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# **R&D** expenditures by field of science and GDP: Which causes which in Canada?

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#### **Abstract**

This paper attempts to reveal the relationship between GDP per capita and R&D expenditure per capita, R&D expenditure per capita on natural sciences and engineering, and R&D expenditure per capita on social sciences and humanities for Canada. Based on data from 1981 to 2014, bootstrap causality test proposed by Hacker and Hatemi-J (2006) show that there is a unidirectional causality from GDP per capita to R&D expenditure per capita, and a unidirectional causality from GDP per capita to R&D expenditure per capita on natural sciences and engineering. However, no causal relationship is observed between R&D expenditure per capita on social sciences and humanities and GDP per capita. These results may point an indirect relationship between the variables or the validity of R&D paradox and the European paradox for Canada.

Keywords: R&D; GDP; economic growth; causality; Canada

JEL Classification Codes: C32, O30, O32, O40

# 1. Introduction

Research and development (R&D) is accepted as one of the key drivers of economic growth today. Economic growth is an indicator of a country's welfare, and it indicates (generally yearly) percentage change in a country's real gross domestic product (GDP). Many countries focus on R&D policies since one of the crucial goals of a country is economic growth. In the simplest form, R&D activities enable knowledge production, innovation, productivity, and technological progress which will bring economic growth. Therefore, there exists a substantial linkage between R&D activities and economic growth.

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Economic growth models proposed by Solow (1956) and Swan (1956) are known as Neoclassical growth theories. They show that how technological progress provides economic growth in an economy. The part of output growth which cannot be explained by labor and capital has entered the literature as 'Solow residual', and attributed to technology. However, these Neo-Classical growth theoreticians assume technology as an exogenous variable. In other words, technology is believed to be constant. After a while, unlike Solow and Swan, the economists who form endogenous growth models explain the technological progress in detail. The word 'endogenous' comes from the fact that technology is included in models as an endogenous variable. These models base technological progress upon some economic variables, and show that how growth is provided. A well-known endogenous growth theory is proposed by Arrow (1962). He defines learning as a product of experience, and suggests an endogenous growth theory that explains shifts in production function through changes in knowledge. Accordingly, knowledge production increases thanks to learning-by-doing, and the economy grows. Lucas (1988) emphasizes the prominence of human capital on growth through schooling as well as learning-by-doing. Lastly, Romer (1990) highlights R&D activities. In his model, R&D is considered as a separate sector, and advances in this sector (i.e. new products developed) provide economic growth.

R&D intensity (R&D expenditures/GDP) is an important indicator in terms of a country's economic performance. First, we look at country groups. According to data from OECD's (2018) statistics website, the intensity is 2.35% for OECD countries, and 1.94% for European Union countries in 2016. In 2015, the intensity for the whole world was recorded as 2.2% (World Bank, 2018). When we look at the top countries in the R&D intensity, we see that the intensity was 4.25% in Israel, 4.24% in Korea, and 3.25% in Sweden. For Canada, this indicator is 1.6% in 2016, and under the country group averages mentioned above (OECD, 2018).

Using various elasticity estimation techniques, most of the studies in the literature show that innovative activities have positive effects on output or productivity. For example, Hanel (2000) for Canada; Wakelin (2001) for the United Kingdom; Sylwester (2001) for G7 countries; Wang and Tsai (2004) for Taiwan; and Ülkü (2004) for OECD countries find positive relationship between innovation activities and output or productivity.

In the literature on causality, four hypotheses are classified by Maradana et al. (2017) by the way of causality between innovation activities and GDP. These are demand-following hypothesis, supply-leading hypothesis, feedback hypothesis, and neutrality hypothesis. These hypotheses can be explained simultaneously with the literature which is summarized in Table 1. Demand-following hypothesis (DFH) is supported when GDP causes innovation activities (see Maradana et al., 2017; Bozkurt, 2015; Ntuli et al., 2015; Santos and Catalão-Lopes, 2014; Cetin, 2013; and Güloğlu and Tekin, 2012). Supply-leading hypothesis (SLH) is supported when innovation activities cause GDP (see Maradana et al., 2017; Ntuli et al., 2015; Cetin, 2013; Santos and Catalão-Lopes, 2014; Peng, 2010; and Yang, 2006). Feedback hypothesis (FH) reflects twoway causality between innovation activities and GDP (see Maradana et al., 2017; Cetin, 2013; and Wu and Zhou, 2006). Finally, neutrality hypothesis (NH) indicates the absence of causality between innovation activities and GDP (see Maradana et al., 2017; Ntuli et al., 2015; Tuna, Kayacan and Bektas, 2015; Sadraoui, Ali and Deguachi, 2014; Santos and Catalão-Lopes, 2014; and Cetin, 2013). As seen on Table 1, the only available causality work for Canada is Ntuli et al. (2015). Among other results, they find GDP causes research output in Canada, which supports DFH.

