Long-run Overweight Levels and Convergence in Body Mass Index

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Abstract

We evaluate the hypothesis of convergence to an optimal long-run body weight worldwide. We formulate a simple rational non-addiction eating model to derive a testable equation that allows us to verify the existence of a long-run body weight as well as its estimation. We use a database of body mass index (BMI) estimates across countries over four decades published by the NCD Risk Factor Collaboration. We find that BMIs converge among European countries but not in the rest of the world. Consistent with the theoretical model, our long-run estimates suggest that European nations will show an average BMI above healthy levels. In particular, females and males will show average BMIs classified as overweight levels (BMI=28.3). Confidence intervals and sensitivity analysis suggest that males might reach long-term BMI levels associated with obesity (BMI>30). We discuss the implications of our findings from the perspectives of health economics and economic development.

JEL Codes: D91, I12, I31, O47.
Keywords: obesity, overweight, body mass index, convergence, rational eating model.

Highlights

- We evaluate the hypothesis of convergence to an optimal long-run body weight worldwide.
- A rational non-addiction eating model is formulated to derive a testable equation to verify the existence of a long-run body weight and its estimation.
- BMIs converge among European countries but not in the rest of the world.
- Long-run BMI estimates suggest that female and male Europeans will show high overweight (BMI=28.3).
- Sensitivity analysis suggests that males might reach long-term BMI levels associated with obesity (BMI>30).

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†Corresponding author. Department of Economics, Ohio University. Office: Bentley Annex 345. E-mail: toledot@ohio.edu. Conflict of Interest: the authors declare that they have no conflict of interest. We would like to thank three anonymous referees for helpful suggestions and comments. We acknowledge the excellent research assistance provided by Emily Schueller and Shaibu Yahaya. We thank Ralf Elsas for kindly providing a Matlab code for one of the estimations. The usual disclaimer applies.

1 Introduction

After years of persistent growth, obesity has become a serious concern owing to its well-known negative effects. Obese individuals have worse labor outcomes, more health problems (diabetes, strokes, and cancer, among others), and spend more on medical treatments for obesity-related diseases.[1] In this context, it would be desirable to characterize the evolution of this disease in the future. In particular, we are interested in assessing whether the upward trend in obesity will continue or become stable at a certain level.

In this study, we approach this question by applying the economic concept of convergence to the average body mass index (BMI, kg/m^2). Convergence rates are relevant to the extent that they give information on how fast certain nations are likely to catch up to those with relatively high BMIs. If countries converge in average BMIs, then all else being equal, the initial conditions do not matter in the long term. Similar to convergence in incomes, convergence in BMIs is not a desirable outcome per se. It also depends on the steady-state level the country approaches and how far that level is from a healthy one.

In practice, convergence in BMIs might arise from convergence in diets and physical activities across nations in an increasingly globalized world (Popkin and Gordon-Larsen, 2004). The increasing similarity in diets worldwide leads to what has been called the dietary convergence phenomenon (e.g., Popkin, 1993; FAO, 2004; Hawkes, 2006; Pingali, 2006; FAO, WFP and IFAD, 2012). Such a global diet is characterized by a greater reliance on staple grains, increased consumption of meat, dairy products, edible oil, salt and sugar, and a lower intake of dietary fiber (FAO, 2004).

Theoretically, we show that, under certain conditions, the concept of convergence in BMIs can be easily derived from a simple rational non-addiction eating model. As other studies show, this model also predicts that the optimal long-run body weight could be above its healthy

[1] For a review of the economic consequences of obesity, see Cawley (2015).

level (Levy, 2002; Dragone, 2009). Thus, the model allows us to derive a testable equation to evaluate convergence and, potentially, to estimate a stable long-run BMI.

Our econometric models are estimated using data from a recently publicly available database (NCD Risk Factor Collaboration, 2016) with estimates of average BMI per gender across 172 countries between 1975 and 2014. We estimate cross-sectional and dynamic panel models for the world and different subsamples including 45 European countries. Given the data features and the empirical model, we employ a consistent and efficient estimator for the dynamic panel following Kiviet (1995) and Bruno (2005a,b), and check the robustness of our results in different dimensions.

We find that BMIs do not converge worldwide. However, European countries converge in BMIs. We argue that this fact might be associated with common food patterns, agricultural policies, and health policies implemented by European countries. Our long-run estimates suggest that European nations will show an average BMI above the upper limit of the range of healthy levels ($BMI = 25$; see WHO, 2000, 2004), and both females and males will show average BMIs classified as overweight levels ($BMI = 28.3$). Confidence intervals and sensitivity analysis suggest that males might reach BMI levels associated with obesity in the long term (average BMIs above 30). According to our point estimates, men converge relatively faster to their steady states than do women.[2] That said, this difference is statistically insignificant across alternative model specifications.

We aim to contribute to three strands of the literature. By testing its predictions about long-run overweight levels and convergence in BMIs, our study relates to the literature on the rational eating model developed over the last two decades (see Philipson and Posner, 1999; Levy, 2002; Lakdawalla and Philipson, 2009; Dragone, 2009; Dragone and Savorelli, 2012;

[2]In particular, the time that it takes to eliminate half the initial gap between the BMI in 1975 and its long-run level is more than a century for women and about half of that for men.

Buttet and Dolar, 2015). To the best of our knowledge, this is the first time the model's predictions have been tested empirically across countries.[3]

Second, we extend the convergence literature in development economics, which usually focused on incomes across individuals or countries, but also extended to other indicators related to health outcomes and living standards such as life expectancy and calorie intake (Ingram, 1992; Hobijn and Franses, 2001; Sab and Smith, 2002; Becker *et al.*, 2005; Mazumdar, 2003; Neumayer, 2003; Kenny, 2005; Ram, 2005; Soares, 2007; Clark, 2011; Weil, 2014; Apergis and Georgellis, 2015). In our view, there is disagreement in the existing literature. On the one hand, a number of studies find either convergence in levels or a fall in cross-country dispersion in life expectancy and other health indicators such as calorie intake and infant survival (Ingram, 1992; Sab and Smith, 2002; Neumayer, 2003; Kenny, 2005). Along this line, Weil (2014) argues that in the last fifty years, the convergence in health has been much faster than the convergence in income. On the other hand, other works suggest a different conclusion. Hobijn and Franses (2001) finds convergence in life expectancy, daily calorie supply, daily protein supply, and infant mortality rate, but only in certain groups of countries. Mazumdar (2003) shows evidence that supports divergence in life expectancy at birth, infant survival rate, calorie intake, and other indicators in a sample of 92 countries. Our contribution lies in this segment of the literature. Though we find convergence in body weights among Europeans, they converge to unhealthy levels and only European nations converge among the countries in our world sample.

Third, our work complements another branch concerned with forecasting of obesity indicators (see Kelly *et al.*, 2008; Wang *et al.*, 2008; Mills, 2009; Stamatakis *et al.*, 2010; Haby *et al.*, 2011; Wang *et al.*, 2011; Finkelstein *et al.*, 2012; Majer *et al.*, 2013). Our evidence suggests that body weights among European nations, on average, may be reaching a stable long-run level, all else equal. In this sense, our findings back the forecasting models that predict a leveling off of European BMIs in the future over those that include linear trend components and predict unbounded growth in obesity indices.

[3]The literature on convergence in obesity is scarce. For the only studies of convergence in obesity prevalence rates across US states, see Li and Wang (2016a,b).

Finally, our findings suggest different policy challenges and research agendas across world regions. In the rest of the world, where we do not observe converge in BMIs, we need to be cautious because of the heterogeneity in BMIs across economies and over time. It is necessary to identify the countries that might be approaching levels of BMI classified as overweight and those that show a sharp upward, perhaps alarming, trend in their BMI series. In that sense, it is desirable to investigate what type of policies could drive countries to converge to healthy levels of BMI. The convergence observed among heterogeneous countries as the European nations, as well as the variety of their policies, lead to the natural task of assessing what kind of public policy (health, food, agricultural, trade, etc.), if any, has been effective to prevent higher obesity and overweight levels. In any case, given the low convergence rates we find in the European sample, policies that seek to reduce obesity in the world have to be as persistent as the BMIs series. For the same reason, any serious evaluation of the effectiveness of such policies would require more than a few years.

The paper proceeds as follows. The next section briefly formulates the model's predictions and explains the economic intuition. Section 3 focuses on the main features of the data. Section 4 presents the empirical models, results, and robustness checks. Section 5 discusses our findings and conjectures some explanations. Section 6 concludes with some final remarks.

2 The Model

Our basic setup considers the most important features that rational eating models in the literature have in common (see, e.g., Philipson and Posner, 1999; Levy, 2002; Dragone and Savorelli, 2012).[4] Consider a representative agent that chooses sequences of consumption and weight in order to maximize

$$U = E_0 \sum_{t=0}^{\infty} \beta^t \left[c_t \left(\overline{c} - \frac{c_t}{2} \right) - \frac{b}{2} \left(w_t - w^I \right)^2 \right] \tag{1}$$

[4]See also Dragone (2009); Lakdawalla and Philipson (2009), Buttet and Dolar (2015), and Barbieri (2016).

subject to a law of motion of weight

$$w_{t+1} - w_t = \phi c_t - \delta w_t + \epsilon_{t+1}, \tag{2}$$

an initial weight ($w_0 > 0$), non-negativity constraints, and the corresponding transversality condition. In this setup, $0 < \beta < 1$ is the subjective discount factor, c denotes food consumption, w is the individual's body weight, $0 < \delta < 1$ is the rate at which the individual burns calories and, therefore, loses weight (e.g., metabolism), ϕ is the marginal effect on weight from one unit of food consumption, $\overline{c}$ can represent the satiation consumption level (more on this below), b is a positive preference parameter, and $w^I > 0$ is the ideal body weight.[5] We include a zero-mean i.i.d. term ϵ_{t+1} to capture measurement errors and non-systematic unpredictable changes in weight observed every period, but that are nil on average (e.g., diet-binge cycles, change in weight owing to illness or accidents, pregnancy, etc.). We assume that the adult individual's weight is rescaled by height and normalized, for simplicity, to 1. Thus, body weight and BMI are equal in the model.

