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Is nutritional improvement a cause or a consequence of economic growth? Evidence from Mauritius

Harris Neeliah
Department of Agricultural and Food Economics, University of Reading

Bhavani Shankar
Department of Agricultural and Food Economics, University of Reading

Abstract

Sustained economic growth in Mauritius has resulted in changes in nutrition patterns. The purpose of this paper is to investigate the existence and direction of causality between calories intake and economic growth. Our results as opposed to findings from the literature, supports the neutrality hypothesis, implying an absence of causality running in either directions. Therefore nutrition policies that are based on reducing calories intake can be envisaged, without negatively impacting on economic growth.

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1. Introduction

Alfred Marshall (1920) noted that the most valuable of all capital is that invested in human beings. Since then much effort has been applied to characterising the nature of human capital. It is a fact that investment in education is an important determinant, but as highlighted by Cole (1971) investment in proper nutrition is another essential factor. Nutrition is the fundamental prerequisite for human welfare and contributes to human and social capital. One view is that better nutrition is a pre-determinant of physical health and this leads to increased productivity and subsequently to economic growth (Correa and Cummins, 1970; Strauss and Thomas, 1998). This model implies that there is a causal relationship running from nutrition to national income or GDP. Several studies have tried to formalise this relationship. Arcand (2001) considered the impacts of two measures of nutritional status, namely prevalence of food inadequacy (PFI) and dietary energy supply (DES), on the growth rate of real GDP per capita for 129 countries from 1960s to 1980s. He reported statistically significant and quantitatively important effect of nutrition on growth and claimed that inadequate nutrition was causing 0.23 to 4.7% loss in the annual growth rate of GDP per capita worldwide, and 0.16 to 4.0 % loss for Sub-Saharan Africa.

On the other hand there is the conventional wisdom that a high level of economic growth can impact positively on the nutrition status of the population. This in turn implies that there can also be a causal relationship in the opposite direction that is going from GDP to the level of nutrition. Over the last decades there have been numerous studies that have depicted the relationship between nutritional status and economic growth. World Bank (1986) pointed out that income growth can alleviate and eventually eliminate inadequate calorie intake, with calorie-income elasticities ranging between 0 and 1. Strauss (1984) estimated an elasticity of 0.82 for Sierra Leone and Dawson and Tiffin (1998) computed an elasticity of 0.34 for India. The latter also conducted Granger causality tests and showed that there was a unidirectional relationship from income to calorie intake. Easterly (1999) reported that an increase in GDP per capita of 1% was associated with an increase in daily calorie intake of 538 kcal/day. Dawson (2002) examined the relationship for Pakistan for the years 1961 to 1998 and found a unidirectional relationship running from real per capita income to daily per capita intake. He also computed an income elasticity of calorie demand of 0.19, thus validating Engel's law.

Thirdly there are cases where there is feedback between calorie intake and GDP. The cause and effect between nutrition and economic growth runs both ways. Just as income growth enhances nutrition security, healthy active, well-nourished citizens are an important precondition for sustained income growth (Benson, 2004). Tiffin and Dawson (2002) estimated a long-run income elasticity of calorie demand of 0.31 for Zimbabwe and also found out that bi-directional causality exists between calorie intake and income.

A fourth possibility is that an increase in income does not result in improvements in calorie intake and an increase in calorie intake does not impact on economic growth. This is referred to as the neutrality hypothesis. The literature is scarce with studies supporting this hypothesis as far as calorie intake and economic growth are concerned.

The literature has shown that there are four lines of inquiry. Firstly there is uni-directional causality running from calorie intake to economic growth. Under such a circumstance, it implies that an economy is dependent on nutrition. Inadequate calorie intake may negatively affect economic growth. If the uni-directional causality is in the opposite direction, it may imply that the economy is not nutrition-dependent and decreasing calorie intake will not have a bearing on economic growth. A bi-directional causality could mean that both calorie intake and economic growth affect each other in a feedback fashion. Lastly, no causality in either direction implies that implementing policies that affect calorie intake would not affect economic growth and *vice versa*. It is important therefore, to ascertain empirically whether there is a causal link between calorie intake and economic growth.

