Capital per Worker
Ledu	Logarithm of Education Spend per Worker
Lhs	Logarithm of Health Spend per Worker
Lallc	Logarithm of Age Standardised Incidence Rates of All Cancers per 100,000

With the sources of the data established it is important to assess the statistical properties of the data being used. Several summary statistics have been generated and are displayed within *table 2*.

Table 2: Summary Statistics

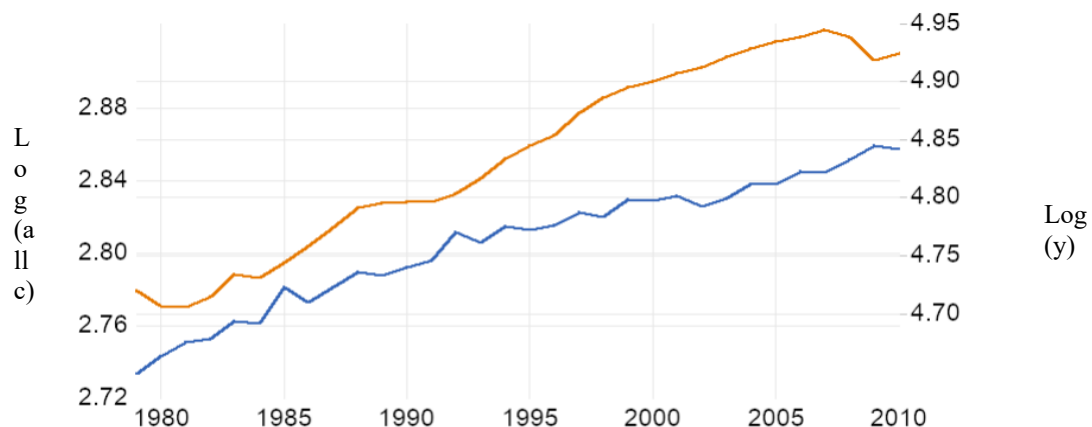
Summary Statistics	<i>Ly</i>	<i>Lk</i>	<i>Ledu</i>	<i>Lhs</i>	<i>Lallc</i>
Mean	4.8356	4.0148	3.5016	3.6426	2.8056
Median	4.8392	4.0354	3.4960	3.5969	2.8135
Minimum	4.7053	3.3994	3.3994	3.3949	2.7335
Maximum	4.9449	4.1731	3.6805	3.9174	2.8588
Variance	0.0066	0.0147	0.0079	0.0320	0.0012
Standard deviation	0.0815	0.1211	0.0887	0.1789	0.0351
Count	32.0000	32.0000	32.0000	32.0000	32.0000

Whilst these summary statistics provide a good indication of the properties of the variables it is also important to visualise their trends over time. This will be shown within the following sections with a breakdown of all the time-series data, describing how they move across the study period of 1979 to 2010.

3.1.1. Cancer incidence rates

All cancer incidence rates have been on a consistent increase since 1979, growing by 33% from the study period of 1979 to 2010 (Cancer Research UK, 2017). This has been in conjunction with an increase in GDP per worker (labour productivity) of 60% during the same period (World Bank, 2022). *Figure 1* shows these variables in logarithm form and their movement over time.

Figure 1: Logarithm of GDP per worker and Logarithm of cancer rate over 1979-2010

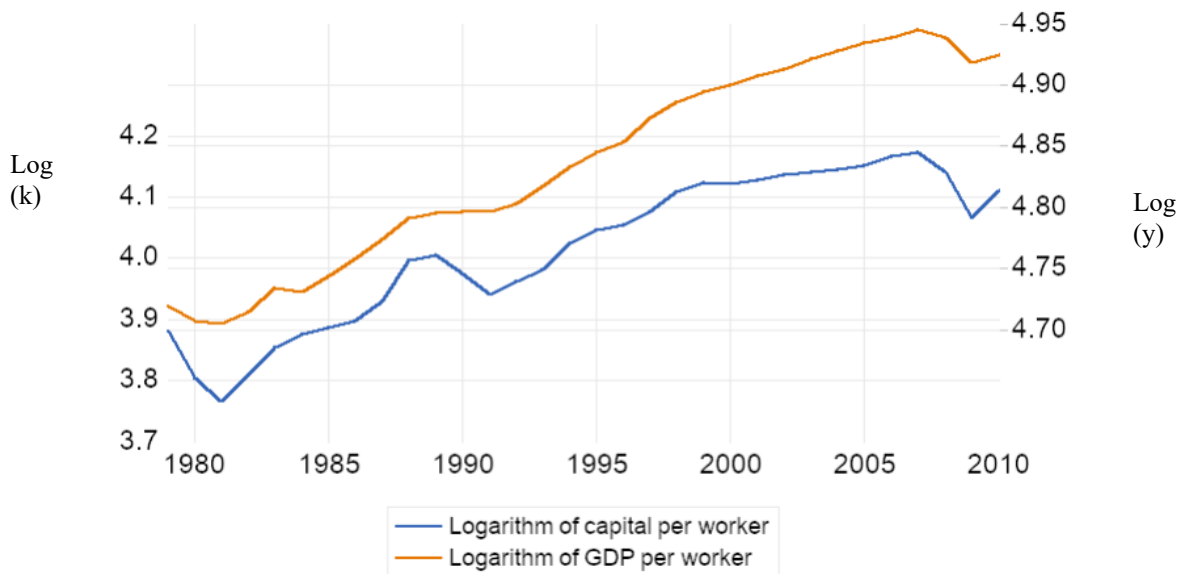


The trends of both data sets are positively correlated with a positive trajectory, increasing over time. Cancer rates do not appear to be affected by the business cycle, with the impact of recession not reflected in incidence rates. The variance of both data sets is relatively low with fluctuations over time being relatively small.

3.1.2. Capital per worker

Capital per worker has been selected to enter the model because of its key role in the determination of output and productivity. This importance was expressed by many theories of economic growth; Neoclassical and endogenous schools of thought are all in consensus on its role (Solow, 1956; Romer, 1986). From 1979 to 2010, capital per worker has increased by 70% (World Bank, 2022). Over the period GDP per worker increased 60% (World Bank, 2022). *Figure 2* depicts these variables in logarithm form and their movement over time.

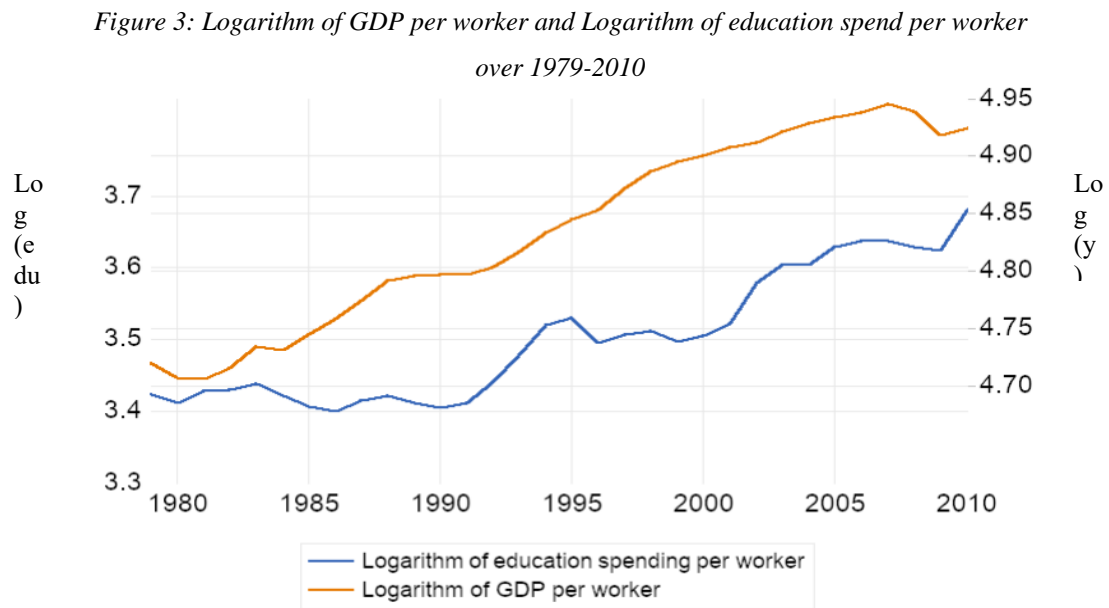
Figure 2: Logarithm of GDP per worker and Logarithm of capital per worker over 1979-2010