We follow Dragone and Savorelli (2012) in interpreting $\overline{c}$, alternatively as the solution of a standard static problem in which the individual maximizes utility by choosing amounts of food and a non-food goods subject to a budget constraint. In such a case, if food is a normal good, $\overline{c}$ increases with income and decreases with the price of food (see Appendix A in Dragone and Savorelli, 2012).

The next two model predictions support the convergence hypotheses that we test in Section 4.[6]

[5]The ideal weight can be understood as a convex combination of a physiologically optimal weight (w^H) and a subjective or socially desirable body weight (w^S). That is, $w^I = \mu w^S + (1-\mu)w^H$, with $0 < \mu < 1$. The introduction of w^I as a weighted average helps to accommodate different setups with only w^H (as in the baseline model by Levy, 2002, Dragone, 2009, and Buttet and Dolar, 2015), or with both w^H and w^S (as in Levy, 2002, Dragone and Savorelli, 2012). Given the exogeneity of w^I, the assumption is virtually innocuous for our analysis.

[6]Appendix A provides the proofs.

Prediction 1. Steady-state overweight. Let $\overline{w}$ be the weight associated with satiation level $\overline{c}$. If $\overline{w} > w^I$ in the steady-state equilibrium, then the optimal body weight is above the ideal weight:

$$w^{ss} > w^I \tag{3}$$

Intuitively, if $\overline{w} > w^I$, then the rational individual faces the trade-off in choosing a weight level between $\overline{w}$ and w^I in the steady state. An individual choosing the satiation level (associated with $w = \overline{w}$) reaches the highest utility from food consumption, but at the cost of high disutility from being far from the ideal level w^I. An individual choosing a level of consumption associated with $w = w^I$ minimizes the disutility from having a weight different from the ideal level, but at the cost of lower utility from consuming food below the satiation point. The optimal weight is a point between these two extremes. An analogous reasoning would disregard weights above $\overline{w}$ or below w^I as optimal. Therefore, the steady-state weight is above the ideal weight.

While the prediction is not novel,[7] to the best of our knowledge, it has not been tested empirically before and it is useful for the analysis that follows. The next proposition will help us formulate the empirical model.

Prediction 2. Convergence in weights. The solution to the linear-quadratic problem above yields an optimal weight that evolves as

$$\Delta w_{t+1} = \alpha_0 + \alpha_1 w_t + \epsilon_{t+1} \tag{4}$$

with $\alpha_0 > 0$ and $-1 < \alpha_1 < 0$. In the equation above, a negative sign on α_1 implies convergence in body weights. An analogous equation, with GDP growth instead of the change in body weight, is derived from a neoclassical growth model in the literature of growth economics (see, e.g., Barro and Sala-i-Martin, 1992; Durlauf *et al.*, 2009).

[7] For a similar proposition, see Levy (2002 and 2009) or Dragone (2009).

As shown in Appendix A.2, α_1 in equation (4) depends on preference parameters (b and β) and coefficients that could be associated with obesity genes (δ and ϕ). The latter is in line with the concept of intergenerational transmission of weight studied by Classen and Thompson (2016) and Dolton and Xiao (2017).[8]

The intuition behind the prediction is as follows. Consider two representative adult individuals from two different countries (say, L and H) that share the same steady-state weight and whose initial body weights are below this steady-state level ($w_{0,L} < w_{0,H} < w_L^{ss} = w_H^{ss}$). In the transitional dynamics, the representative individual with low weight tends to gain weight faster than the individual with higher weight because the former is further from her steady-state equilibrium and, therefore, the utility from increasing consumption is larger than the disutility from gaining weight (compared to the latter individual). It is optimal for her to gain weight faster than the individual who is closer to her steady state. The high-weight individual still enjoys utility from increasing consumption over the disutility from gaining weight, but in a smaller magnitude than the low-weight individual. The equation above allows us to test convergence and, in turn, to verify if the deterministic steady-state (height-adjusted) body weight is above the ideal level. The latter will hold if $w^{ss} = -\alpha_0/\alpha_1 > w^I$.

3 Data

The NCD Risk Factor Collaboration (2016; NCD-RisC, henceforth) developed the BMI database. The data collection considers 1,698 population-based data sources, with more than 19.2 million adult participants (9.9 million men and 9.3 million women) in 200 countries and territories between 1975 and 2014. NCD-RisC aggregates the population-based survey to construct data at a country level. Thus, our unit of observation is a country. When we mention the average BMI of a female or a male individual in our empirical analysis, we refer to the representative (female or male) individual of each nation. This is an important point to bear in mind when

[8]See Silventoine *et al.* (2010) for a review of the evidence of genetic influence in obesity.

interpreting our results. That said, we also report estimates using population weights as a robustness check (more on this in the next section).

From this database, we first focus only on economies with GDP data available. Thus, our world sample consists of 172 economies. We split the full sample in different world regions but, for the sake of simplicity, we group countries into European and non-European regions only. Other subdivisions of the non-Europe sample (e.g., Asia-Oceania, Africa, or the Americas) in the empirical analysis that follows provide similar conclusions.[9] The criterion adopted defines Europe in a wide sense to maximize the number of observations. Hence, our European sample contains 45 countries and the non-European sample includes 127 countries (for the list of countries, see Appendix B). Table 1 shows the descriptive statistics of the BMI indicators and other variables used in the analysis.

This database is suitable for a study of convergence due to its long-run BMI series and the coverage of a broad collection of countries and years. Moreover, the BMI series are age-standardized and do not use self-reported height and weight.[10] Likewise, we deal with some potential issues worth mentioning. First, we opt to use unadjusted BMI series, that is, those that were not calculated using covariates (income, urban population rate, and schooling years) to minimize any artificial influence of such covariates in our results. Table 1 shows descriptive statistics of both datasets. As we can see, there are not important differences in the summary statistics.[11] Second, the BMI estimates tend to show – as one could expect – high standard errors during the initial periods of the sample, especially in the 1970s. We therefore assess the sensitivity of our main estimates to a truncated sample that starts in 1980.[12]

[9] The results from other subdivisions are available upon request.

[10] This is important because self-reported data "are subject to biases that vary by geography, time, age, sex, and socioeconomic characteristics" (NCD-RisC, 2016; p.1378).

[11] The unadjusted dataset was kindly provided by the NCD-RisC upon our request. The NCD-RisC notes that BMI data with and without covariates lead to almost identical results in most economies, except those that do not have any data available. These are Brunei, Bermuda, and North Korea. The latter is not part of our analysis due to the lack of GDP data. We use adjusted data (calculated with covariates) as a robustness check only.

[12] Naturally, another potential limitation is related to BMI data quality particularly in low-income developing economies.

The rest of the controls variables are PPP-adjusted real GDP per capita, human capital indices, and urbanization rates. The first two come from the Penn World Tables (see Feenstra *et al.*, 2015), whereas the latter comes from the World Development Indicators (World Bank).

[Table 1 about here.]

4 Estimation and Results

In this section, we use equation (4) as well as cross-sectional and panel data to test the hypothesis of convergence in BMIs and verify whether the long-run BMI, if it exists, is above its healthy level.

4.1 Cross-sectional Evidence

Let w_{it} be the average height-adjusted body weight or BMI index for a country i in period t. In Appendix A.3, we show that equation (4) implies a relationship between the initial weight and the average change in weight between the initial and final periods ($(1/T)(w_{iT} - w_{i0})$):

$$\frac{w_{iT} - w_{i0}}{T} = \gamma_0 + \gamma_1 w_{i0} + \nu_{iT} \tag{5}$$

where T is the final sample period and ν_{iT} is a classical error term. The change in BMI is properly rescaled by the number of periods to have changes at a yearly frequency and results comparable with the panel estimations below. Our interest is to estimate the parameters γ_0, γ_1, and test the null hypothesis $\gamma_1 \geq 0$, with the alternative of convergence, $\gamma_1 < 0$. We can obtain the steady-state BMI value with the parameter estimates and the ratio $-\gamma_0/\gamma_1$ (Appendix A.3 shows the derivations).

We estimate the model using average BMI data for both the representative female and the representative male individual. Table 2 displays the parameter estimates and (bootstrapped) standard errors for the three samples (world, non-Europe, and Europe). The table also shows

other estimates and statistics, including the steady-state BMI, its 95%-confidence interval, the p-value related to the null hypothesis that the long-run BMI is lower than 25 (steady-state under-weight), the p-value related to the null hypothesis that $\gamma_1 \geq 0$ (no convergence), and the half-life (number of years it takes to eliminate half the initial gap between the 1975 BMI and its steady-state level).[13] We assume that the ideal BMI, w^I, equals the healthy BMI value of 25. This is probably a conservative value since it is simply the upper bound of the range between 18.5 and 25 found in the literature.[14]

[Table 2 about here.]

We begin by estimating the model using the world sample. According to columns (1)-(2) in Table 2, we find convergence in BMIs among representative females, but not among representative males of each country. The slope parameter is negative and statistically significant at conventional levels only for females. Columns (3)-(4) reveal no convergence among non-European women and men. In contrast, the results in columns (5)-(6) allow us to reject the null of no convergence for the European sample. In other words, two conclusions emerge from this first set of results. First, Europe seems to be a convergence club in BMIs. Second, the convergence of female individuals' weights observed in the full sample is driven mainly by Europe's observations because there is no evidence of convergence in the rest of the world.