The intent of this study is to examine the causality between R&D expenditure per capita, R&D expenditure per capita by field of science, and GDP per capita for Canada. To my knowledge, there is no study that uses R&D expenditures by field of science for any country. The results indicate the presence of two causal relationships: First, GDP per capita causes R&D expenditure per capita. Second, GDP per capita causes R&D expenditure per capita on natural

sciences and engineering. But, no causality is observed between R&D expenditure per capita on social sciences and humanities and GDP per capita.

In the rest of the paper, second part explains data, models and methodology, third part gives empirical results, and the last part concludes.

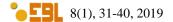
Table 1. Selected studies on the causality between innovation activities and GDP.

Author	Time	Country	Supported Hypothesis
Maradana et al. (2017)	1989- 2014	19 European countries	SLH for Belgium, Denmark, Finland, France, Germany, Italy, Norway, Poland, Portugal, Sweden, and United Kingdom; DFH for Austria, Czech Republic, Ireland, Netherlands, Romania, and Spain; FH for Panel; NH for Greece
Bozkurt (2015)	1998- 2013	Turkey	DFH
Ntuli et al. (2015)	1981- 2011	OECD countries	SLH for Finland, Hungary, Mexico, and United States; DFH for Austria, Canada, France, Israel, Italy, New Zealand, Poland, and United Kingdom; NH for remaining countries
Tuna, Kayacan and	1990-	Turkey	NH
Bektaş (2015)	2013	22: 1 .: 1 1	N///
Sadraoui, Ali and Deguachi (2014)	1970- 2012	32 industrial and developing countries	NH
Santos and Catalão-Lopes (2014)	1987- 2008	8 European countries	SLH for France and Spain; DFH for Netherlands; NH for Portugal, Belgium, Germany, Ireland, and United Kingdom
Çetin (2013)	1981- 2008	9 European countries	SLH for Austria; DFH for Denmark, Spain and Portugal; FH for Finland and France; NH for Holland, Ireland and Italy
Güloğlu and Tekin (2012)	1991- 2007	High-income OECD countries	DFH
Peng (2010)	1987- 2007	China	SLH
Wu and Zhou (2007)	1953- 2004	China	FH
Yang (2006)	1951- 2001	Taiwan	SLH

# 2. Data, models and methodology

#### 2.1. Data

Empirical analyses employ yearly data for Canada covering 1981-2014. The data obtained from OECD's (2018) statistics website include GDP per capita (constant 2010 prices and PPPs, US\$) and gross domestic expenditure on R&D by sector of performance and by field of science (constant 2010 prices and PPPs, US\$). Sector of performance is set to total intramural to get total numbers. R&D expenditures data include three series, namely gross domestic R&D expenditure



on natural sciences and engineering, gross domestic R&D expenditure on social sciences and humanities, and gross domestic R&D expenditure on all fields of science. These series are multiplied by 1 million to get rid of 'millions' notation and also divided by total population series acquired from World Bank's (2018) World Development Indicators to get per capita values.

Figure 1 displays the timeline of the R&D series used in this study. As seen, R&D expenditure per capita on social sciences and humanities are substantially lower than R&D expenditure per capita on natural sciences and engineering. Until 2001, R&D expenditure per capita on natural sciences and engineering has increased considerably. However, R&D expenditure per capita on social sciences and humanities follows almost a straight path.

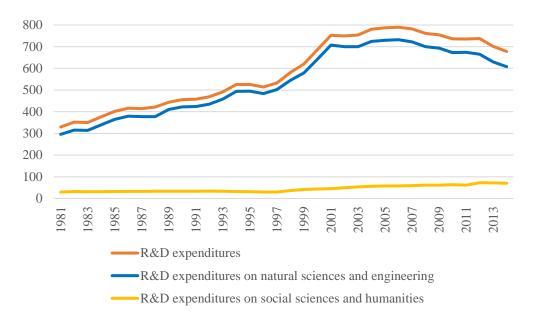


Figure 1. R&D expenditures in Canada (per capita, constant 2010 prices, PPPs, US\$).