During this period, the logarithm of capital per worker had a larger variance being almost double that of the logarithm of GDP per worker. Looking at the movements between the two variables, both seem to be a mirror reflection of each other. However, when recessions hit, for example in 2008, capital per worker contracts to a greater extent.

3.1.3. Education spend per worker

The importance of knowledge in the determination of productivity was suggested by Romer (Romer, 1986). As education spending on primary, secondary and tertiary education institutions increase, the knowledge stock of the economy increases which in turn increases aggregate productivity. The influence of education has been empirically proven with education investment having a significant effect on productivity growth (de la Fuente, 2011). *Figure 3* shows this variable in logarithm form and its movement over time.



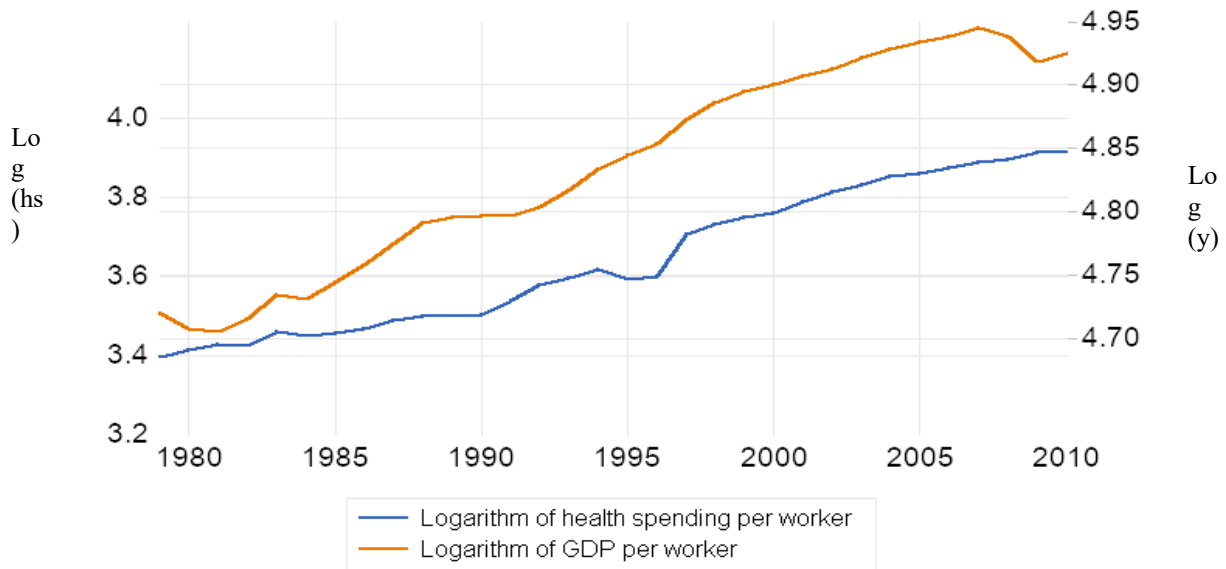
During the study period, education spending per worker, which is inclusive of primary, secondary and tertiary institutions, increased by 81%. Education spending does not appear to be influenced by the business cycle, with education spending increasing in 2008-2009.

3.1.4. Health spend per worker

Health spending has been included within the model for several reasons. Firstly, empirical studies have shown the existence of a cointegrating relation between health spending and GDP (Bloom *et al*, 2001; Raghupathi and Raghupathi 2020). These previous results suggest that health expenditure is a determinant of economic growth, adding relevance for its inclusion within our study. Moreover, the health of the labour force forms a key component of human capital within the economy (Bleakley, 2010; Perkins *et al.*, 2013). As a result of this,

health expenditure can be seen as creating an improvement in the health of the labour force, and therefore offers an avenue of further investigation for this study to explore – in addition to the principal research on the effect of cancer rates. *Figure 4* shows this variable in logarithm form and its movement over time.

Figure 4: Logarithm of GDP per worker and Logarithm of health spend per worker over 1979-2010



Out of all the variables within our model health spending per worker has increased the most over the study period, increasing by 233% in 32 years.

3.2. Deriving the production function

To establish the theoretical basis of our model, this study follows in the footsteps of many other studies by deriving the production function (Dua and Khandelwal, 2019; Romer, 1986; Solow, 1956), into a form that is augmented to our study's aim of investigating the effect of cancer rates on productivity. We will start with the Cobb-Douglas production function, where there are two inputs: physical capital (K_t) and human capital (H_t), along with an exogenous constant representing total factor productivity (TFP) (A_t). These inputs equal the GDP of the UK economy (Y_t). This function is shown in equation 1. By setting the restriction ($0 < \alpha < 1; 0 < \beta < 1$) this allows for the possibility of non-constant returns to scale.

$$Y_t = A_t K_t^\alpha H_t^\beta \quad (1)$$

$$0 < \alpha < 1; 0 < \beta < 1$$

With the aggregate production function formed we now derive the function to a per worker form by dividing by the total labour force. The results of this are seen in equation 2.

$$y_t = A_t k_t^\alpha h_t^\beta \quad (2)$$

This equation now shows all inputs and outputs in per worker form. With (y_t) symbolising GDP per worker, the labour productivity measurement this study will be using. (k_t) representing capital per worker and (h_t) representing human capital per worker. With the equation now in per worker form we can derive the inputs of human capital per worker (h_t), the results of which are shown in equation 3.

$$\sum_{t=1979}^{2010} h_t^\beta = \sum_{t=1979}^{2010} (edu_t^\gamma \times hs_t^\delta \times allc_t^\omega) \quad (3)$$

Within the study human capital per worker (h_t) will be comprised of: total education expenditure (edu_t), total health expenditure (hs_t) and all cancer incidence rates ($allc_t$). As this study will use a log-log function form within its empirical analysis, the function will be depicted in their logarithm form. Equation 4 shows the per worker production function in log form. Equation 5 shows the per worker composition of human capital in log form.

$$\ln(y_t) = A_t + \alpha \ln(k_t) + \beta \ln(h_t) \quad (4)$$

$$\sum_{t=1979}^{2010} \beta \ln(h_t) = \sum_{t=1979}^{2010} (\gamma \ln(edu_t) + \delta \ln(hs_t) + \omega \ln(allc_t)) \quad (5)$$