Figures 1 and 2 illustrate the cross-sectional evidence of convergence. The scatter plots display pairs of the initial (1975) BMI and the average change in BMI between 1975 and 2014 for female and male individuals from each country. The figures also show the corresponding regression fit. The graphs suggest a catching-up effect in Europe (bottom panels in Figures 1

[13] When the null of no convergence is not rejected, the steady-state estimate and its variance take unreasonable values (e.g., negative long-run BMI with large variance). Hence, we report estimates and statistics of interest only if the null of no convergence is rejected.

[14] For example, Berrington de Gonzalez *et al.* (2010) conclude that the age-standardized rate of death from any cause is generally lowest among individuals with a BMI of 22.5 to 24.9. The WHO (2000, 2004) considers any BMI between 18.5 and 24.99 as normal weight. As we mentioned in Section 2, the ideal weight or BMI is not necessarily equal to the healthy BMI. However, because we do not observe ideal BMIs by country, we assume the most conservative value of a healthy BMI as the ideal BMI, which would be consistent with the literature above-mentioned.

and 2): countries with relatively low BMIs in 1975 tended to gain weight faster, on average, than those with relatively high BMIs. The opposite is shown in the non-European sample for males, whereas the convergence coefficient is slightly negative but statistically insignificant for females in non-European nations.

[Figure 1 about here.]

[Figure 2 about here.]

According to the cross-sectional evidence, the average female in Europe converge at a rate of 0.019 (kilograms per square meter) per year, whereas the average male shows a lower rate, 0.010. Seemingly, these are relatively low rates of convergence. In contrast to the rates of convergence in per capita GDP (around 0.02 per year),[15] these rates might look small. From our viewpoint, this is not surprising. Strictly speaking, these rates are not comparable. Body weights are stocks, whereas per capita GDP measures are flows and are therefore susceptible to more abrupt changes over a given period time. Likewise, in the rational eating model discussed above, the individual's body weight is a stock and food consumption is a flow.[16]

Table 3 shows a number of robustness checks for the European sample. Columns (1)-(2) display robust LS estimates, which seek to minimize the effects of potential influential observations on our estimates (see, for example, the dots at the upper left corner in the European panel of Figure 1).[17] As expected, the slope parameters are negative and statistically significant at conventional levels. Columns (3)-(4) show that the corresponding estimates are not considerably different if we restrict the sample period and start in 1980. Next, we include other

[15] This is the so-called "iron-law" rate of 2% (see Barro, 2015).

[16] If we extend the model above by including the costs of changing consumption habits in the utility function, as in Dragone (2009), we could obtain another theoretical reason to explain the slow convergence in body weights. As the author contends, in that case, the optimal path implies a slower convergence, but with fluctuations above and below the steady-state body weight. Another component of the persistence of BMI is the intergenerational transmission of genes (Classen and Thompson, 2016).

[17] We use an M-estimator with a Huber objective function and Gaussian efficiency of 95%. The results using an MM-estimator provide similar conclusions and are available upon request.

regressors to verify if these controls might influence our conclusions. To that end, we estimate

$$\frac{w_{iT} - w_{i0}}{T} = \gamma_0 + \gamma_1 w_{i0} + \boldsymbol{\gamma_2'}(\mathbf{x_{i0}} - \mathbf{x_{i0}^*}) + \nu_{iT} \tag{6}$$

where $\boldsymbol{\gamma_2}$ denotes a column vector of coefficients and $(\mathbf{x_{i0}} - \mathbf{x_{i0}^*})$ is a column vector of controls expressed in deviations from their long-run levels. These control variables include GDP per capita, a human capital index, and urbanization rate (see Appendix B for further details about the definitions and sources). We include controls in percent deviations from their long-run levels to create an empirical model consistent with the steady-state equilibrium implied by the theoretical model (i.e, $\mathbf{x_{i0}} - \mathbf{x_{i0}^*} = 0$ in the long run). We estimate long-run components using a Hodrick-Prescott trend for GDP per capita, and sample means for human capital index and urbanization rate.

As columns (5)-(6) in Table 3 show, the parameters of these controls are not statistically significant (with p-values of 0.653 and 0.94) and the main conclusions do not change substantially. We observe relatively high standard errors, which, in turn, enlarge the confidence intervals for the long-run BMI estimates (see columns (5) and (6)). This might be related to a smaller sample compared to that used in the baseline models, or the inclusion of statistically irrelevant regressors.

[Table 3 about here.]

In the last robustness check of this cross-sectional analysis, we re-estimate the baseline models using adjusted BMI data (columns (7)-(8)). The results again verify both model's predictions. The parameter values and signs are as expected. The convergence rates (0.016 and 0.012) are slightly different from those in the baseline case (0.019 and 0.010), but the differences are not substantial.

4.2 Panel Data Evidence

In this section we exploit both the cross-sectional and time dimension of the data. For that purpose, we estimate:

$$\Delta w_{it} = \alpha_0 + \alpha_1 w_{it-1} + \epsilon_{it} \tag{7}$$

with $\epsilon_{it} = \eta_i + \mu_t + \nu_{it}$, $E_t \epsilon_{it} = 0$, where η_i and μ_t represent fixed country and time effects with zero mean.[18] In light of the theoretical model, fixed country effects may arise from heterogeneities such as different ideal weights or satiation points across countries. Time effects, in turn, allow us to capture either common shocks that affect BMIs differentially over time or short-run trend components as part of a transitional dynamic toward a steady state.[19]

Our panel estimations imply a cross-sectional dimension (N) similar to the time series dimension (T). It is well known that for small T and large N, the fixed-effect or least-squares dummy variable (LSDV) estimator of an autoregressive panel data model is inconsistent (Nickell, 1981). Prior studies propose a number of estimators that attempt to deal with this issue. Monte Carlo evidence suggests that LSDV is inconsistent but has a smaller variance than the IV and GMM estimators do (Arellano and Bond, 1991; Kiviet, 1995; Judson and Owen, 1999). Along this line, Kiviet (1995) proposes an approximation to the small sample bias of the LSDV estimator. This biased-corrected fixed effects estimator (LSDVC) tends to show a lower bias and a lower root mean squared error compared to the IV and GMM estimators even when the panel is unbalanced (Bruno, 2005). Based on Monte Carlo simulations, Judson and Owen (1999) also recommend the LSDVC estimator. Moreover, recent Monte Carlo studies coincide and suggest that bias-corrected fixed-effects estimators are the most accurate and robust esti-

[18] In the jargon of the growth convergence literature, we are not testing for absolute convergence, but for conditional convergence; that is, conditional to each country's characteristics.

[19] We reject the null hypothesis that the panels contain unit root processes in our samples (against the alternative of stationary panels) using the Levin, Lin and Chu (2002) test at standard levels of significance. Likewise, we reject the null hypothesis that all panels contain unit root processes in our samples (against the alternative that at least one panel is stationary) using the Fisher test (PP Chi-square statistic) at standard levels of significance. We use a constant, a linear trend, and Bartlett kernel in each test. The statistics and p-values are available upon request.

mators compared to the IV and GMM estimators (Flannery and Hankins, 2013; Dang *et al.*, 2015). It is worth highlighting that the LSDVC assumes exogeneity in the regressors, except for the lagged dependent variable. This assumption constitutes a limitation in some cases, but it is virtually harmless for the purposes of our study because we are mainly interested in the constant and the auto-regressive parameter. We do not include other regressors in our baseline model, though when we do, it is for sensitivity purposes and we are not particularly interested in the coefficients on those regressors. All things considered, we opt to estimate the model using LSDVC.

Once again, we first report estimates and statistics for the three samples. Table 4 reports our main panel data results using LSDVC. We confirm the conclusion from the cross-sectional analysis that females' BMIs converge in the world sample (see column (1)) only because there is convergence in Europe (column (5)).

Columns (5)-(6) show our preferred (baseline) results. We find the expected signs and values for our model parameters and the convergence rates are statistically significant. Again, we find relatively low rates of convergence. Female and male Europeans converge at rates of 0.006 and 0.015 per year, respectively. If we assume that the mean heights of females and males are 1.70 and 1.80 meters (approximately 5.6 and 5.9 ft.), respectively, the estimated convergence rates imply that, over a decade, the average woman and average man gain approximately 0.18 kg. (0.4 pounds) and 0.5 kg. (1.1 pounds), respectively. In turn, the estimated convergence rates entail half-lives of 110 and 45 years, respectively (last row of Table 4). In other words, it takes more than a century to eliminate half the gap between the female BMI in 1975 and its long-run level.

Our baseline results also show that the steady-state weights are above healthy levels as the theoretical model predicts (recall prediction 1). In the long run, European nations will be overweight with a BMI level of 28.3 (columns (5)-(6) from Table 4). For male weights, we obtain wider 95%-confidence intervals. Statistically speaking, the female confidence interval ([27.8 29.1]) neither includes nor is included in the male interval ([27.9 32.5]).

[Table 4 about here.]

Table 5 reports a few additional exercises to verify the robustness of our baseline results. As in the cross-sectional analysis, we control for other potential regressors and estimate

$$\Delta w_{it} = \alpha_0 + \alpha_1 w_{it-1} + \boldsymbol{\alpha_2'}(\mathbf{x_{it}} - \mathbf{x_{it}^*}) + \epsilon_{it} \tag{8}$$

where $\boldsymbol{\alpha_2}$ is a column vector of parameters, and $(\mathbf{x_{it}} - \mathbf{x_{it}^*})$ is a column vector of controls in deviations from long-run levels similar to that defined above. In this case, we verify our previous results again (see columns (1)-(2)). Convergence is present among European nations and the long-run BMIs are above healthy levels.

Columns (3)-(4) show the results when we drop the first periods, those that show more uncertainty in the BMI estimates, and start to estimate in 1980. Our results are not sensitive to this modification and the main conclusions are unchanged. Finally, columns (5)-(6) display the estimates when we use the adjusted BMI data reported by NCD-RisC (2016). In all of these robustness checks, we obtain statistically significant convergence rates in the BMIs levels of both European females and males. The steady-state overweight (average) BMI levels among European countries (in female and male samples) are a robust finding. Moreover, note that confidence intervals for male Europeans include BMI levels above 30 (column (6) in Table 4, columns (2), (4), and (6) in Table 5) and the estimates using adjusted data deliver a point estimate of 30.8 (column (6) in Table 5). That is, based on these data, the results support the possibility of a long-run obesity among European males.