# 2.2. Models and methodology

Following the literature, GDP per capita is simply described as functions of R&D expenditures per capita. All variables are used in their natural logarithms, and abbreviated as ln *GDP* for GDP per capita; ln *RD* for R&D expenditure per capita; ln *RDN* for R&D expenditure per capita on natural sciences and engineering; and ln *RDS* for R&D expenditure per capita on social sciences and humanities. Models used in causality analyses can be indicated in vector autoregression (VAR) form with lag augmentations as follows:

$$Model(A) - A_t = \beta_0 + \beta_1 A_{t-1} + \dots + \beta_{p_a} A_{t-p_a} + \dots + \beta_{p_a+d_a} A_{t-p_a-d_a} + \varepsilon_t$$
 (1)

$$Model\left(B\right) - B_{t} = \theta_{0} + \theta_{1}B_{t-1} + \dots + \theta_{p_{b}}B_{t-p_{b}} + \dots + \theta_{p_{b}+d_{b}}B_{t-p_{b}-d_{b}} + \epsilon_{t} \tag{2}$$

$$Model(C) - C_t = \gamma_0 + \gamma_1 C_{t-1} + \dots + \gamma_{p_c} A_{t-p_c} + \dots + \gamma_{p_c+d_c} \gamma_{t-p_c-d_c} + e_t$$
 (3)

Here,  $A_t$ ,  $B_t$ , and  $C_t$  are  $2 \times 1$  ln GDP and ln RD; ln GDP and ln RDN; and ln GDP and ln RDS vectors, respectively. Also,  $\beta_0$ ,  $\theta_0$  and  $\gamma_0$  are  $2 \times 1$  constant term vectors, and  $\beta_r$ ,  $\theta_r$ , and  $\gamma_r$  are  $2 \times 2$  coefficient matrices for lag r = 1, 2, ..., p. Finally,  $\varepsilon_t$ ,  $\varepsilon_t$ , and  $e_t$  are  $2 \times 1$  error vectors.

Note that p is optimum lag length for associated VAR model, and to be determined using Hatemi-J Criterion (HJC) (Hatemi-J 2003, 2008; Hacker and Hatemi-J, 2008), which combines Schwarz (1978) and Hannan and Quinn (1979) criteria, and gives one optimum lag length. d is maximum integration order of the series, to be detected by unit root tests Augmented Dickey-Fuller (1981) (ADF) and Phillips-Perron (1988) (PP), in the related VAR model. Sub-indices of

p and d indicate the models in which p and d belong to.

The test proposed by Granger (1969) is commonly used in investigating causation between the variables interested. However, the test results may lead to void implication, if the series are not stationary (Granger and Newbold, 1974). Also, causality between integrated variables in their levels cannot be tested using VAR models, since asymptotic distribution theory is not valid, as Sims, Stock and Watson (1990) point. Toda and Yamamoto (1995) indicate the estimation of VAR models that are formulated in their levels, and testing general constraints in parameter matrices, tough the processes are integrated or cointegrated at random levels. The null hypothesis of "k'th element of  $A_t$  does not Granger-cause j'th element of  $A_t$ " can be checked through modified Wald statistic (MWALD), as Toda and Yamamoto (1995) indicate. Employing Monte Carlo simulations, however, Hacker and Hatemi-J (2006) clarify that if the sample is small, and error terms are autoregressive conditional heteroskedastic (ARCH) and non-normal, then MWALD may give invalid results. To solve this problem, they make use of bootstrap correction technique, and get credible critical values. For bootstrapping, Model (A) is estimated with null of no causality first. Thereafter, bootstrapped data  $A_t^*$  are generated based on estimated coefficients  $(\hat{\beta}_0, \hat{\beta}_1, ..., \hat{\beta}_{p_a})$ , the essential data  $(A_{t-1}, ..., A_{t-p_a})$ , and bootstrapped residuals  $(\hat{\varepsilon}_t^*)$ . Bootstrapped residuals are contingent upon T arbitrary draws with replacement from the regression's modified residuals. Every single draw has probability equals to 1/T, where T is sample size. To make the expected value of the bootstrapped residuals exactly zero, the mean of the resulting set of drawn modified residuals is subtracted from every modified residual in that set. Using leverages, modified residuals, which are the unadjusted residuals of the regression that are set to have constant variance, are obtained. Bootstrap simulation is performed 1000 times and MWALD statistic is estimated in every stage. Then the  $\alpha$ 'th upper quantile of the distribution of bootstrapped MWALD statistic is found and the  $\alpha$ -level bootstrap critical values  $(c_{\alpha}^*)$  are obtained. If MWALD is greater than  $c_{\alpha}^*$ , then the null is rejected. The same procedure is also valid for Model (B) and Model (C).