Uncovering this causality for Mauritius is highly conjectural. Mauritius has witnessed rapid economic growth since the early 1980s (World Bank, 2007) to achieve a GDP/capita of US$ 5059 in 2005 (UNDP, 2007). This growth has concomitantly lead to an increase in disposable income for the average Mauritian, resulting in fundamental changes in food consumption patterns, characterised by an increase in total calorie intake and accompanied with a shift in the composition of the diet towards more meat, eggs, dairy products as well as more fats and oils. This nutrition transition has brought about a rapid increase in the prevalence of overweight, obesity and associated non-communicable diseases (NCDs). These have reached epidemic proportions in the past few decades, with respective prevalence of 13.3% for diabetes mellitus, 12.1% for hypertension, 30% for hypercholesterolaemia and 40% for overweight and obesity (MoH[1], 1995). The parent ministry responsible for health conducted a NCDs survey in 2004 and reported 15% prevalence for Type 2 diabetes, 30% prevalence for hypertension, 35.7% for overweight and obesity and that a number of risk factors remain highly prevalent (MoHQL, 2006). These findings show that the NCDS remain a major public health problem.

Therefore there is need to develop, implement and strengthen policies to curb NDCs. The presence or absence of a relationship between calorie intake and GDP is an important consideration in developing and formulating such policies. This paper attempts to provide this information by using a consistent approach to depict the absence or existence of causality and its direction to better inform nutrition policy formulation. The remainder of the paper is organised as follows: section 2 presents the methodology, section 3 explains the data and gives the results and section 4 summarises and concludes.

2. Methodology

2.1. Granger-causality and stationarity

The Granger-causality test is a convenient and very general approach for detecting any presence of a causal relationship between two variables. A time series (X) is said to Granger-cause another time series (Y) if the prediction error of current y declines by using past values of X in addition to past values of Y. This test is used in this study.

The series to be used in the Granger causality test needs to be stationary. A stationary series has basic statistical properties which are invariant with respect to time. Here we are adopting the weak stationarity concept. Thus the data are assumed to be stationary if the means, variances and covariances of the series are independent of time. The reasons behind data being non-stationary can be varied, the primal one is due to the presence of unit roots. Practically most economic data are not stationary, but are rather integrated or non-stationary.

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[1] MoH stands for Ministry of Health and MoHQL stands for Ministry of Health and Quality of Life

According to Stock and Watson (1989), using non-stationary data in causality tests can give spurious results as correlation could persist even in large time series. Therefore the unit roots of the series are tested to check the stationarity of each variable. The Philips-Perron (Philips and Perron, 1988) test is used, as it is known to be robust to a variety of serial correlations and time-dependent heteroskedasticities (Yoo, 2005). If any of the series is found to be non-stationary and it becomes stationary after differencing once, it is said to be integrated of order one, I(1). Therefore an I(1) can be first-differenced before the Granger causality test can be applied to it.

2.2. Cointegration

When series are integrated, conventional test statistics may be a poor guide as to whether such relationships between them exist. If two integrated variables are not integrated there can be no long-run relationship between them and subsequent regressions will be spurious. Tests for cointegration constitute tests of whether such relationships exist. Such tests have been suggested as means to test whether the equilibrium propositions of economic theory. If the selected variables are not cointegrated, the resulting model cannot represent such long-run relationship.

Cointegration between two variables requires the satisfaction of two conditions. Firstly the two series must have a similar basic statistical property that is they must be integrated of the same order. Secondly there should exist some linear combination of the series such that $Z_t = Y_t - a - bX_t$ is stationary even though the individual series for y and x are not. This linear combination is simply the residual from a static ordinary least squares regression of y on x. Such a regression is known as the cointegrating regression. If two series are I(1) then their linear combination will typically be I(1). It is only when there is cointegration that there would be a linear combination which is I(0). In other words this linear combination cancels out the stochastic trends in the I(1) series, resulting in the regression to be non-spurious.

Cointegration says nothing about direction of causation, but only that a long-run relationship between the variables exists or not. It implies Granger causality in at least one direction.