[Table 5 about here.]

Further modifications of the baseline model that consider population weights and different divisions of the European sample such as (i) Mediterranean nations, (ii) the European Union, and (iii) Eastern Europe (including the Russian Federation), with their corresponding complements, can be found in the Appendix (Tables A1 and A2). The lists of countries in each

subgroup are included in Appendix B2 and the note of Table A2. We find that BMIs converge when we use population weights and even within those subsamples of European countries. Notably, we find convergence in BMI among EU members. Likewise, steady-state BMIs are above healthy levels in these alternative specifications using a 5% significance level.

It is worth highlighting that, despite we comment some gender heterogeneities in point estimates of convergence rates and long-run BMIs, such differences are statistically insignificant across the various specifications we report in the robustness checks. That is, we cannot affirm that we find robust gender differences across our alternative specifications (see columns (5) and (6) in Table 4, as well as Tables 5, A1, and A2).

5 Discussion

The upward trends in BMIs, sometimes viewed as alarming, seem to exist worldwide. Europe is an exception to a certain extent. In the terminology of growth economics, European nations form a convergence club. The evidence above suggests that Europeans converge in height-adjusted weights. Put differently, the rising trend of BMIs in Europe is, at least on average, related to a catching-up effect. Through the lens of the simple theoretical model discussed in Section 2, it constitutes a trajectory toward a steady-state equilibrium. Nevertheless, convergence of BMIs is not a desirable outcome per se. As we commented in the introduction, it also depends on the long-run equilibrium that these dynamic behaviors approach and the distance of this equilibrium from a healthy level. The steady-state estimates in the baseline models suggest that European nations are converging towards an average BMI of 28.3, a relatively high level in the overweight BMI category. Again, this value is above the upper limit of the range of healthy BMIs and most of the confidence intervals for long-term BMIs do not include healthy levels. These results motivate a key question: Why do adults' weights converge in Europe and not in the rest of the world? After we address this question, we discuss the implications of our results for obesity forecasting.

5.1 Why do European countries converge in weight?

European nations show convergence in BMI because their governments –in contrast to those from other world regions– have regularly applied food and health policies that, combined with other market regulations and trade policies, have tended to moderate, directly or indirectly, intentionally or unintentionally, the growth in their citizens' weights.

Common food patterns and agricultural policies

Some literature suggests the formation of a common food pattern in European countries. Grigg (1993) argues the existence of convergence in the nutritional composition of European diets. Gil *et al.* (1995) and Gracia and Albisu (2001) predict a relatively low growth in food consumption in the European Union (EU) because it has reached a level close to its maximum. Furthermore, it is possible that the historical regulation of the agricultural sector, jointly with trade barriers, determined the number of available food products and possible shaped consumption habits towards healthier foods to a certain extent.[20] The European Common Agricultural Policy may have contributed to the homogenization of food production and consumption patterns in that direction. As long as consumption habits persist over time, then our findings of convergence in BMIs could be related in part to this pattern of food consumption, which seems to be more frequently observed in Europe than in other world regions. This implies, in turn, a flatter BMI curve over time, and consequently, a higher convergence rate among European nations.

Health policies

Sisnowski *et al.* (2015) report a wide array of supranational and national regulatory approaches that the EU members have followed to reduce obesity. Supranationally, EU regulations cover aspects such as consumer information through nutrition labeling, marketing practices, food reformulation, and setting-specific nutritional standards, among others. At the national level, governments' efforts aimed to regulate food advertising (4 out of 28 EU coun-

[20] In this line of argument, Cutler *et al.* (2003) contend that people in more regulated countries, and particularly, more regulated agricultural sectors, tend to be less obese.

tries), nutrition labeling (6 countries), product reformulation (6 countries), and taxation (3 countries).

Some examples can illustrate this point. First, the most important differences in the policies applied in the EU with respect to those in the US rely on food advertising, product reformulation, and taxation (see Table 2 in Sisnowski *et al.*, 2015). Second, Denmark, France, Hungary, and Iceland have used different tax instruments to change food purchasing behaviors.[21] Third, limitations to trans fat content are in place in six EU states (Austria, Belgium, Denmark, Greece, Sweden, and the UK) and Iceland.

Though the attempts and advances in food and health policies in Europe probably played a role in curbing BMI growth, such policies probably had limited effectiveness for at least two reasons. First, these policies were applied especially over the last years of our period of analysis, which is consistent with the view that it was not until the 2000s that the EU focused on overweight individuals and obesity (Kurzer and Cooper, 2011). Second, as Sisnowski *et al.* (2015) argue, such regulations are limited in reach and scope. Thus, although these food and health policies could have helped convergence in BMI, they can be only part of a more comprehensive explanation.[22]

Through the lens of the simple rational eating model, the preferences for local healthy goods and especially the concern about healthy levels of weight, probably represented or induced by the policies mentioned above, can be captured by the parameter that governs the disutility of deviations of the individual's weight from the healthy weight (b in the utility function). It can be shown that the higher the value of b, the higher the convergence rate toward a (lower) steady state.[23]

[21] Denmark abolished the tax on saturated fat in 2013.

[22] Social environmental characteristics can be another mechanism through which health policies can influence BMIs. For example, Raftopolou (2017) shows that green areas or neighborhood safety positively affect individual and women's BMI and obesity in Spain.

[23] On the other hand, from a macroeconomic viewpoint, one could argue that convergence in height-adjusted weight is a consequence of convergence in per capita GDP. However, the empirical evidence is not conclusive about per capita GDP convergence in European economies (see, e.g., Cappelen *et al.*, 2003). In addition, even if convergence in income per person holds in our sample, there might be third factors, such as human capital, institutions, or technology advances, behind both types of convergence. For instance, Cutler *et al.* (2003; p.116)

5.2 Implications for BMI forecasting

Epidemiological forecasting focuses on predicting the BMI and prevalence rates needed for population health planning and evaluation. The rational eating model jointly with our results, also has implications in BMI forecasting, at least for Europe.

Several models and techniques have been proposed to predict BMI: multiple linear regression models (Haby *et al.*, 2011) and generalized additive models (Majer *et al.*, 2013), among others. Other works focus on forecasting prevalence rates mostly related to overweight individuals and obesity (Kelly *et al.*, 2008; Mills, 2009; Wang *et al.*, 2011; Finkelstein *et al.*, 2012). The use of linear trend components to forecast BMI or prevalence rates is not unusual (Stamatakis *et al.*, 2010; Haby *et al.*, 2011; Wang *et al.*, 2011; Finkelstein *et al.*, 2012). Notably, Wang *et al.* (2008) projects a prevalence rate of 100% in the overweight and obese categories in the US by 2048.

In our view, the reduced-form of the rational eating model derived and estimated above suggests an alternative to predict average long-term BMIs. If the average short- or medium-term trends we observe are actually a transitional dynamic toward a long-run equilibrium, as our model suggests, then there exists an upper bound to any forecast of future BMI that practitioners and forecasters should consider in European countries.[24] Put differently, our findings back the forecasting models that predict a leveling off of BMIs in the future over those that include linear trend components and predict unbounded growth in BMIs.

6 Summary and Final Remarks

In this paper, we examine two predictions of the rational non-addiction eating model: long-run overweight BMI levels and convergence in BMIs level. Despite the upward and seemingly

contend that "Over the broad sweep of history, improvements in health and income are both the consequence of new ideas and new technology, and one might or might not cause the other."

[24] Likewise, we should observe an upper limit to prevalence rates if the distribution of BMIs and the parameters that govern its variance do not change in the long run.

alarming trends in the prevalence of overweight and obesity in many countries, we identified a world region in which average BMIs tend to converge across economies: Europe. In turn, we found limits to the rational eating model in explaining long-run dynamics of body weights. We believe that much still remains to do to extend or improve this theoretical framework.

Our results suggest different policy challenges and research agendas across world regions. In the rest of the world, in which we do not observe converge in BMIs, we need to be cautious because of the heterogeneity in BMIs across economies and over time. It is required to identify the countries that might be approaching levels of BMI classified as overweight and those that show a sharp upward, perhaps alarming, trend in their BMI series. In that sense, it is desirable to determine what type of policies could drive countries to converge to healthy levels of BMI. The convergence observed among heterogeneous countries as the European nations, as well as the variety of their policies, lead to the natural task of assessing what kind of public policy (health, food, agricultural, trade, or other[25]), if any, has been effective to prevent higher long-run BMIs.

In any case, given the low convergence rates found in Europe, probably due to the persistence in eating habits and physical activities, policies that seek to reduce obesity in the world have to be as persistent as the BMIs series. For the same reason, any serious evaluation of the effectiveness of such policies would require more than a few years.

[25] Educational policy could be added to the list (see, e.g., Etilé, 2014).

Appendix

A Mathematical Appendix

A.1 Proof of Prediction 1.