Unit root tests and diagnostic checks on stability, serial correlation, and normality were run on Eviews 10. Leveraged bootstrap simulations, HJC, and autoregressive conditional heteroskedasticity LaGrange multiplier (ARCH LM) test by Hacker and Hatemi-J (2005) were applied on GAUSS Light 9 by running modules of Hacker and Hatemi-J (2009a), (2009b), and (2009c), respectively.

# 3. Empirical results

Outcomes of ADF and PP unit root tests presented in Table 2 support that the variables are stationary in their first differences. Therefore, augmentation lags (*d*) are set 1 for all models. When we set the maximum lag order to 3, HJC suggests optimal lag orders 2 for Model (A) and Model (B), and 1 for Model (C). VAR estimations of all models pass stability, serial correlation, and ARCH LM tests. However, when Model (A) and Model (B) pass normality test, Model (C) fails (see Tables A1, A2, A3, and A4 in Appendices).

Table 2. Results of unit root tests.

-	ADF (Constant)		ADF (Trend)		PP (Constant)		PP (Trend)	
	Level	1 <sup>st</sup> Dif.	Level	1 <sup>st</sup> Dif.	Level	1 <sup>st</sup> Dif.	Level	1 <sup>st</sup> Dif.
ln GDP	-1.5596	-4.5433***	-2.6576	-4.5598***	-0.5090	-4.5433***	-2.0731	-4.5777***
ln RD	-1.6440	-3.0894**	-0.1269	-3.5997**	-2.1647	-3.0595**	0.3701	-3.5346*
ln <i>RDN</i>	-1.7942	-3.2036**	0.1254	-4.1002**	-2.3593	-3.1544**	0.6300	-3.7832**
ln RDS	0.2293	-4.2285***	-2.1190	-4.3265***	0.0446	-4.2285***	-1.5683	-4.3497***

Note: \*, \*\* and \*\*\* indicate 10%, 5% and 1% significance levels, respectively. t-stats for ADF and adjusted t-stats for PP. Lag length is chosen by Schwarz information criterion for ADF. Barlett kernel is employed as spectral estimation method and the bandwidth is determined using the Newey–West method for PP.

Now three reasons can be put forward on the selection of bootstrap causality procedure proposed by Hacker and Hatemi-J (2006). First, having a small sample with 34 observations. Second, having non-stationary series. Third, non-normal distributed residuals of Model (C).

*Table 3.* Results of bootstrap causality tests.

Null hypothesis	MWALD	1% bootstrap critical value	5% bootstrap critical value	10% boot- strap critical value
ln RD does not Granger-cause ln GDP	0.453	14.391	8.369	5.812
ln GDP does not Granger-cause ln RD	$9.050^{**}$	12.979	7.695	5.926
ln RDN does not Granger-cause ln GDP	0.055	12.491	8.084	5.962
ln GDP does not Granger-cause ln RDN	$8.269^{**}$	13.707	7.853	5.695
ln RDS does not Granger-cause ln GDP	1.827	6.559	3.553	2.428
ln GDP does not Granger-cause ln RDS	1.725	8.621	5.537	3.702

Note: \*\* represents the rejection of the null at 5% significance level.

According to the results given in Table 3, null hypotheses of 'ln *GDP* does not Granger-cause ln *RD*' and 'ln *GDP* does not Granger-cause ln *RDN*' are both rejected at 5% significance level. Consequently, there is a unidirectional causality from GDP per capita to R&D expenditure per capita, and a unidirectional causality from GDP per capita to R&D expenditure per capita on natural sciences and engineering in Canada.

#### 4. Conclusions

In this study, the relationship between per capita GDP and R&D expenditures per capita by field of science are examined for Canada over the period from 1981 to 2014. Having a small sample, non-stationary series and non-normal distributed error terms are the reason why bootstrap causality test proposed by Hacker and Hatemi-J (2006) is chosen. The first finding points to a unidirectional causality from GDP per capita to R&D expenditure per capita. This result is compatible with the results of Maradana et al. (2017); Bozkurt (2015); Çetin (2013); and Güloğlu and Tekin (2012). It is particularly in harmony with Ntuli et al. (2015) whose results show causality from GDP to research output for Canada. Also, a unidirectional causality from GDP per capita to R&D expenditure per capita on natural sciences and engineering is found. However, no causal relationship is observed between R&D expenditure per capita on social sciences and humanities and GDP per capita.