2.3 Granger causality test

If two series are cointegrated then the error-correction model must be used instead of the standard Granger-causality to investigate both short and long-run causality. Error correction models are a particular form of dynamic econometric model. According to this specification, changes in the dependent variable in response to changes in the explanatory variables aim at restoring the long-run relationship between them. The long-run relationship reflects cointegration between the variables under study. This relationship is represented in (1) and (2).

$$\Delta \mathrm{Y}_{\mathrm{t}} = \beta_{10} + \sum_{\mathrm{i}=1}^{\mathrm{n}} \beta_{11\mathrm{i}} \Delta \mathrm{Y}_{\mathrm{t-i}} + \sum_{\mathrm{j}=1}^{\mathrm{m}} \beta_{12\mathrm{j}} \Delta \mathrm{X}_{\mathrm{t-j}} + \beta_{13} \varepsilon_{\mathrm{t-1}} + \mu_{\mathrm{1t,}} \tag{1}$$

$$\Delta \mathrm{X}_{\mathrm{t}} = \beta_{20} + \sum_{\mathrm{i}=1}^{\mathrm{p}} \beta_{21\mathrm{i}} \Delta \mathrm{Y}_{\mathrm{t-i}} + \sum_{\mathrm{j}=1}^{\mathrm{q}} \beta_{22\mathrm{j}} \Delta \mathrm{X}_{\mathrm{t-j}} + \beta_{23} \varepsilon_{\mathrm{t-1}} + \mu_{\mathrm{2t,}} \tag{2}$$

where Δ is the first difference operator, X_t and Y_t represent the natural logarithms of calorie intake and real GDP respectively. Δ is the difference operator, *n*, *m*, *p*, *q* are the number of lags, βs are parameters to be estimated, μ_ts are the serially uncorrelated error terms and ε_{t-1} is the error correction term (ECT) which is derived from the long-run cointegration relationship, $Y_t = \eta_0 + \eta_1 X_t + \varepsilon_t$ where ηs are parameters to be estimated and ε_t is the error term.

Sources of causation can be identified by testing for significance of the coefficient on the lagged variables in Eqs. (1) and (2). According to Masih and Masih (1996) weak Granger causality can be interpreted as 'short-run' causality in the sense that the dependent variable only responds to short-term shocks to the stochastic environment. We therefore test $H_0 : \beta_{12j} = 0$ against $\mathrm{H_1} : \beta_{12\mathrm{j}} \neq 0$ for Eq. (1) and $\mathrm{H_0} : \beta_{21\mathrm{i}} = 0$ against $\mathrm{H_1} : \beta_{21\mathrm{i}} \neq 0$ for Eq.(2).

Another source of causation is the ε_{t-1} term in Eqs. (1) and (2). Through the ε_{t-1}, an EC model offers an alternative test of causality. The coefficient on the ε_{t-1} represents how fast deviations from the long-run equilibrium are eliminated following changes in each variable. If for example β_{13} in Eq. (1) is zero, then LGDP does not respond to deviation from the long-run equilibrium in the previous period, that is there is Granger non-causality in the long-run.

We also check whether the two sources of causation are jointly significant, in order to check for Granger causality. This is done by testing the joint hypotheses $H_0 : \beta_{12j} = 0$ and $\beta_{13} = 0$ for all *j* in Eq. (1) or $\mathrm{H_0} : \beta_{21\mathrm{i}} = 0$ and $\beta_{23} = 0$ for all *i* in Eq. (2). These tests are referred to as strong causality tests. If there is no causality in either direction, the 'neutrality hypothesis' holds.