The Lagrangean function associated with the optimization problem is

$$\mathcal{L} = \sum_{t=0}^{\infty} \beta^t E_t \left\{ c_t \left(\overline{c} - \frac{c_t}{2} \right) - \frac{b}{2} \left(w_t - w^I \right)^2 + \lambda_t \left[w_{t+1} - (1-\delta) w_t - \phi c_t - \epsilon_{t+1} \right] \right\} \tag{1}$$

where λ_t is the Lagrange multiplier. The first-order conditions are

$$\frac{\partial \mathcal{L}}{\partial c_t} = \overline{c} - c_t - \phi \lambda_t = 0 \tag{2}$$

$$\frac{\partial \mathcal{L}}{\partial w_{t+1}} = \lambda_t - \beta E_t \left[b \left(w_{t+1} - w^I \right) + (1-\delta) \lambda_{t+1} \right] = 0 \tag{3}$$

$$\frac{\partial \mathcal{L}}{\partial \lambda_t} = w_{t+1} - (1-\delta) w_t - \phi c_t = 0 \tag{4}$$

The non-stochastic steady-state equilibrium is characterized by the following equations:

$$\lambda = \frac{\overline{c} - c}{\phi} \tag{5}$$

$$\lambda = \beta \left[b \left(w - w^I \right) + (1-\delta) \lambda \right] \tag{6}$$

$$c = \frac{\delta w}{\phi} \tag{7}$$

Equations (5) and (6) imply

$$\frac{\overline{c} - c}{\phi} = \frac{\beta b \left(w - w^I \right)}{1 - \beta (1-\delta)}$$

Using equation (7) to eliminate c and rearranging terms,

$$\left[\left[1 - \beta (1-\delta) \right] \delta / \phi + \phi \beta b \right] w = \left[1 - \beta (1-\delta) \right] \overline{c} + \phi \beta b w^I$$

Given that $c = \frac{\delta w}{\phi}$, we define now $\overline{w} \equiv \frac{\phi \overline{c}}{\delta}$, so then

$$[[1 - \beta(1-\delta)]\delta/\phi + \phi\beta b]\, w = [1 - \beta(1-\delta)]\,(\delta\overline{w}/\phi) + \phi\beta b w^I$$

Solving for w yields the steady-state equilibrium

$$w^{ss} = \alpha_w \overline{w} + (1 - \alpha_w) w^I$$

with $\alpha_w \equiv \frac{[1-\beta(1-\delta)]\delta}{[1-\beta(1-\delta)]\delta + \phi^2\beta b}$ and $0 < \alpha_w < 1$. Put differently,

$$w^{ss} = \alpha_w(\overline{w} - w^I) + w^I.$$

Thus, if $\overline{w} > w^I$, then $w^{ss} > w^I$.

A.2 Proof of Prediction 2.

By the method of undetermined coefficients, we guess that $c_t = A + Bw_t$, with parameters A and B to be determined.

The law of motion of weight implies that

$$w_{t+1} = \phi A + (1 - \delta + \phi B)\, w_t + \epsilon_{t+1} \tag{8}$$

Plugging the guess, (8), and (2) in (3) we obtain

$$\frac{\overline{c} - (A + Bw_t)}{\phi} = \beta E_t \left[b\left[\phi A + (1 - \delta + \phi B)\, w_t + \epsilon_{t+1} - w^I\right] + (1-\delta)\left(\frac{\overline{c} - (A + Bw_{t+1})}{\phi}\right)\right]$$

Using (8) again and rearranging, we get

$$(\overline{c} - A) - Bw_t = \phi^2\beta b A - \phi\beta b w^I + \beta(1-\delta)\left[\overline{c} - A(1 + \phi B)\right] +$$

$$+\left[\phi\beta b\left(1-\delta+\phi B\right)-\beta\left(1-\delta\right)B\left(1-\delta+\phi B\right)\right]w_t$$

Therefore, we can equalize terms as follows

$$\overline{c}-A=\phi\beta b\left(\phi A-w^I\right)+\beta\left(1-\delta\right)\left[\overline{c}-A\left(1+\phi B\right)\right] \tag{9}$$

and

$$-B=\phi\beta b\left(1-\delta+\phi B\right)-\beta\left(1-\delta\right)B\left(1-\delta+\phi B\right) \tag{10}$$

The solution for A in (9) yields

$$A^*=\frac{\left[1-\beta\left(1-\delta\right)\right]\overline{c}+\phi b\beta w^I}{1-\beta\left[(1-\delta)(1+\phi B^*)-\phi^2 b\right]}$$

In addition, equation (10) can be rewritten as a quadratic form:

$$B^2-\psi B-b=0$$

with $\psi\equiv\left[\frac{1-\beta(1-\delta)^2+\phi^2\beta b}{\beta(1-\delta)\phi}\right]$ and whose solution has the form

$$B^*=(\psi\pm\sqrt{\psi^2+4b})/2$$

Given the solution for weight, in the steady state: $w^{ss}=\phi A^*+(1-\delta+\phi B^*)w^{ss}$, we have that

$$w^{ss}=\frac{\phi A^*}{\delta-\phi B^*}$$

Because $w^{ss}>0$, then either $A^*>0$ and $\delta-\phi B^*>0$ or $A^*<0$ and $\delta-\phi B^*<0$. In the second case, the law of motion of weight has a negative intercept ($\phi A^*<0$) and a slope larger than

one on the (w_t, w_{t+1}) plane. Therefore, we discard this possibility since it corresponds to an unstable equilibrium. The saddle-path equilibrium is, thus, given by $A^* > 0$ and $\delta - \phi B^* > 0$ with $B^* = (\psi - \sqrt{\psi^2 + 4b})/2 < 0$. This implies that the the law of motion of weight has a positive intercept and a slope lower than one on the (w_t, w_{t+1}) plane. In terms of equation (4) from Section 2, we obtain $\alpha_0 \equiv \phi A^* > 0$ and $\alpha_1 \equiv -\delta + \phi B^* < 0$.

A.3 Derivation of Equation (5)

We can rewrite equation (4) in period T for every country i as

$$w_{iT} = \alpha_0 + (1 + \alpha_1) w_{iT-1} + \epsilon_{iT} \tag{11}$$

By backward substitution in equation (11), we obtain

$$w_{iT} = \alpha_0 \sum_{j=0}^{T-1} (1 + \alpha_1)^j + (1 + \alpha_1)^T w_{i0} + \sum_{j=0}^{T-1} (1 + \alpha_1)^j \epsilon_{iT-j} \tag{12}$$

Under convergence, it is true that $-1 < \alpha_1 < 0$ and, hence,

$$\sum_{j=0}^{T-1} (1 + \alpha_1)^j = -\frac{[1 - (1 + \alpha_1)^T]}{\alpha_1} > 0$$

Hence, we can rewrite (12) as

$$w_{iT} = \frac{-\alpha_0 [1 - (1 + \alpha_1)^T]}{\alpha_1} + (1 + \alpha_1)^T w_{i0} + \sum_{j=0}^{T-1} (1 + \alpha_1)^j \epsilon_{iT-j} \tag{13}$$

Or, in absolute changes as

$$w_{iT} - w_{i0} = \frac{-\alpha_0 [1 - (1 + \alpha_1)^T]}{\alpha_1} + \left[(1 + \alpha_1)^T - 1\right] w_{i0} + \sum_{j=0}^{T-1} (1 + \alpha_1)^j \epsilon_{iT-j} \tag{14}$$

Dividing both sides by T, which is a constant, and defining $\gamma_0 \equiv -\alpha_0[1-(1+\alpha_1)^T]/(\alpha_1 T)$, $\gamma_1 \equiv (1/T)[(1+\alpha_1)^T - 1]$, and $\nu_{iT} \equiv (1/T)\sum_{i=0}^{T-1}(1+\alpha_1)^j \epsilon_{i,T-j}$, we obtain equation (5) from Section 4:

$$\frac{w_{iT} - w_{i0}}{T} = \gamma_0 + \gamma_1 w_{i0} + \nu_{iT}$$

In the non-stochastic steady-state equilibrium, $w_{it} = w_{it-1} = w^{ss}$, for every t including the initial and last period, and the optimal weight is

$$w^{ss} = -\frac{\gamma_0}{\gamma_1} = \frac{\alpha_0[1-(1+\alpha_1)^T]/(\alpha_1 T)}{(1/T)[(1+\alpha_1)^T - 1]} = -\frac{\alpha_0}{\alpha_1} > 0.$$

B Data

B.1 Definitions and sources

- BMI: body mass index, expressed in kilograms per square meters. Source: NCD Risk Factor Collaboration (2016). The *unadjusted data* are the dataset estimated without covariates. The *adjusted data* are the dataset estimated with covariates as reported in NCD-Risc (2016). NCD-RisC collects 1,698 population-based data sources, with more than 19.2 million adult participants (9.9 million men and 9.3 million women) in 200 countries and territories between 1975 and 2014. Each of those sources have their own sampling method. NCD-RisC aggregates, with use of a consistent protocol, the population-based survey to aggregate data at a country level. Further details about the data sources can be found in the Appendix of NCD-Risc (2016; Table 2).

- GDP per capita: Expenditure-side real GDP at chained PPPs (in mil. 2011 US$ dollars) divided by population (in mil. of persons). Source: Penn World Tables 9.0; Feenstra *et al.*, (2015).

- Human capital index: based on years of schooling and returns to education; see human capital in Penn World Tables. Source: Penn World Tables 9.0; Feenstra *et al.*, (2015).

- Urbanization rate: percentage of population living in urban areas as defined by national statistical offices. It is calculated using World Bank population estimates and urban ratios from the United Nations World Urbanization Prospects. Source: World Development Indicators (World Bank).

- Controls are constructed based on the last three series above and expressed in percent deviations from their long-run levels. The long-run component of logged PPP-adjusted real GDP per person is estimated using its corresponding Hodrick-Prescott trend. The long-run components of the logs of the human capital index and the urbanization rate are defined as their corresponding sample means over time.

- Population weights –used in the models reported in Table A1– were constructed using total population and female/male percentage of population for each country. Population series are from Penn World Tables 9.0 (Feenstra *et al.*, 2015). Female and male population percentage series are from World Bank's World Development Indicators.