3.3. Autoregressive distributed lag model (ARDL) set up

This study will use the autoregressive disturbed lag model (ARDL) approach to cointegration in order to assess whether there exists a cointegrating relationship between the variables, in addition to discovering the short and long-run relationships within the model. This model has been used within previous health economics studies (Andres and Halicioglu, 2010; Ullah *et al.*, 2019). The ARDL approach to cointegration is a relatively recent advancement in econometric modelling, developed by Pesaran *et al* in 2001. (Pesaran *et al.*, 2001). Below is equation 6 which represents our application of the ARDL process.

$$\begin{aligned} \Delta Ly_t = & A_0 + \sum_{i=1}^{q1} a_1 \Delta Ly_{t-i} + \sum_{i=0}^{q2} a_2 \Delta Lk_{t-i} + \sum_{i=0}^{q3} a_3 \Delta Ledu_{t-i} \\ & + \sum_{i=0}^{q4} a_4 \Delta Lhs_{t-i} + \sum_{i=0}^{q5} a_5 \Delta Lallc_{t-i} + a_6 Ly_{t-1} + a_7 Lk_{t-1} + a_8 Ledu_{t-1} + a_9 Lhs_{t-1} + a_{10} Lallc_{t-1} + v_t \end{aligned} \quad (6)$$

One of the main objectives is to examine the existence of a long-run relationship between the dependant and independent variables. To test for this cointegration a bounds test will be applied to the model through F-statistics, with the null hypothesis being ($H_0: a_6 = a_7 = a_8 = a_9 = a_{10}$). If there is a long-run cointegration relationship within the model then the null hypothesis will be rejected. The rejection criteria will be based on the relation of the F-statistic test value to the bound values. If the F-statistic is above the upper bound of the bounds test interval at the 5% level of significance, then a cointegrating relationship has been established. Conversely, if the F-statistic falls below the 5% lower bound interval then we fail to reject the null. An inconclusive result would occur if the F-statistic falls in between the bounds test intervals.

The ARDL model does have benefits which make it advantageous to apply to this study. As the model is a single line equation the issue of endogeneity is less pronounced, this is because the model is relieved of residual correlation (Nkoro and Uko, 2016). Moreover, both the short-run and long-run coefficients are generated. The model requires all variables to be stationary at their level $i(0)$ and/or at the first difference $i(1)$. This will be tested through the implementation of the Augmented Dickey-Fuller (ADF) test and the Phillips-Perron (PP) test.

The ARDL model will also be extended into an error correction representation (Peraran *et al.*, 2001). This will be beneficial for our study as it allows us to determine a speed of adjustment parameter. This being the speed at which y_t adjusts to the independent variables within the model. A representation of the ARDL error correction model can be found below in equation 7.

$$\Delta Ly_t = A_0 + \sum_{i=1}^{q1} a_1 \Delta Ly_{t-i} + \sum_{i=0}^{q2} a_2 \Delta Lk_{t-i} + \sum_{i=0}^{q3} a_3 \Delta Ledu_{t-i} + \sum_{i=0}^{q4} a_4 \Delta Lhs_{t-i} + \sum_{i=0}^{q5} a_5 \Delta Lallc_{t-i} + \lambda EC_{t-1} + v_t \quad (7)$$

The error correction term EC_{t-1} replaces the level variables within equation 5. If this error term is significant, it shows that there exists a cointegrating relationship between the variables – providing another avenue of testing for cointegration within the ARDL model. The ARDL model will automatically select the optimal lag length for each variable that best suits the chosen criterion.

The results of the study will incorporate several diagnostic statistics. This is to determine that the model has been correctly specified and that all assumptions hold. Among these tests will be the Breusch–Godfrey LM test (Breusch, 1978; Godfrey, 1978). This is a test of serial correlation between the residuals and therefore tests for the presence of autocorrelation, which creates biased results. Normality and heteroskedasticity tests will also be

applied to further add creditability to the model specification. Cumulative sum control (CUSUM) and cumulative sum control of squares (CUSUMSQ) graphs will also be applied. These test for stability within the model given any unexpected changes with the results being displayed graphically. This study will also use the Ramsey reset test to detect model misspecification in the ARDL model. This empirical analysis will be conducted through EViews, a statistics program.

4. Results

The following section will comprise of statistical preparation of the ARDL model, checking the stationary of the variables and the existence of cointegration. This will be followed by the results of the ARDL model. The results of the model will be statistically interpreted within this section, with the proceeding discussion offering a detailed contextual analysis of the results.

4.1. Unit root tests and Bounds Test

To progress with the next steps within the methodology the stationary of the variable must be established. *Table 3* shows the results of the ADF and PP tests.

Table 3: Tests for unit-roots within variables

Variable	ADF				PP			
	i(0)	<i>k</i> lags	i(1)	<i>k</i> lags	i(0)	<i>k</i> lags	i(1)	<i>k</i> lags
Ly	-1.0707	1	-3.5749*	1	-0.8288	1	-4.0581*	0
Lk	-1.2191	2	-6.0316*	1	-2.1606	2	-4.6016*	21
Ledu	-2.1723	1	-2.3976	5	-1.7592	5	-3.0975*	6
Lhs	-2.4077	1	-4.7177*	1	-2.1738	6	-5.7866*	14
Lallc	-3.4465	0	-10.3631*	0	-3.3687	2	-10.7233*	1
Test for unit-roots in variables. The null hypothesis is the variable does have a unit-root. * indicates a rejection of the null hypothesis at the 5% level of significance								

From the table above we can see that all variables are stationary at their 1st difference, *i(1)*. This means the requirement of stationarity for the ARDL method is filled, meaning estimation can continue. A notable result is that of *Ledu* where we fail to reject the null in the ADF test suggesting that the variable is non-stationary in both its level and 1st difference. However, the PP test indicates that the variable is stationary in its 1st difference. In this situation the existence of a positive test is dominant over the ADF test result – one positive test is enough to

conclude stationary. However, the possible effects of the ADF test result and its implications on the model will be tested further. The ADF test result may be a result of a structural break within the data, because of this possibility the CUSUM and CUMSUMSQ tests will be applied to the model to indicate if the model suffers from a possible break by testing for model stability. If this test reports a result within the upper and lower 5% significance bounds, then analysis can continue – *see appendices 9 and 10*.

With the stationarity conditions of the ARDL model filled we can now assess if there is a cointegration relationship in the model. To do this the bounds test is applied. *Table 4* shows the results of the bounds test – *see appendix 2*.

Table 4: Bounds test for cointegration results

Bounds test F-statistic intervals					
90%		95%		99%	
i(0)	i(1)	i(0)	i(1)	i(0)	i(1)
2.525	3.560	3.058	4.223	4.280	5.840
Calculated model F-Statistic					
F _c (<i>Ly</i> <i>Lk</i> , <i>Ledu</i> , <i>Lhs</i> , <i>Lallc</i>)			5.946*		
* indicates a rejection of the null hypothesis at the 5% level of significance					

From the above results we can see that cointegration does exist within the model variables, suggesting that a long-run relationship does exist. This is because the model f-statistic is greater than the upper bound of the 95% interval ($5.946 > 4.223$). This means we can reject the null hypothesis ($H_0: a_6 = a_7 = a_8 = a_9 = a_{10}$) – *refer to equation 6*. With the presence of cointegration within the model confirmed we can now move to estimate the ARDL model, displaying both long-run and short-run coefficients – in addition to running model diagnostics. *Table 5* shows the overall results of the ARDL model for this study – *see appendices 2 to 10*.