In the absence of cointegration between GDP and calorie intake, equations (1) and (2) respectively become (3) and (4). A standard Granger causality test is then run, namely $H_0 : \beta_{12j} = 0$ against $\mathrm{H_1} : \beta_{12\mathrm{j}} \neq 0$ for Eq. (3) and $\mathrm{H_0} : \beta_{21\mathrm{i}} = 0$ against $\mathrm{H_1} : \beta_{21\mathrm{i}} \neq 0$ for Eq.(4).

$$\Delta \mathrm{Y_t} = \beta_{10} + \sum_{\mathrm{i=1}}^{\mathrm{n}} \beta_{11\mathrm{i}} \Delta \mathrm{Y_{t-i}} + \sum_{\mathrm{j=1}}^{\mathrm{m}} \beta_{12\mathrm{j}} \Delta \mathrm{X_{t-j}} + \mu_{1\mathrm{t},} \tag{3}$$

$$\Delta \mathrm{X_t} = \beta_{20} + \sum_{\mathrm{i=1}}^{\mathrm{p}} \beta_{21\mathrm{i}} \Delta \mathrm{Y_{t-i}} + \sum_{\mathrm{j=1}}^{\mathrm{q}} \beta_{22\mathrm{j}} \Delta \mathrm{X_{t-j}} + \mu_{2\mathrm{t},} \tag{4}$$

3. Empirical results

3.1. Data

To investigate whether there is a causal relationship between calorie intake consumption and economic growth, data covering the period 1965 to 2005 are used. Calorie intake is expressed in calories/capita/day which are derived from national food balance sheets (FAO. 2005). The current GDP series was obtained from the Central Statistical Office and was transformed into real terms using GDP deflator obtained from the World Development Indicators (World Bank, 2005). Real GDP is expressed in million Mauritian rupees (MRU). The two series are presented in Fig. 1. It can be seen that they have both generally been trending upwards since 1965, suggesting a positive correlation between them.

Figure 1. Calories intake and real GDP

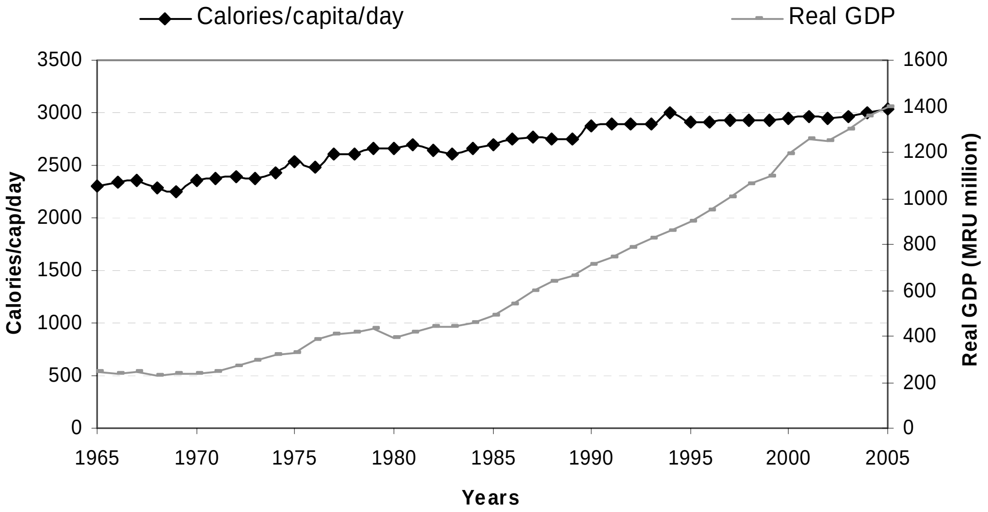
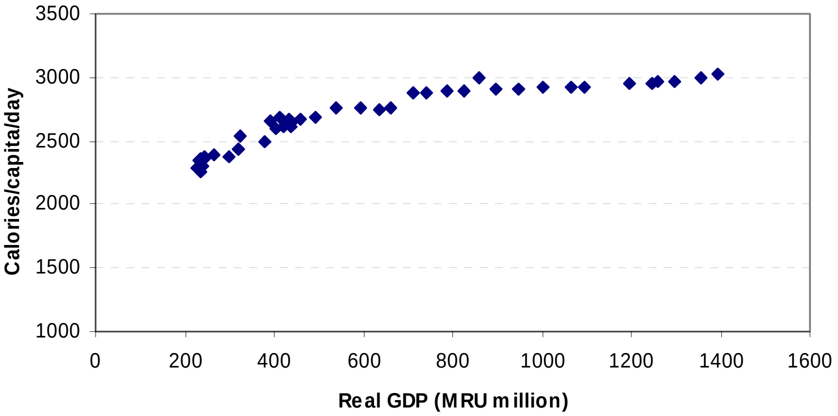