B.2 Samples

The Europe sample comprises 45 economies: Albania, Armenia, Austria, Azerbaijan, Belarus, Belgium, Bosnia and Herzegovina, Bulgaria, Croatia, Cyprus, Czech Republic, Denmark, Estonia, Finland, France, Georgia, Germany, Greece, Hungary, Iceland, Ireland, Italy, Kazakhstan, Latvia, Lithuania, Luxembourg, Macedonia (TFYR), Malta, Moldova, Montenegro, Netherlands, Norway, Poland, Portugal, Romania, Russian Federation, Serbia, Slovakia, Slovenia, Spain, Sweden, Switzerland, Turkey, Ukraine, and United Kingdom. These economies come from 7 different world regions defined by the database authors (see NCD Risk Factor Collaboration, 2016). We omit Andorra and Greenland due to the absence of GDP data.

The non-Europe sample includes 127 countries and territories: Algeria, Angola, Antigua and Barbuda, Argentina, Australia, Bahamas, Bahrain, Bangladesh, Barbados, Belize, Benin, Bermuda, Bhutan, Bolivia, Botswana, Brazil, Brunei Darussalam, Burkina Faso, Burundi,

Cabo Verde, Cambodia, Cameroon, Canada, Central African Republic, Chad, Chile, China, China (Hong Kong SAR), Colombia, Comoros, Congo, Costa Rica, Cote d'Ivoire, Djibouti, Dominica, Dominican Republic, DR Congo, Ecuador, Egypt, El Salvador, Equatorial Guinea, Ethiopia, Fiji, Gabon, Gambia, Ghana, Grenada, Guatemala, Guinea, Guinea Bissau, Haiti, Honduras, India, Indonesia, Iran, Iraq, Israel, Jamaica, Japan, Jordan, Kenya, Kuwait, Kyrgyzstan, Lao PDR, Lebanon, Lesotho, Liberia, Madagascar, Malawi, Malaysia, Maldives, Mali, Mauritania, Mauritius, Mexico, Mongolia, Morocco, Mozambique, Myanmar, Namibia, Nepal, New Zealand, Nicaragua, Niger, Nigeria, Oman, Pakistan, Panama, Paraguay, Peru, Philippines, Qatar, Rwanda, Saint Kitts and Nevis, Saint Lucia, Saint Vincent and the Grenadines, Sao Tome and Principe, Saudi Arabia, Senegal, Seychelles, Sierra Leone, Singapore, South Africa, South Korea, Sri Lanka, Sudan, Suriname, Swaziland, Syrian Arab Republic, Taiwan, Tajikistan, Tanzania, Thailand, Togo, Trinidad and Tobago, Tunisia, Turkmenistan, Uganda, United Arab Emirates, United States of America, Uruguay, Uzbekistan, Venezuela, Vietnam, Yemen, Zambia, Zimbabwe.

The Mediterranean Europe sample includes 12 economies: Albania, Bosnia and Herzegovina, Croatia, Cyprus, France, Greece, Italy, Malta, Montenegro, Slovenia, Spain, and Turkey.

The European Union sample comprises 28 economies: Austria, Belgium, Bulgaria, Croatia, Cyprus, Czech Republic, Denmark, Estonia, Finland, France, Germany, Greece, Hungary, Ireland, Italy, Latvia, Lithuania, Luxembourg, Malta, Netherlands, Poland, Portugal, Romania, Slovakia, Slovenia, Spain, Sweden, and United Kingdom.

The Eastern Europe sample includes 21 economies: Albania, Armenia, Azerbaijan, Belarus, Bosnia and Herzegovina, Bulgaria, Croatia, Czech Republic, Estonia, Georgia, Hungary, Latvia, Lithuania, Moldova, Montenegro, Poland, Romania, Russian Federation, Serbia, Slovakia, and Ukraine.

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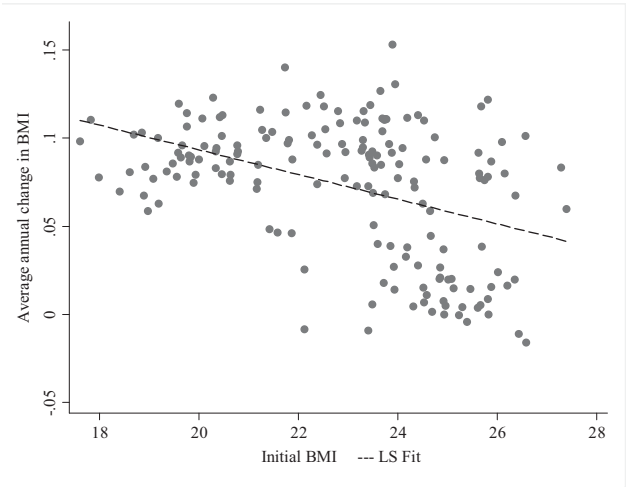
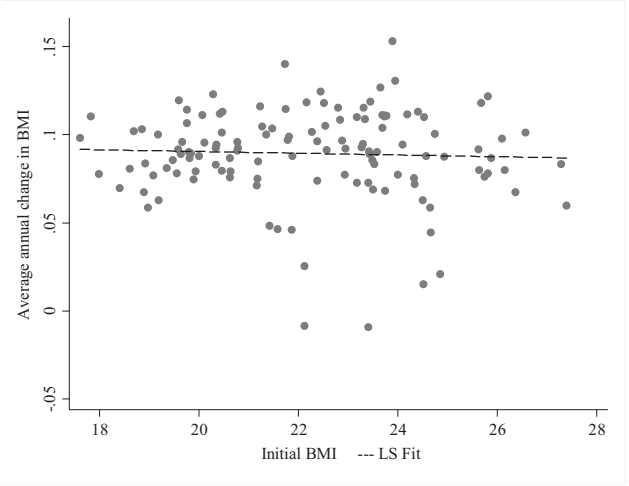
Panel A. World sample



Panel B. Non-Europe sample



Panel C. Europe sample

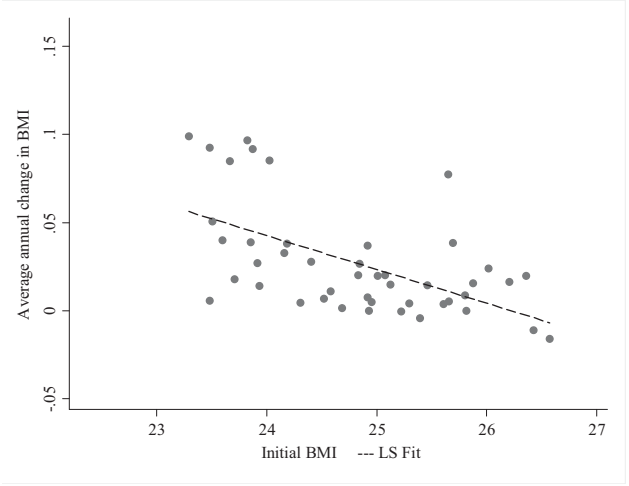
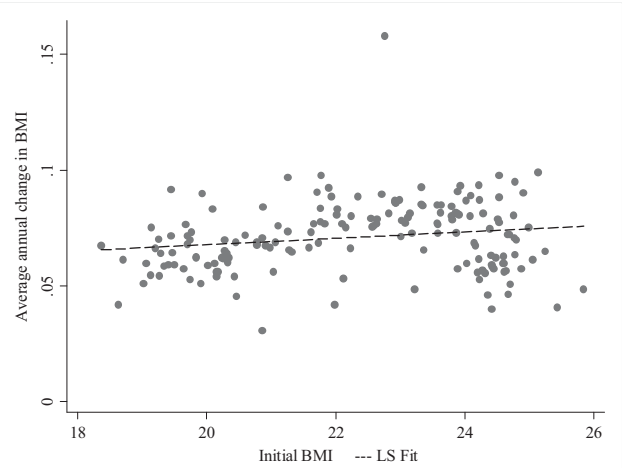


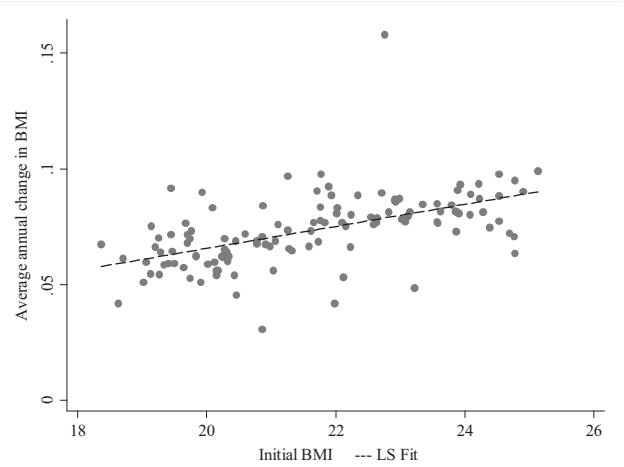
Figure 1: Average Change in Female BMI and Initial BMI.

Note: The parameters estimates and robust standard errors of each cross-sectional regression are reported in Table 2, columns (1), (3), and (5).

Panel A. World sample



Panel B. Non-Europe sample



Panel C. Europe sample

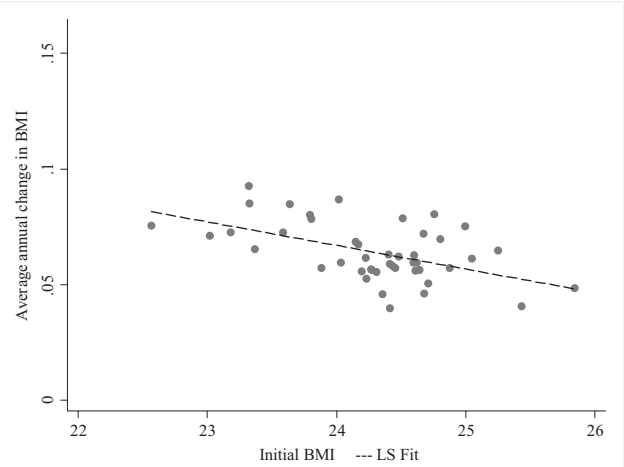


Figure 2: Average Change in Male BMI and Initial BMI.