4.2. ARDL model results

Table 5: ARDL model results

Long-run estimated coefficients from ARDL approach to cointegration, Automatic lag selection (3,3,1,2,2) based on Akaike Information Criterion, 1979-2010.			
Dependant variable Ly_t			
Regressor	Coefficient	Standard error	T-statistic
C	1.8629*	0.1106	16.8482
Lk_t	0.3835*	0.0199	19.1863
$Ledu_t$	0.0719*	0.0183	3.9321
Lhs_t	0.1187*	0.0165	7.1912
$Lallc_t$	0.2668*	0.0572	4.6678
Error correction and short-run results			
Dependant variable ΔLy_t			
Regressor	Coefficient	Standard error	T-statistic
ΔLy_{t-1}	0.7744*	0.1487	5.2086
ΔLy_{t-2}	0.3974*	0.1267	3.1351
ΔLk_t	0.2676*	0.0227	11.8123
ΔLk_{t-1}	-0.2919*	0.0563	-5.1899
ΔLk_{t-2}	-0.2129*	0.0457	-4.6626
$\Delta Ledu_t$	0.0225	0.0307	0.7325
ΔLhs_t	0.2011*	0.0267	7.5392
ΔLhs_{t-1}	-0.0797**	0.0280	-2.8466
$\Delta Lallc_t$	-0.0405	0.1047	-0.3864
$\Delta Lallc_{t-1}$	-0.2036**	0.0931	-2.1873
EC_{t-1}	-1.7606*	0.2505	-7.0283
Diagnostics			
\bar{R}^2	0.9424 λ_{LM}	2.3340 λ_N	3.9574
F-stat	5.9459 λ_H	13.695 λ_R	-0.3080
Tables 5 notes: * 1%, ** 5%, ***10% levels of significance represented. λ symbol represents test: LM - LM test for serial correlation, H - Bruesch-Pagan-Godfrey heteroskedasticity test, N - Normality test, R - Ramsey reset test for misspecification. With LM and H following adjusted R^2			

Table 5 shows the results of the ARDL model. Given that the bounds test results in *Table 4* confirm the existence of a cointegrating relationship we can proceed with the interpretation of the long-run coefficients. As the variables are in their log-log function form the coefficients can be interpreted as an elasticity. All the coefficients are significant at the 1% level. In the long-run a 10% increase in capital per worker will lead to a 3.84% increase in productivity. A 10% increase in education expenditure per worker will lead to a 0.72% increase in productivity in the long-run. If health expenditure per worker increases by 10% this will lead to a 1.2% increase in productivity in the long-run. When incidence rates for all cancers increase by 10% this leads to a 2.7% increase in productivity.

Looking at the short-run results we find that an increase of 10% in current capital per worker leads to a 2.7% increase in productivity. However, the lagged effects of increased capital per worker are negative. Given a 10% increase, the one-year lagged capital per worker variable has a -2.9% effect on labour productivity. Similarly, a 10% increase in the two-year lagged variable of capital per worker leads to a -2.1% decrease in labour productivity. All the short-run capital per worker coefficients are significant at the 1% level. This model also finds that the effect of an increase in education expenditure per worker in the short-run is not statistically significant. If current health expenditure per worker increases by 10% this will lead to a 2% increase in labour productivity in the short-run. This result is significant at 1%. However, if we increase the one-year lagged health expenditure per worker variable, this will lead to a -0.8% decrease in labour productivity. This is statistically significant at the 5% level. This model finds that the effect of current cancer incidence rates is not statistically significant. However, the one-year lagged cancer incidence variable does have a statistically significant result at the 5% level. Given a 10% increase in one-year lagged cancer incidence rates, labour productivity will decrease by -2%. The error correction term which captures the dynamic relationship between the short-run and long-run coefficients, is also significant showing that there is a strong dynamic relationship.

Diagnostic tests have also all been favourable for the model. The LM test indicated that the model has no serial correlation between the residuals, with the null hypothesis of no serial correlation failing to be rejected at 5%. The Breusch-Pagan-Godfrey heteroskedasticity test also confirmed that the residuals are homoscedastic, as we fail to reject the null hypothesis of homoscedasticity at 5%. The residuals of the model are normally distributed, with the null hypothesis of normality failing to be rejected at the 5% level. The model is correctly specified, with the Ramsey reset test showing a favourable result. Finally, there is stability within the model, with both the CUSUM and CUSUMSQ graphs falling within the 5% upper and lower bounds – *see appendices 9 and 10*. This positive result from the CUSUM and CUSUMSQ test indicates that a possible structural break within the education variable is not affecting the stability of the model. In addition, the adjusted R squared is 0.94, which suggests that 94% of the variance within the dependant variable (labour productivity) is explained by the independent variables. With the results now interpreted the next section will comprise of the discussion where these results will be compared with the findings within the literature review, with possible explanations of the results being investigated.

5. Discussion

This section of the study will focus on the contextual application of the results from *table 5*, relating the finding to existing literature to explain what the results mean for the topic. In addition, this section will look at the limitations involved with the model and its results gathered; to critically evaluate the methodology. There will also be a section on policy recommendations based on the evidence from the results and this discussion, where this study will look at how to maximise labour productivity in the presence of cancer rates.

5.1. Discussion of results

The ARDL model has successfully confirmed a relationship between cancer rates and labour productivity, whilst meeting all the ARDL criteria and satisfying all diagnostic tests. Starting with the short-run results most variables followed sign expectations. Capital per worker had a positive effect on productivity, with a 10% increase approximately increasing annual labour productivity by \$2185 per worker – using 2010 labour productivity. This result is supported by the literature. As capital inputs increase it enables workers to produce more within a given amount of time, expanding their productive capacity and increasing output (Owyang and Shell, 2018). Furthermore, this result is also supported by economic theory with both Solow and Romer's models suggesting that an increase in capital will lead to increases in output (Solow, 1956; Romer, 1986). The short-run coefficient on education expenditure is statically insignificant, but this may be due to the nature of the variable. The education expenditure variable captures spending on; primary, secondary, and tertiary institutions – with the labour force variable containing working individuals aged 16+. It would therefore be reasonable to suggest that the education variable may not be capturing any short-run effect as the majority of workers are not engaged in education, and therefore do not benefit in the short-run from an increase – supported by the existence of a significant long-run result. Applying the short-run result of health expenditure, a 10% in health spending would lead to a \$1774 increase in annual labour productivity – using 2010 labour productivity. This result suggests that a healthier work force leads to an increase in workers' productive capacity. Many studies support this result (Raghupathi and Raghupathi, 2020; Bloom *et al*, 2001). Bloom *et al* found that as life expectancy increases so does labour productivity, supporting our conclusion that an increase in health spending per worker leads to an increase in labour productivity. As health spending increases treatments may become less invasive, allowing workers to undergo treatments whilst not being removed from the labour force. Health conditions given increased health spending may be diagnosed earlier and more accurately preventing conditions from worsening over time. Moreover, access to quality healthcare may improve, as the quality of local facilities increase – relieving the pressures of geographical immobility of healthcare.