Figure 2 plots real GDP against calorie intake /capita/day. It shows that in general the nutritional status of Mauritius is correlated with its level of economic development, but also shows that this correlation is stronger when real GDP is below MRU 600 million as compared to when GDP is above MRU 600 million. The plot also tends to show the initial phases of the convergence effect, whereby the beneficial effect of nutrition on economic growth tapers off once calorie per capita per day intake exceeds 3000. The next section systematically checks whether there is a causality between intake and economic growth over a period of 40 years in Mauritius

Figure 2. Plot of calories intake and real GDP



3.2 Unit roots tests

The variables used in the models are respectively LCAL, natural logarithm of calorie intake per capita and LGDP, natural logarithm of real GDP. The PP provides the formal test for unit root in this study. The PP values of -0.334 and 0.496 for LEC and LGDP in levels are not significantly negative (Table 1.). This indicates the existence of unit roots and that both series are non-stationary. Therefore any causal inferences derived from them would be invalid. The series are therefore first-differenced and the PP tests carried on them. Non-stationarity is rejected for both series at the 1% level of significance, that is they are both I(1). The Granger-causality models are consequently estimated with the first-differenced series.

Table 1. Results of Phillips-Perron (PP) unit root tests

	Levels	First differences
Variables	PP values	PP values
LCAL	-1.03	-7.42[a]
LGDP	0.496	-5.90[a]

Note: [a] significant at the 1% level

3.3 Co-integration tests

Given that both calorie intake and GDP are integrated of order 1, it was checked whether they were co-integrated over the sample period. The results of the Johansen co-integration test for both series are reported in Table 2.

Table 2. Results of Johansen Cointegration tests

Null hypothesis	Trace statistic	1% Critical value
The number of co-integrating equation is zero (R=0)	5.33	20.04
The number of co-integrating equation is at most one ($R \leq 1$)	0.025	6.65

The results of the Johansen cointegration test are presented in Table 2. The trace statistics show that the null hypothesis of absence of cointegration (R=0) between the calorie intake and the GDP series cannot be rejected at the 1% level of significance, in favour of the presence of at least one cointegrating equation $(R \geq 1)$. The null hypothesis of the existence of at the most one cointegrating equation is also not rejected at the 1% level of significance. Therefore the absence of a cointegrating equation between GDP and calories intake implies that there is no long-run equilibrium relationship between the two series in Mauritius. Therefore the standard Granger causality test is used to investigate causality.

3.4. Granger causality

Regressions (3) and (4) were run to investigate short-run causality. The respective optimal lag lengths were chosen as i=6 and j=11 for equation (3) and i=11 and j=7 for equation (4), using Akaike's Information Criteria. This method removed the ambiguity involved in the arbitrary choice of the different lag lengths.

Once the model has been estimated, the cumulative sum of recursive residuals (CUSUM) test is applied to assess parameter constancy and check whether there might be a structural break. The results indicate no instability in the coefficients as the plots in figures 3 and 4 show that the CUSUM strays within the 5% critical bounds of parameter stability. The models are therefore stable over time. It appears that applying the standard Granger-causality test does not suffer from any problem caused by a structural break.