Note: The parameters estimates and robust standard errors of each cross-sectional regression are reported in Table 2, columns (2), (4), and (6).

Table 1. Descriptive statistics

Variable	No. of observations	Mean	Standard deviation	Minimum	Median	Maximum
Body Mass Index (unadjusted data)						
World sample						
Female	6880	24.32	2.31	17.60	24.68	30.57
Male	6880	23.74	2.13	18.37	24.06	29.00
Europe sample						
Female	1800	25.31	0.87	23.29	25.24	28.66
Male	1800	25.48	0.92	22.57	25.44	27.92
Non-Europe sample						
Female	5080	23.97	2.55	17.60	23.92	30.57
Male	5080	23.13	2.10	18.37	22.94	29.00
Body Mass Index (adjusted data)						
World sample						
Female	6880	24.23	2.44	17.15	24.57	30.65
Male	6880	23.62	2.21	18.5	23.92	29.07
Europe sample						
Female	1800	25.16	0.94	22.30	25.12	28.59
Male	1800	25.39	1.01	22.42	25.34	27.82
Non-Europe sample						
Female	5080	23.90	2.70	17.15	23.84	30.65
Male	5080	23.00	2.18	18.50	22.83	29.07
Other variables						
World sample						
GDP per person (PPP 2011 US$)	6521	12537.2	17209.2	142.4	6423.71	221818.5
Human capital index	5481	2.19	0.71	1.01	2.14	3.73
Urbanization rate (%)	6825	52.05	23.61	3.52	51.59	100.00
Europe sample						
GDP per person (PPP 2011 US$)	1515	21070.4	13770.3	1251.4	19562.4	95175.7
Human capital index	1365	2.89	0.43	1.36	2.93	3.73
Urbanization rate (%)	1785	65.97	13.94	31.29	66.84	97.82
Non-Europe sample						
Real GDP per person	5006	9954.7	17312.7	142.4	4340.8	221818.5
Human capital index	4116	1.96	1.96	1.01	1.85	3.72
Urbanization rate (%)	5040	47.12	24.35	3.52	43.01	100

Notes: The world sample includes 172 countries and territories (127 non-European and 45 European countries) over the 1975-2014 period. The Europe sample includes: Albania, Armenia, Austria, Azerbaijan, Belarus, Belgium, Bosnia and Herzegovina, Bulgaria, Croatia, Cyprus, Czech Republic, Denmark, Estonia, Finland, France, Georgia, Germany, Greece, Hungary, Iceland, Ireland, Italy, Kazakhstan, Latvia, Lithuania, Luxembourg, Macedonia (TFYR), Malta, Moldova, Montenegro, Netherlands, Norway, Poland, Portugal, Romania, Russian Federation, Serbia, Slovakia, Slovenia, Spain, Sweden, Switzerland, Turkey, Ukraine, and United Kingdom. The unadjusted data are the dataset without covariates. The adjusted data are the dataset with covariates as reported in NCD-Risc (2016). GDP per person is real GDP at chained PPP (in 2011 US$ dollars). Human capital index is based on years of schooling and returns to education. Real GDP and human capital series are from Penn World Tables 9.0 (Feenstra et al., 2015). Urbanization rate series are from World Bank's World Development Indicators. For further details, see Appendix B.

Table 2. Convergence in BMIs, cross-sectional results
Dependent variable: Average annual change in BMI

	World sample		Non-Europe sample		Europe sample	
	Female	**Male**	**Female**	**Male**	**Female**	**Male**
	(1)	**(2)**	**(3)**	**(4)**	**(5)**	**(6)**
Constant	0.233 ***	0.040 ***	0.100 ***	-0.030 **	0.502 ***	0.311 ***
	(0.023)	(0.012)	(0.017)	(0.012)	(0.121)	(0.057)
Initial BMI	-0.0070 ***	0.0014 **	-0.0005	0.0048 ***	-0.0192 ***	-0.0102 ***
	(0.0011)	(0.0005)	(0.0008)	(0.0006)	(0.0048)	(0.0024)
Initial year	1975	1975	1975	1975	1975	1975
Controls	No	No	No	No	No	No
R-squared	0.178	0.031	0.002	0.187	0.326	0.276
No. of observations	172	172	127	127	45	45
Additional estimates and statistics						
Convergence	Yes	No	No	No	Yes	Yes
Steady-state BMI	33.32	…	…	…	26.22	30.57
95%-Confidence interval	[29.7 36.9]	…	…	…	[25.6 26.9]	[27.6 33.5]
P-value (Ho: SS BMI<=25)	0.000	…	…	…	0.000	0.000
P-value (Ho: no convergence)	0.000	0.994	0.274	1.000	0.000	0.000
Half life (years)	98.6	…	…	…	35.8	67.7

Notes: ∗p< 0.1, ∗∗p< 0.05, ∗∗∗p< 0.01, for the null hypothesis that the constant/slope equals zero. The dependent variable is (BMI(T)-BMI(0))/T, where 0 and T denote the initial and last year of each sample. Bootstrapped standard errors are reported below each LS coefficient estimate and calculated using 2000 replications (columns 1-8). P-value (Ho: SS BMI<=25) refers to the null that the steady-state BMI is equal to or lower than 25 (healthy level). P-value (Ho: no convergence) is related to the null that the slope of the initial BMI is equal to or higher than 0. If this p-value is lower than 0.05, we report "Yes" in the Convergence line. The steady-state BMI is calculated as the constant divided by the negative of the coefficient of the initial BMI. The half-life is estimated as ln(0.5)/ln(1+coefficient of initial BMI).

Table 3. Convergence in BMIs, European cross-sectional results -- robustness checks
Dependent variable: Average annual change in BMI

	Robust LS		LS		LS		LS, adjusted data	
	Female	**Male**	**Female**	**Male**	**Female**	**Male**	**Female**	**Male**
	(1)	**(2)**	**(3)**	**(4)**	**(5)**	**(6)**	**(7)**	**(8)**
Constant	0.435 ***	0.307 ***	0.424 ***	0.286 ***	0.417 *	0.280 **	0.438 ***	0.361 ***
	(0.127)	(0.052)	(0.144)	(0.063)	(0.232)	(0.119)	(0.105)	(0.052)
Initial BMI	-0.0166 ***	-0.0100 ***	-0.0159 ***	-0.0090 ***	-0.0167 *	-0.0089 *	-0.0163 ***	-0.0120 ***
	(0.0050)	(0.0022)	(0.0057)	(0.0026)	(0.0099)	(0.0050)	(0.0043)	(0.0022)
Initial year	1975	1975	1980	1980	1975	1975	1975	1975
Controls	No	No	No	No	Yes	Yes	No	No
P-value significance of controls	…	…	…	…	0.653	0.940	…	…
R-squared	0.319	0.314	0.186	0.190	0.174	0.167	0.251	0.318
No. of observations	45	45	45	45	26	26	45	45
Additional estimates and statistics								
Convergence	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Steady-state BMI	26.22	30.62	26.66	31.76	25.03	31.50	26.80	30.02
95%-Confidence interval	[25.6 26.9]	[27.9 33.4]	[25.5 27.9]	[27.6 35.9]	[22.3 27.8]	[22.8 40.2]	[25.4 28.1]	[27.8 32.2]
P-value (Ho: SS BMI<=25)	0.000	0.000	0.003	0.001	0.490	0.067	0.004	0.000
P-value (Ho: no convergence)	0.001	0.000	0.003	0.000	0.046	0.037	0.000	0.000
Half life (years)	41.4	68.9	43.3	76.6	41.2	77.6	42.1	57.3

Notes: *p< 0.1, **p< 0.05, ***p< 0.01, for the null hypothesis that the constant/slope equals zero. The dependent variable is (BMI(T)-BMI(0))/T, where 0 and T denote the initial and last year of each sample. The robust LS is an M-estimator with a Huber objective function. Robust standard errors are reported below each robust LS coefficient estimate (columns 1-2). Bootstrapped standard errors are reported below each LS coefficient estimate and calculated using 2000 replications (columns 3-8). The p-value of significance of controls refers to the joint null that the slopes of the controls (real GDP per capita, a human capital index, and urbanization rate, all expressed in deviations from long-run values) are equal to zero. P-value (Ho: SS BMI<=25) refers to the null that the steady-state BMI is equal to or higher than 25 (healthy level). P-value (Ho: no convergence) is related to the null that the slope of the initial BMI is equal to or lower than 0. If this p-value is lower than 0.05, we report "Yes" in the Convergence line. The steady-state BMI is calculated as the constant divided by the negative of the coefficient of the initial BMI. The half-life is estimated as ln(0.5)/ln(1+coefficient of initial BMI). The adjusted data is the dataset estimated with covariates as reported in NCD-Risc (2016).