The main focus of this report is the effect of cancer rates on productivity, and as such the coefficients on the short-run cancer rates offer an interesting interpretation. The non-lagged cancer incidence coefficient is statistically insignificant. However, the one-year lagged cancer incidence rate coefficient is significant, with a 10% increase in incidence leading to a loss of -\$1711 in labour productivity per worker – using 2010 labour productivity. This is the equivalent of a loss of -\$54 billion in lost GDP – using 2010 GDP. This means that the

previous year of incidence rates influences current year labour productivity, in the short-run. Possible explanations for this result could be that after the first year of diagnosis individuals are more likely to leave the labour force, rather than straight after diagnosis. This explanation is supported by several studies (Polachek and Tatsiramos, 2019; Barnay *et al*, 2019). Both studies support an employment participation decrease from the year following diagnosis. This would mean that as individuals diagnosed with cancer leave the labour force, human capital decreases along with an exit of the knowledge held by that individual – something that Romer’s endogenous growth model suggests leads to a reduction in productivity, which is what is seen in the model results (Romer, 1986). Moreover, the effect of treatment on the physical and mental wellbeing of a patient may not occur within the year of diagnosis but peak after, given that cancer is a progressive disease (Robinson *et al*, 2015) – reducing their productive capacity. In addition to this a spill-over effect may exist, with cancer being a progressive disease family and friends may take time off work to assist in care giving activities – resulting in lagged negative externality effects. As discussed within the literature review this is supported by de Moor *et al*, who describe how informal caregiving by the family unit reduces not just the patient’s labour participation but that of the family member (de Moor *et al*, 2017). This lagged effect supports the hypothesis that cancer rates effect labour productivity, proving that the effect is significant in the short-run. Both explanations could be symbolic of a lack of succession planning within organisations. Cancer is often an unexpected adverse health event so the failure to implement sufficient succession plans may lead to a temporary decrease in total labour productivity, as the organisation has not planned for a skill gap to be filled. This may also be symbolic of a labour supply constraint, with the organisation not able to find individuals with the experience or knowledge needed to carry out the vacated role. However, despite cancer incidence rates having a negative effect on labour productivity in the short-run, what is the relevance in the long-run? This idea of short-run relevance relates to the business cycle theory proposed by Keynes in his “*General theory*” (Keynes, 1936). Within his theory despite short-run deviations, GDP reverts to its long-run level – but this return to the full employment level could be achieved quicker given intervention. Arguably this is what the model has shown, with a significant short-run negative effect of cancer rates, but a significant long-run positive effect of cancer rates. But despite this the short-run does still matter. On a micro-level this model proves that labour productivity is reduced, which may be a result of labour market exits – reducing the welfare of these individuals. It would therefore it could be seen as immoral to disregard the falling wellbeing in the short-run given the argument of long-run convergence. Taking the Keynesian view, the short-run contraction in productivity due to cancer incidence rates needs to be corrected to reach the long-run convergence quicker and to also avoid any lasting short-run impact. This view gives us the rationale to proceed with recommendations that relieve the short-run contractionary pressure of cancer incidence rates.

The model has reported all the variables as significant in the long-run. In addition, all variables have been reported as having a positive effect on labour productivity, including cancer incidence rates. Starting with the physical capital effect on labour productivity, the results show that a 10% increase will lead to an increase of \$3279 in labour productivity gains – using 2010 GDP per worker. This is supported by both Romer’s model and the Solow model (Solow, 1956; Romer, 1986). Both these theories support the idea of long-run expansions in labour productivity given greater capital intensity. Greater capital accumulation leads to an expansion in the economic capacity of the economy. However, within the Solow model this capital accumulation is dependent on

the rate of investment being greater than the rate of depreciation (Solow, 1956). The result is also supported empirically (Guest, 2011; Owyang and Shell, 2018), these studies found a similarly positive effect. Education per worker also has a significant positive effect on labour productivity, a contrast to its short-run effect. A 10% increase in productivity leads to a \$588 increase in labour productivity – using 2010 GDP per worker. As discussed briefly earlier this may be because of the nature of the education variable. The variable captures primary, secondary and tertiary education spending. As this is a long-run result, significance in this rather than in the short-run may be because the beneficiaries of this spending have entered the labour force – applying the better funded education to the workplace, enhancing their productivity. Similar long-run conclusions were found in many studies (Mallick *et al*, 2016; Forbes *et al*, 2010). The long-run effect of health expenditure per worker was positive and significant. A 10% increase in health spending leads to a \$998 increase in labour productivity gains – using 2010 GDP per worker. This supports our study’s hypothesis that a healthier labour force leads to a more productive labour force – with increases in health expenditure per worker symbolic of improvements in quality of care and treatment. Moreover, this provides a greater mandate for the policy recommendations to follow. Not only will the policies reduce the short-run effect of cancer incidence rates, but they will also stimulate long-run productivity growth through the health spending channel. This idea of health driven productivity gains are supported by many studies (Raghupathi and Raghupathi, 2020; Bloom *et al*, 2001).

The long-run effect of cancer incidence rates on labour productivity offers an interesting insight. The long-run result suggests that given a 10% increase in cancer rates, \$2242 will be added to labour productivity gains – using 2010 GDP per worker. This suggests there is a long-run convergence with the effect of cancer not having a negative effect on labour productivity. This seems counter-intuitive, as in the long-run given the mortality rate of cancer we would expect the labour force to decrease, reducing the stock of knowledge within the economy – resulting in contractions in productivity and economic growth, something supported by the mechanics of Romer’s model (Romer, 1986). However, there may be exogenous forces that influence the risk of cancer but also increase the productivity of workers. For example, the addition of technological change may increase the productivity capacity of workers in the long-run, but this same technology may lead to a more sedentary lifestyle, a lack of physical activity associated with an increased cancer risk (Kerr *et al*, 2017; Marchand *et al*, 1997). This result provides insight into future avenues of research with the investigation of bi-directional causality between productivity and cancer rates, but also the investigation into the determination of cancer rates.

Overall, the results have proven the negative effect cancer rates have on labour productivity, but only in a lagged short-run case. This means that cancer incidence does not have a lasting impact on the long-run equilibrium level of labour productivity and economic growth. Within this section reasons for this have been discussed with these being: a short-run removal of labour – resulting in a contraction in knowledge capital within the economy, and a negative externality effect on the family unit – with informal carers having to leave work to care for ill family members. These are all derived from the fact that cancer is a progressive disease justifying the existence of a lagged effect. The following section will examine ways to relieve this short-run contractionary pressure so that the positive long-run effect can be achieved sooner, in addition to the argument of improving welfare among these individuals.

5.2. Policy recommendations

With the existence of short-run contractionary pressures on labour productivity being confirmed, this section will look at possible policies and recommendations to reach the long-run positive equilibrium sooner – minimising the effect on these short-run contractions.

As discussed in the previous section, with the effect only being felt in the short-run, this may be symbolic of firms being unable to fill the skill gap left by individuals that leave due to cancer. This suggests two possible issues: there may be a labour market constraint with not enough experienced workers to fill high value-added roles, and a lack of internal succession planning within firms. Therefore, the first recommendation of this study is to attempt to relieve this labour supply constraint. Cancer is an unexpected adverse health event with often little indication of its existence until diagnosis. It is therefore important to have adequate succession planning within firms. This succession planning prepares firms for the eventuality that experienced and skilled workers may be unexpectedly removed from their positions, not just due to cancer but also a range of other unexpected events. This will mean that the firm is ready if a cancer diagnosis within their work force was to occur. Firms meet this challenge by: internal training – making sure they have internal workers that could take over different areas of the business, and having procedures in place when it comes to the external recruitment of positions – making sure that the firm has a continuity plan in the face of labour market constraints. This recommendation is most important when it comes to niche, high value-added roles, where firms rely on a worker to carry out day to day business activities. This is arguably linked to Romer's growth model where knowledge has increasing productivity returns, with knowledge being a function of the number of workers (Romer, 1986). A smooth continuity of business, driven by succession planning will reduce the short-run impact of labour force removal in the face of cancer diagnosis – through the preservation and retention of knowledge.