Figure 3. Plot of CUSUM when LGDP is the dependent variable

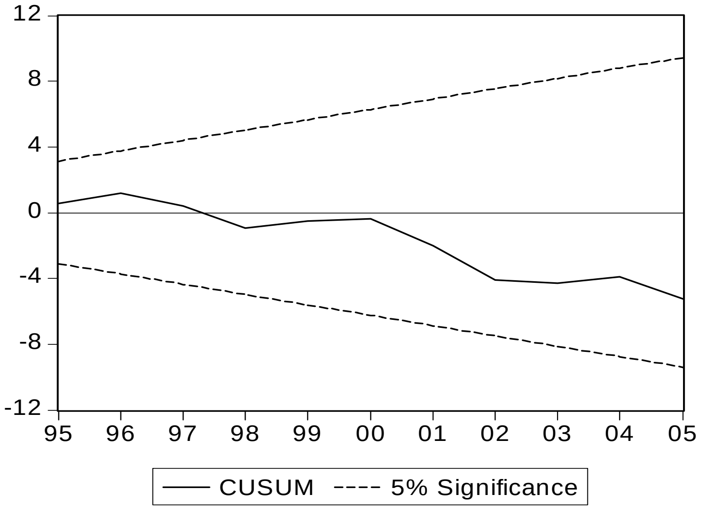
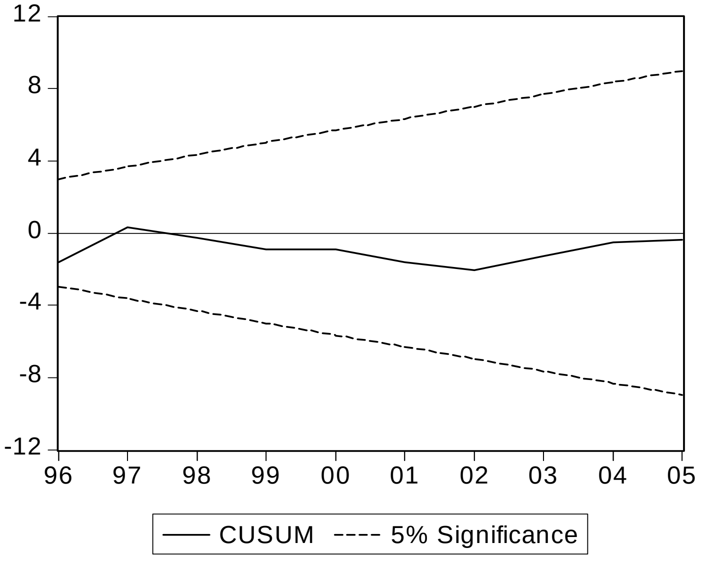


Figure 4. Plot of CUSUM when LCAL is the dependent variable



In the absence of the presence of a long-run relationship between calorie intake and real GDP an F test is used to determine whether a short-run relationship exists in either direction. The results are presented in Table 3. Both null hypotheses cannot be rejected at the 5% level, implying that short-run Granger causality does not run from calorie intake to GDP and *vice-versa*.

Table 3. Results of causality tests based on the error correction model

Null hypothesis	F statistics	P value
CAL does not granger cause GDP	1.88	0.1546
GDP does not Granger cause CAL	2.38	0.0915

4. Summary and conclusions

This article has used cointegration and Granger causality tests and uncovered the absence of both short- and long-run causality between daily per capita calorie intake and GDP. The neutrality hypothesis holds in Mauritius, whereby past values of calorie intake have no explanatory power for the current value of economic growth and *vice versa*. This finding contrasts with those from the literature (Dawson and Tiffin, 1998; Tiffin and Dawson, 2002) that have most of the time found unidirectional or bidirectional Granger causality between these two variables). This finding is rare, but is explained by a decoupling in calorie intake and economic growth that could be due to a more erratic growth in GDP as compared to the lower and more sustained growth in calories intake.

Other middle-income countries are continuing to experience changes in lifestyle, structures of diet, disease patterns, thus exacerbating the nutrition transition problem (Popkin and Ng, 2007). The same phenomenon will continue to affect Mauritius and can potentially worsen the already difficult situation as far as NCDs are concerned. The MoHQL has developed dietary guidelines to try to curb the problem by reducing the population's levels of major risk factors. (MoHQL, 2006). One of the recommendations is to reducing saturated fat, cholesterol intake and intake of refined sugars. This is tantamount to consuming a reduced-calorie diet. The implication of our results is that implementing a policy of reducing calorie intake from the actual level to curb the NCDs can be pursued and will not compromise economic growth and can be more strongly enforced as a nutrition intervention strategy. Here there is need to highlight that this policy should be adopted along with the myriad of other guidelines, as laid down in MoHQL (2006) and should target population segments that have higher than required calorie intake.

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