Table 4. Convergence in BMIs, panel data results
Dependent variable: Annual Change in BMI

	World sample		Non-Europe sample		Europe sample	
	Female	Male	Female	Male	Female	Male
	(1)	(2)	(3)	(4)	(5)	(6)
Constant	0.117 ***	-0.397 ***	-0.013	-0.871 ***	0.178 ***	0.432 ***
	(0.010)	(0.027)	(0.010)	(0.020)	(0.016)	(0.058)
Lagged BMI	-0.0020 ***	0.0211 ***	0.0046 ***	0.0438 ***	-0.0063 ***	-0.0153 ***
	(0.0005)	(0.0012)	(0.0005)	(0.001)	(0.0007)	(0.0024)
Year effects	Yes	Yes	Yes	Yes	Yes	Yes
No. of countries	172	172	127	127	45	45
No. of observations	6880	6880	5080	5080	1800	1800
Balanced panel	Yes	Yes	Yes	Yes	Yes	Yes
Additional estimates and statistics						
Convergence	Yes	No	No	No	Yes	Yes
Steady-state BMI	58.87	…	…	…	28.33	28.27
95%-Confidence Interval	[51.4 121.8]	…	…	…	[27.8 29.1]	[27.9 32.5]
P-value (Ho: SS BMI<=25)	0.000	…	…	…	0.000	0.000
P-value (Ho: no convergence)	0.000	1.000	1.000	1.000	0.000	0.004
Half life (years)	347.9	…	…	…	109.9	45.0

Notes: *p< 0.1, **p< 0.05, ***p< 0.01, for the null hypothesis that the constant/slope equals zero. Robust standard errors are reported below each parameter estimate. All the regression models include fixed country effects. LSDVC is biased-corrected fixed effects estimator, based on an analytical approach (Kiviet (1995), Bruno (2005)). We use a third-level accuracy to approximate the bias and system GMM as the initial consistent estimator. The steady-state BMI is calculated as the constant divided by the negative of the coefficient of the lagged BMI. P-values, standard errors, and confidence intervals are calculated using 2000 bootstrap replications. P-value (Ho: SS BMI<=25) refers to the null that the steady-state BMI is equal to or lower than 25 (healthy level). P-value (Ho: no convergence) is related to the null that the slope of the lagged BMI is equal to or higher than 0. If this p-value is lower than 0.05, we report "Yes" in the Convergence line. The half-life is estimated as ln(0.5)/ln(1+coefficient of lagged BMI).

Table 5. Convergence in BMIs, European panel data results -- robustness checks
Dependent variable: Annual Change in BMI

	Europe - Full sample		Europe - 1980-2014 sample		Adjusted BMI data	
	Female	Male	Female	Male	Female	Male
	(1)	(2)	(3)	(4)	(5)	(6)
Constant	0.257 ***	0.384 ***	0.223 ***	0.537 ***	0.475 ***	0.425 ***
	(0.017)	(0.004)	(0.024)	(0.072)	(0.041)	(0.049)
Lagged BMI	-0.0093 ***	-0.0129 ***	-0.0080 ***	-0.0194 ***	-0.0174 ***	-0.0138 ***
	(0.0007)	(0.0025)	(0.0006)	(0.0029)	(0.0016)	(0.0019)
Year effects	Yes	Yes	Yes	Yes	No	No
Controls	Yes	Yes	No	No	Yes	Yes
Statistical significance	Yes	Yes	…	…	Yes	Yes
No. of countries	39	39	45	45	39	39
No. of periods (average)	35	35	35	35	35	35
No. of observations	1365	1365	1575	1575	1365	1365
Balanced panel	No	No	Yes	Yes	No	No
Additional estimates and statistics						
Convergence	Yes	Yes	Yes	Yes	Yes	Yes
Steady-state BMI	27.76	29.89	27.81	27.71	27.33	30.79
95%-Confidence Interval	[27.3 28.4]	[28.5 34.6]	[27.4 28.3]	[27.7 31.8]	[27.0 27.9]	[29.6 32.9]
P-value (Ho: SS BMI<=25)	0.000	0.000	0.000	0.007	0.000	0.001
P-value (Ho: no convergence)	0.000	0.000	0.000	0.000	0.000	0.000
Half life (years)	74.5	53.6	86.0	35.4	39.5	49.9

Notes: ∗p< 0.1, ∗∗p< 0.05, ∗∗∗p< 0.01, for the null hypothesis that the constant/slope equals zero. Robust standard errors are reported below each parameter estimate. All the regression models include fixed country effects. LSDVC is biased-corrected fixed effects estimator, based on an analytical approach (Kiviet (1995), Bruno (2005)). We use a third-level accuracy to approximate the bias and system GMM as the initial consistent estimator. The steady-state BMI is calculated as the constant divided by the negative of the coefficient of the lagged BMI. P-values, standard errors, and confidence intervals are calculated using 2000 bootstrap replications. P-value (Ho: SS BMI<=25) refers to the null that the steady-state BMI is equal to or lower than 25 (healthy level). P-value (Ho: no convergence) is related to the null that the slope of the lagged BMI is equal to or higher than 0. If this p-value is lower than 0.05, we report "Yes" in the Convergence line. The half-life is estimated as ln(0.5)/ln(1+coefficient of lagged BMI). The adjusted data is the dataset estimated with covariates as reported in NCD-Risc (2016).

Table A1. Convergence in BMIs, European panel data results using population weights
Dependent variable: Annual Change in BMI

	LSDVC with Population Weights	
	Female	**Male**
	(1)	**(2)**
Constant	0.328 ***	0.715 **
	(0.064)	(0.277)
Lagged BMI	-0.0124 ***	-0.0270 **
	(0.003)	(0.011)
No. of countries	45	45
No. of observations	1755	1755
Additional estimates and statistics		
Convergence	Yes	Yes
Steady-state BMI	26.46	26.46
95%-Confidence Interval	[25.4 28.2]	[25.1 36.7]
P-value (Ho: no convergence)	0.000	0.010
Half life (years)	55.5	25.3

Notes: *p< 0.1, **p< 0.05, ***p< 0.01, for the null hypothesis that the constant/slope equals zero. Bootstrapped standard errors are reported below each parameter estimate. All the regression models include fixed country effects and time effects. LSDVC is biased-corrected fixed effects estimator, based on an analytical approach (Kiviet (1995), Bruno (2005)). We use a third-level accuracy to approximate the bias and system GMM as the initial consistent estimator. The steady-state BMI is calculated as the constant divided by the negative of the coefficient of the lagged BMI. P-values, standard errors, and confidence intervals are calculated using 2000 bootstrap replications. P-value (Ho: no convergence) is related to the null that the slope of the lagged BMI is equal to or higher than 0. If this p-value is lower than 0.05, we report "Yes" in the Convergence line. The half-life is estimated as ln(0.5)/ln(1+coefficient of lagged BMI). Population weights were constructed using total population and female/male percentage of population for each country. Population series are from Penn World Tables 9.0 (Feenstra et al., 2015). Female and male population percentage series are from World Bank's World Development Indicators.

Table A2. Convergence in BMIs, European panel data results -- Extensions
Dependent variable: Annual Change in BMI

	Mediterranean Europe		Non-Mediterranean Europe		European Union		non-European Union		Eastern Europe		Non-Eastern Europe	
	Female	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female	Male
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
Constant	0.226 ***	0.356 ***	0.189 ***	0.508 ***	0.323 ***	0.285 ***	0.116 ***	0.749 ***	0.073 ***	0.593 ***	0.202 ***	0.175 ***
	(0.023)	(0.082)	(0.015)	(0.064)	(0.018)	(0.068)	(0.015)	(0.093)	(0.017)	(0.082)	(0.016)	(0.025)
Lagged BMI	-0.0078 ***	-0.0122 ***	-0.0062 ***	-0.0164 ***	-0.0118 ***	-0.0094 ***	-0.0035 ***	-0.0255 ***	-0.0025 ***	-0.0220 ***	-0.0068 ***	-0.0049 ***
	(0.0009)	(0.0034)	(0.0006)	(0.0024)	(0.001)	(0.003)	(0.001)	(0.004)	(0.001)	(0.003)	(0.001)	(0.001)
No. of countries	12	12	33	33	28	28	17	17	21	21	24	24
No. of observations	468	468	1287	1287	1092	1092	663	663	819	819	936	936
Balanced panel	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Additional estimates and statistics												
Convergence	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Steady-state BMI	28.94	29.26	30.72	30.91	27.39	30.21	33.03	29.36	29.51	26.94	29.88	36.10
95%-Confidence Interval	[28.4 29.9]	[27.8 38.2]	[29.9 32.0]	[30.5 35.0]	[27.1 27.7]	[28.3 42.3]	[31.2 38.0]	[29.1 32.0]	[28.0 34.9]	[26.8 30.3]	[29.2 30.9]	[32.0 47.6]
P-value (Ho: SS BMI<=25)	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
P-value (Ho: no convergence)	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.001
Half life (years)	88.3	56.5	112.2	41.8	58.3	73.0	197.7	26.8	279.6	31.1	102.0	142.4

Notes: *p< 0.1, **p< 0.05, ***p< 0.01, for the null hypothesis that the constant/slope equals zero. Bootstrapped standard errors are reported below each parameter estimate. All the regression models include fixed country effects and time effects or a trend component such as log trend or quadratic trend. LSDVC is biased-corrected fixed effects estimator, based on an analytical approach (Kiviet (1995), Bruno (2005)). We use a third-level accuracy to approximate the bias and system GMM as the initial consistent estimator. The steady-state BMI is calculated as the constant divided by the negative of the coefficient of the lagged BMI. P-values, standard errors, and confidence intervals are calculated using 2000 bootstrap replications. P-value (Ho: SS BMI<=25) refers to the null that the steady-state BMI is equal to or lower than 25 (healthy level). P-value (Ho: no convergence) is related to the null that the slope of the lagged BMI is equal to or higher than 0. If this p-value is lower than 0.05, we report "Yes" in the Convergence line. The half-life is estimated as ln(0.5)/ln(1+coefficient of lagged BMI). Mediterranean Europe includes Albania, Bosnia and Herzegovina, Croatia, Cyprus, France, Greece, Italy, Malta, Montenegro, Slovenia, Spain, and Turkey. The European Union (EU) comprises Austria, Belgium, Bulgaria, Croatia, Cyprus, Czech Republic, Denmark, Estonia, Finland, France, Germany, Greece, Hungary, Ireland, Italy, Latvia, Lithuania, Luxembourg, Malta, Netherlands, Poland, Portugal, Romania, Slovakia, Slovenia, Spain, Sweden, and United Kingdom. Eastern Europe includes Albania, Armenia, Azerbaijan, Belarus, Bosnia and Herzegovina, Bulgaria, Croatia, Czech Republic, Estonia, Georgia, Hungary, Latvia, Lithuania, Moldova, Montenegro, Poland, Romania, Russian Federation, Serbia, Slovakia, and Ukraine.