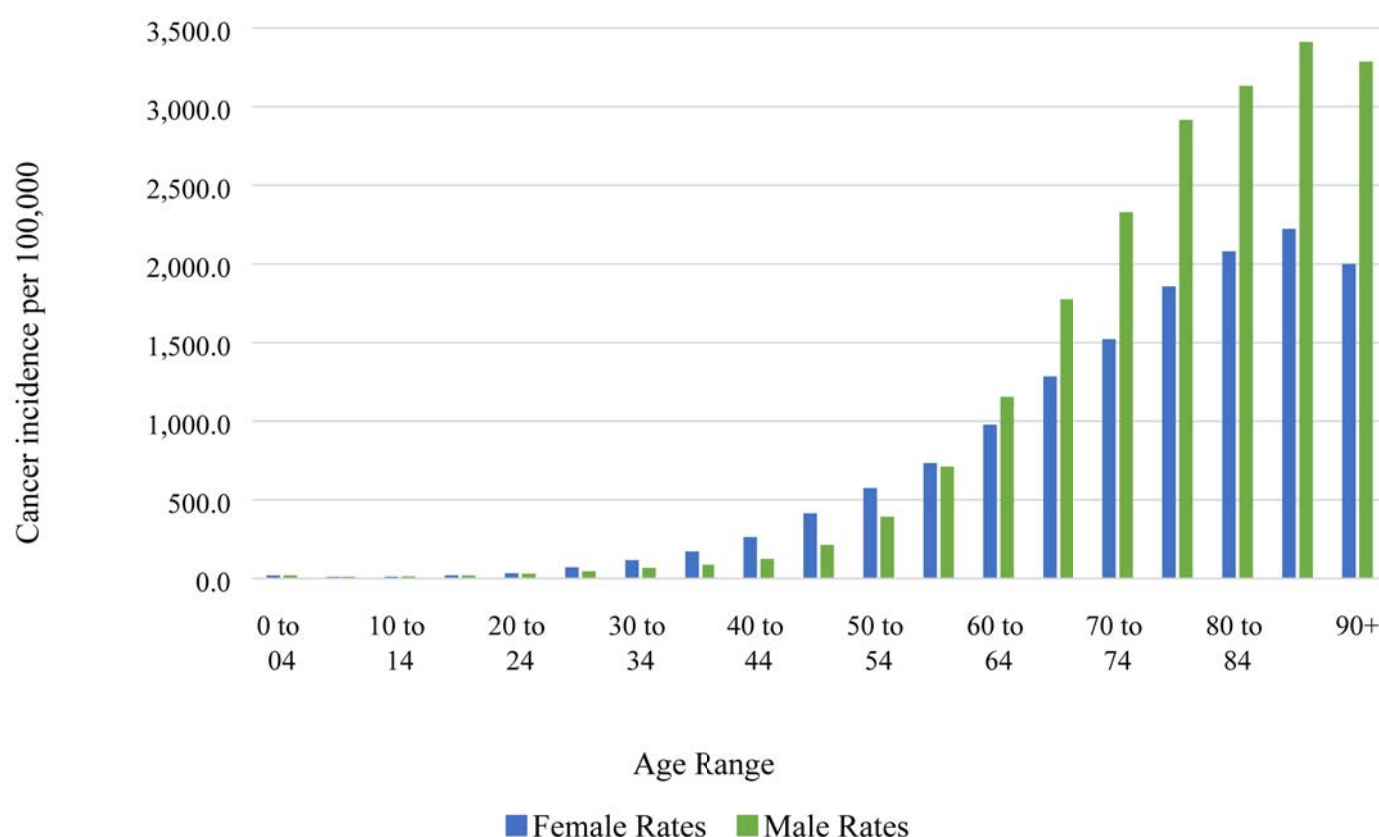
In addition to the previous recommendation, the confirmation of a short-run lagged effect may be the consequence of negative externality effect on the wider family unit, who may have to undertake informal care giving duties. This removal was reported by several studies (Veenstra *et al.*, 2017; de Moor *et al.*, 2017). These studies were examined within the literature review and showed a consensus on the existence of negative externality effects on the family unit of the cancer patient, with the progressive nature of the disease resulting in temporary adjustments in their work schedule or even labour force removal. This process of participation adjustment by family members over the course of the disease will lead to a contraction in labour supply. This may assist in the explanation of the negative short-run lagged result. This policy recommendation will be aimed at relieving the negative externality of informal cancer care. To combat this extra funding could be devoted to cancer care and support, aimed at assisting the family with at home care. With this extra funding more support nurses and carers could be deployed to the homes of patients, taking over from the informal family carers and reducing the effect of participation adjustment. This could be funded by a tax on the wealthiest in society or through an increase in capital gains tax. Capital gains represent a tax rate of only 10% to 28%, whilst income tax ranges from 20% to 45% (HM Revenue and Customs, 2022). This represents a taxation bias on fixed permanent income, over the accumulation of wealth from assets. This disproportionately benefits the wealthiest

in society. An increase in the capital gains tax would narrow the gap, whilst also funding the cancer care support package. However, this policy would be dependent on the labour supply of carers, with there currently being a shortage (Cangiano and Shutes, 2010). This could be solved through the implementation of an attractive wage rate, which would be set to attract new individuals into the care industry – expanding labour supply.

5.3. Limitations

This section of the report will assess the limitations of the study, looking at the methodology and the study framework. One of the largest limitations of this study lies in its aggregation of the labour force and cancer rates, through its macroeconomic approach. The cancer data used looks at the cancer incidence rates from the ages of 15 to 90+, within this each age group has a different risk of cancer diagnosis. Figure 5 demonstrates this increase in cancer risk by age (Cancer Research UK, 2021).

Figure 5: Cancer incidence by age 2016 to 2018



As the empirical method used treats all age groups, sex, and occupation in the labour force as equally at risk of cancer, this may not capture the actual observed effect of cancer incidence rates on labour productivity. Therefore, the results of this study can be seen as an upper bound approximation of cancer's effect on productivity. Despite this the study is still valuable, offering the first look into the effect of cancer rates on

productivity, through a macroeconomic approach. The results of this study offer an insight into the possible effect within the population and gives rise to the possibility of future research. This limitation could be relieved by investigating this research question on a micro-level, looking at a sample of cancer patients. However, this would involve a high intensity of data collection.

Moreover, another limitation lies in the cancer variable itself. By using all cancers rather than specific cancers this study assumes that all cancers have the same level of effect on labour productivity. This may not be true within the population; some cancer may be easier to treat with less invasive treatments and be more detectable. This is a result of the study's macroeconomic approach. Similarly, this could be relieved in future research by looking at different cancers specifically and on a sample basis.

6. Conclusion

This study has investigated the effect of cancer on labour productivity, taking a macroeconomic approach with a health augmented production function. Using the ARDL approach to cointegration, this study was able to draw conclusions on the dynamic effect of cancer rates in the short-run and long-run. Within the introduction three main objectives were mentioned, these being to: examine the effect of cancer rates on labour productivity, assess if a negative relationship exists, and recommend policies to reduce cancer rates and expand labour productivity.