As seen, no evidence that supports 'supply-leading hypothesis' is found for Canada. In other words, R&D expenditures does not cause economic growth. In point of R&D expenditure per capita and R&D expenditure per capita on natural sciences and engineering, the results clearly show R&D expenditures originate from economic growth. These findings indicate the validity of 'demand-following hypothesis' in Canada. In this regard, the country can stimulate innovation activities as the economy grows.

In terms of social sciences, 'neutrality hypothesis' is supported for Canada. Thus, R&D expenditures on social sciences and humanities and GDP have no effect on each other. As shown in data subsection, Canada spends far less money on R&D in social sciences and humanities than in natural sciences and engineering. This can limit both quantity and quality of social studies, and be the reason why the causal link is broken. This field deals with humans, who are also economic agents that constitute the economy, by its very nature. Thus, R&D expenditures on social sciences can be related to economic growth through social channels. That is to say, there can be an indirect relationship.

All of these findings resembles Swedish version of R&D paradox and the European paradox.

Swedish version of R&D paradox indicates high R&D expenditure but comparatively low GDP (Ejermo, Kander, and Henning, 2011). Likewise, European paradox indicates high performance in science and low performance in high-tech sectors (European Commission, 1995).

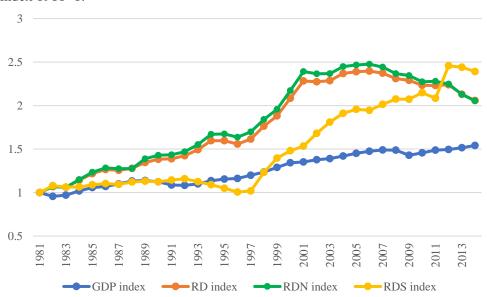


Figure 2. GDP per capita and R&D expenditures per capita by field of science, index 1981=1.

Ejermo, Kander, and Henning (2011) investigate the timeline of value added and R&D expenditures by sector. They explain the growing gap between value added and R&D expenditures in fast-growing manufacturing and service sectors in Sweden as R&D paradox. Following Ejermo, Kander, and Henning (2011), same procedure is applied using variables employed in empirical analysis. The variables are indexed (1981=1) to get a clean comparison. The timeline is given on Figure 2. Index forms of GDP per capita, R&D expenditure per capita, R&D expenditure per capita on natural sciences and engineering, and R&D expenditure per capita on social sciences and humanities abbreviated as GDP index, RD index, RDN index, and RDS index, respectively. It is seen that the gap between GDP index and RDN index, typically widens in time. There is almost no gap between GDP index and RDS index in 1981-1993 when the gap is negative in 1994-1998. After 1998, the gap becomes positive and also widens in time. These findings can be due to R&D and European paradoxes.

Finally, further study in this area is required. Then, future research can examine the existence of the possible indirect causality between R&D expenditures and economic growth. Also, validity of R&D and European paradoxes for Canada can be investigated in detail.

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# Appendix A – Additional tables

*Table A1.* Roots of characteristic polynomial.

Model (A)		Model (B)		Model (C)	
Root	Modulus	Root	Modulus	Root	Modulus
0.979034- 0.089139i	0.983083	0.985065-0.083223i	0.988574	0.982276	0.982276
0.979034+0.089139i	0.983083	0.985065+0.083223i	0.988574	0.837936	0.837936
0.241528-0.264432i	0.358135	0.219617-0.271096i	0.348891		
0.241528+0.264432i	0.358135	0.219617+0.271096i	0.348891		

Table A2. VAR residual serial correlation LM tests.

Model (A)		Model (B)		Model (C)		
Lags	LRE-Stat*	Prob	LRE-Stat*	Prob	LRE-Stat*	Prob
1	7.4512	0.1139	6.3732	0.1730	5.7718	0.2169
2	3.2891	0.5107	4.0887	0.3941	2.7459	0.6012
3	3.7636	0.4389	3.4711	0.4823	1.4408	0.8371

<sup>\*</sup>Edgeworth expansion corrected likelihood ratio statistic.

*Table A3.* Probabilities for ARCH effects.

Model (A)	Model (B)	Model (C)	
0.5440	0.3680	0.4920	

Table A4. VAR residual normality tests.

Model (A)		Model (	<b>B</b> )	Model (C	Model (C)	
Jarque-Bera	Prob	Jarque-Bera	Prob	Jarque-Bera	Prob	
4.7132	0.3180	4.9554	0.2919	13.8304	0.0079	