To examine the possible effect of cancer rates on labour productivity, this study first looked at the current literature. In the short-run there seemed to be a consensus on the impact that cancer inflicts on labour market outcomes and negative externality effects. Existing studies pointed to the removal of individuals with a cancer diagnosis from the labour market (Polachek and Tatsiramos, 2019; Barnay *et al.*, 2019). Moreover, many studies reported a negative externality effect on the family of cancer patients, with family having to take up informal care duties in the face of the progressive disease (de Moor *et al.*, 2017). In the long-run, the literature could not reach a consensus, with some studies reporting a phased employment return (Mehnert, 2011), but others pointing to a significant retirement proportion (Bradley and Bednarek, 2002), with cancer possibly only accelerating existing retirement plans. With the literature examined, gaps within the research were found with the main one being the investigation into the aggregate effect of cancer rates on labour productivity, with most studies on the topic being sample-based investigations. This gap led to a non-existent consensus on cancer's effect in the long-run and even variations on its short-run effect. This study set out to fill this gap. An augmented Cobb-Douglas production function was formed to take health into consideration within the production decision. This formed the basis of the ARDL empirical approach being used to determine the effect of cancer rates on labour productivity. The results gathered presented an interesting interpretation, with cancer rates having a positive effect in the long-run and a negative short-run lagged effect. In the long-run, a 10% increase in cancer incidence led to a \$2242 increase in additional labour productivity. In the short-run, a 10% increase in one year lagged cancer incidence led to a -\$1711 loss in labour productivity per worker. These results along with the literature review filled this study's first objectives. The reasons into why this negative

short-run effect existed was also explored. The results mirrored that of much of the literature, with cancer patients making a phased return through their recovery. The long-run effect suggested that a convergence existed, with cancer not impacting the long-run productive capacity of the economy.

The study then offered recommendations to correct the short-run labour productivity contraction, allowing the economy to reach the long-run equilibrium sooner. Given that cancer is an unexpected event, the short-run impact on productivity could be symbolic of a lack of succession planning. If firms do not plan for the eventuality that their highest value-added employees unexpectedly leave, the firm may be left with a position that the labour market may not be able to quickly fill. Firstly, the supply of labour may not possess the expertise required to carry out the role. Secondly, the firm may not have developed internal training processes that allow existing employees to transfer between roles. In the face of cancer diagnosis these are reasons why the firm may struggle to maintain their productive capacity. The presence of negative externalities may also be a factor. As mentioned previously family members may be required to undertake informal care duties, requiring them to adjust their labour participation. To relieve this externality, this study looked at possible improvements in the financing of the care system, with professionals taking over from the family. These recommendations will move the UK economy closer to achieving the two UN SDGs mentioned within the introduction – these being: good health and wellbeing, plus decent work and economic growth (UNGA, 2015).

Finally, this study looked at the limitations of the methodology. Given the Macroeconomic approach, the data used meant that the results suffered from a cohort effect, with all ages, occupations, and genders having the same cancer risk. This meant that the results of this study can be seen as an upper bound approximation of cancer's effect. Moreover, by including all cancer incidence rates rather than specific cancers, we assume that the effect of cancer on labour productivity is universal regardless of cancer type. These limitations do present opportunities for future research. This methodology could be adapted to take a micro-approach, looking at specific cancer types, ages and genders. A micro-approach will relieve the model of a cohort effect, by removing aggregation. Additionally, the investigation of bi-directional causality could be investigated to assess if productivity has an effect on cancer rates. If bi-directional causality is found, this may present a new opportunity for the development of a health augmented endogenous growth model. Future research may also be conducted on other health conditions such as cardiovascular diseases, which may capture external pressures such as stress more accurately. However, despite its limitations, this study has successfully identified a new way of looking at the economic cost of progressive disease – offering a new application of economic methodology into the investigation of cancer's effect on labour productivity.

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Appendices

Appendix 1 – Descriptions of model variables

Variable	Source	Comments
Ly <i>Logarithm of GDP per worker</i> 1979 - 2010	World Bank GDP (Constant 2015 USD) OECD Labour Force	GDP divided by annual labour force
Lk <i>Logarithm of capital per worker</i> 1979 - 2010	World Bank Gross Capital Formation (Constant 2015 USD) OECD Labour Force	Capital formation divided by annual labour force
Ledu <i>Logarithm of education spend per worker</i> 1979 - 2010	World Bank Education spending (% GDP) GDP (Constant 2015 USD) OECD Labour Force	Education spending (calculated through % GDP) divided by annual labour force Statistics for 1979, 1993 and 1997 were missing – calculate by the average of previous and following year
Lhs <i>Logarithm of health spend per worker</i> 1979 – 2010	World Bank GDP (Constant 2015 USD) OECD Health spending (% GDP) Labour Force	Health spending (calculated through % GDP) divided by annual labour force
Lallc <i>Logarithm of all cancer incidence rates</i> 1979 – 2010	Cancer Research UK Observed European age-standardised incidence rates per 100,000 in UK Available on the cancer research UK incidence projection data sheet, which contains Observed rates and Observed age standardised rates, based on (Smittenaar <i>et al</i> , 2016)	Excluding Non-Melanoma Skin Cancer, Benign Brain Other CNS and Intracranial Tumours

Appendix 2 – ARDL model Bounds test

F-Bounds Test		Null Hypothesis: No levels relationship		
Test Statistic	Value	Signif.	I(0)	I(1)
Asymptotic: n=1000				
F-statistic	5.945898	10%	2.2	3.09
k	4	5%	2.56	3.49
		2.5%	2.88	3.87
		1%	3.29	4.37
Finite Sample: n=35				
Actual Sample Size	29	10%	2.46	3.46
		5%	2.947	4.088
		1%	4.093	5.532
Finite Sample: n=30				
		10%	2.525	3.56
		5%	3.058	4.223
		1%	4.28	5.84

Appendix 3 – ARDL Long-run results

Levels Equation				
Case 2: Restricted Constant and No Trend				
Variable	Coefficient	Std. Error	t-Statistic	Prob.
LK	0.383458	0.019986	19.18625	0.0000
LEDU	0.071960	0.018301	3.932144	0.0017
LH	0.118727	0.016510	7.191216	0.0000
LALLC	0.266844	0.057167	4.667769	0.0004
C	1.862948	0.110573	16.84816	0.0000
EC = LY - (0.3835*LK + 0.0720*LEDU + 0.1187*LH + 0.2668*LALLC + 1.8629)				

Appendix 4 – ARDL error correction model results

ARDL Error Correction Regression
 Dependent Variable: D(LY)
 Selected Model: ARDL(3, 3, 1, 2, 2)
 Case 2: Restricted Constant and No Trend
 Date: 05/07/22 Time: 14:20
 Sample: 1979 2010
 Included observations: 29

ECM Regression				
Case 2: Restricted Constant and No Trend				
Variable	Coefficient	Std. Error	t-Statistic	Prob.
D(LY(-1))	0.774398	0.148676	5.208613	0.0002
D(LY(-2))	0.397355	0.126743	3.135112	0.0079
D(LK)	0.267597	0.022654	11.81232	0.0000
D(LK(-1))	-0.291988	0.056261	-5.189889	0.0002
D(LK(-2))	-0.212951	0.045672	-4.662626	0.0004
D(LEDU)	0.022517	0.030742	0.732454	0.4769
D(LH)	0.201098	0.026674	7.539151	0.0000
D(LH(-1))	-0.079737	0.028011	-2.846630	0.0137
D(LALLC)	-0.040451	0.104677	-0.386433	0.7054
D(LALLC(-1))	-0.203598	0.093080	-2.187344	0.0476
CointEq(-1)*	-1.760611	0.250504	-7.028278	0.0000
R-squared	0.942404	Mean dependent var	0.007563	
Adjusted R-squared	0.910407	S.D. dependent var	0.008321	
S.E. of regression	0.002491	Akaike info criterion	-8.870842	
Sum squared resid	0.000112	Schwarz criterion	-8.352213	
Log likelihood	139.6272	Hannan-Quinn criter.	-8.708414	
Durbin-Watson stat	2.352324			

* p-value incompatible with t-Bounds distribution.

Appendix 5 – ARDL LM-test results

Breusch-Godfrey Serial Correlation LM Test:

Null hypothesis: No serial correlation at up to 3 lags

F-statistic	0.291753	Prob. F(3,10)	0.8305
Obs*R-squared	2.333965	Prob. Chi-Square(3)	0.5060

Appendix 6 – ARDL Heteroskedasticity test results

Heteroskedasticity Test: Breusch-Pagan-Godfrey

Null hypothesis: Homoskedasticity

F-statistic	0.775498	Prob. F(15,13)	0.6843
Obs*R-squared	13.69500	Prob. Chi-Square(15)	0.5488
Scaled explained SS	5.107768	Prob. Chi-Square(15)	0.9912

Appendix 7 – ARDL Ramsey reset test results

Ramsey RESET Test

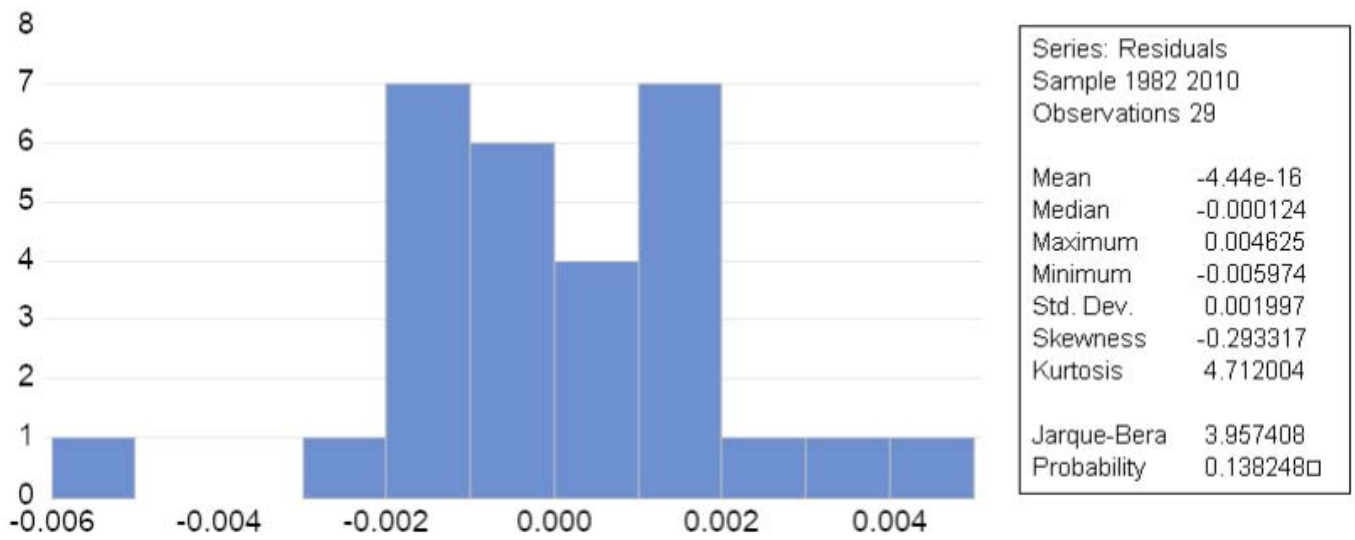
Equation: UNTITLED

Omitted Variables: Squares of fitted values

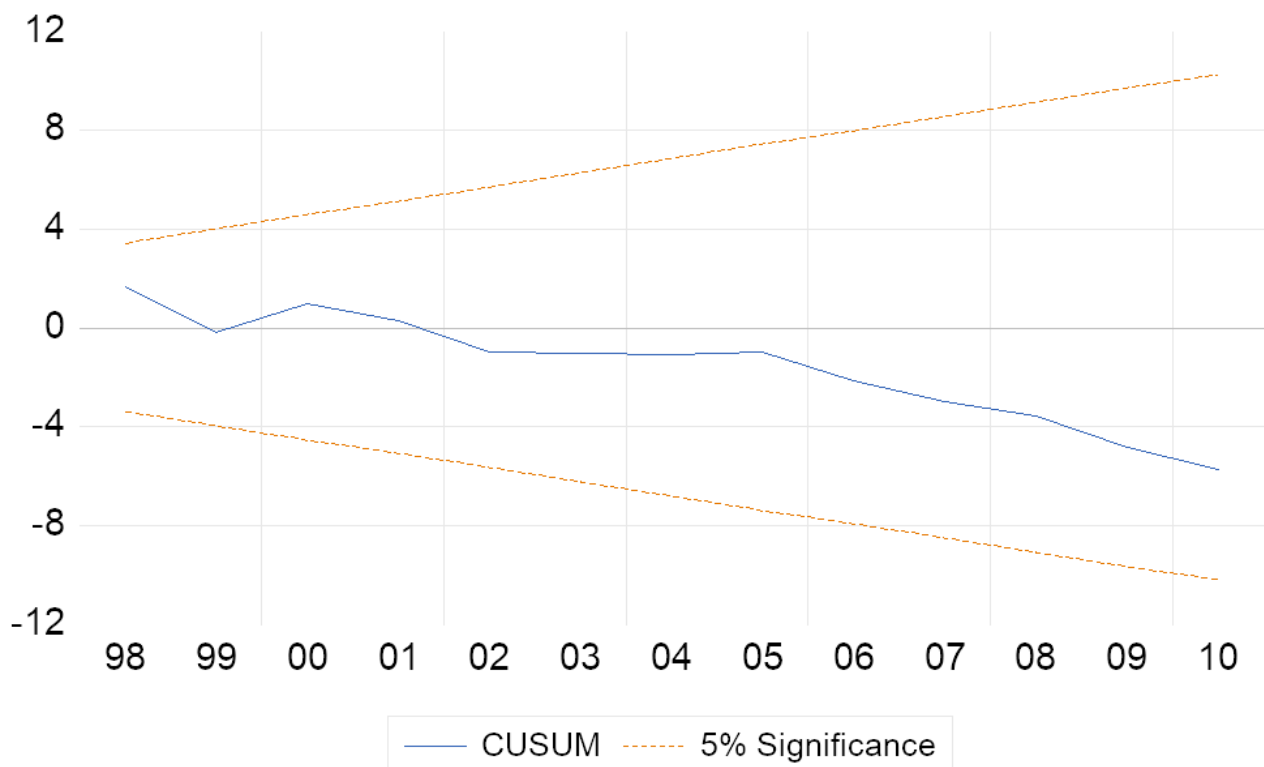
Specification: LY LY(-1) LY(-2) LY(-3) LK LK(-1) LK(-2) LK(-3) LEDU LEDU(-1) LH LH(-1) LH(-2) LALLC LALLC(-1) LALLC(-2) C

	Value	df	Probability
t-statistic	0.491598	12	0.6319
F-statistic	0.241669	(1, 12)	0.6319
Likelihood ratio	0.578229	1	0.4470

Appendix 8 – ARDL Normality test results



Appendix 9 – ARDL model CUSUM results



Appendix 10 – ARDL model CUSUMSQ results

