# The contribution of urbanization to non-communicable diseases: evidence from 173 countries from 1980 to 2008

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## Abstract

It is widely believed that the expanding burden of non-communicable diseases (NCDs) is in no small part the result of major macro-level determinants. We use a large amount of new data, to explore in particular the role played by urbanization – the process of the population shifting from rural to urban areas within countries – in affecting four important drivers of NCDs world-wide: diabetes prevalence, as well as average body mass index (BMI), total cholesterol level and systolic blood pressure. Urbanization is seen by many as a double-edged sword: while its beneficial economic effects are widely acknowledged, it is commonly alleged to produce adverse side effects for NCD-related health outcomes. In this paper we submit this hypothesis to extensive empirical scrutiny, covering a global set of countries from 1980-2008, and applying a range of estimation procedures. Our results indicate that urbanization appears to have contributed to an increase in average BMI and cholesterol levels: the implied difference in average total cholesterol between the most and the least urbanized countries is 0.40 mmol/L, while people living in the least urbanized countries are also expected to have an up to 2.3 kg/m$^2$ lower BMI than in the most urbanized ones. Moreover, the least urbanized countries are expected to have an up to 3.2 p.p. lower prevalence of diabetes among women. This association is also much stronger in the low and middle-income countries, and is likely to be mediated by energy intake-related variables, such as calorie and fat supply per capita.



Keywords: Non-communicable diseases; hypertension; cholesterol; diabetes; BMI; urbanization.

## 1. Introduction

There is abundant evidence on the substantial disease burden caused by non-communicable diseases (NCDs) and risk factors (Danaei et al., 2006; Ezzati et al., 2002; S Lewington et al., 2002; Sarah Lewington et al., 2007), not only in high- but also in low- and middle-income countries (LMICs). According to recent WHO estimates, NCDs were responsible for 68% of the world's deaths in 2012, with more than 40% of those considered as 'premature', i.e. occurring before the age of 70. Almost three quarters of all NCD deaths and the vast majority (82%) of premature deaths occur in LMICs (WHO, 2014). NCDs and related risk factors, including hypertension, obesity, hypercholesterolemia and diabetes may also lead to complications impairing people's ability to live active and productive lives (Suhrcke et al., 2006).

It is widely believed that the expanding burden of NCDs is in no small part the result of major macro-level drivers (Hanefeld, 2015). In this paper we make use of a large amount of new data to explore in particular the role played by urbanization – the process of the population shifting from rural to urban areas within countries (Allender et al., 2008). Urbanization is a factor that is commonly viewed as double-edged sword: while otherwise known to promote higher living standards (Cochrane, 1983), or economic growth – at least up to a point (Henderson, 2003)– some public health researchers and advocates have emphasised the allegedly negative side-effects that urbanization may have in terms of enhancing the prevalence of NCD-related risk factors (Allender et al., 2011). Should these claims stand up to further empirical scrutiny – which is what we seek to provide here – then there may be a more informed case for policy to seek ways to confront the challenge of harnessing the benefits of this major macro process while at the same time avoiding its potential collateral damage.

Why might urbanization contribute to the spread of NCD-related risk factors? First, urbanization may be related to the nutritional transition towards diets high in saturated fats, sugar and calories (Popkin, 1999), thereby contributing to the spread of obesity, diabetes and high blood pressure. Such diets can also contribute to higher total blood cholesterol levels (Howell et al., 1997; Sacks & Katan, 2002; Schaefer et al., 2009). Second, urbanization may be related to the reduction in energy expenditure because of the structural economic transition from agriculture to less physically demanding service employment (Popkin, 1999). Urbanization may also lead to less energy expenditure for a number of other reasons, for example because of greater car use and lower level of physical exercise (Monda et al., 2007).

Owing to the efforts of the Global Burden of Metabolic Risk Factors of the Chronic Diseases Collaborating Group, we are now in a position to explore quantitatively how four major NCD-related risk factors – average body mass index (BMI), average total cholesterol (TC), mean systolic blood pressure (SBP) and the prevalence of diabetes – respond to urbanization in a large, global set of countries, using annual data from 1980 to 2008, separately by gender. We also explore two potential pathways through which urbanization may be linked to these risk factors.

The contribution of our paper is in bringing together several unique datasets, in order to rigorously examine, for the first time to our knowledge, the role of urbanization (defined as the proportion of people living in urban areas, as per national statistical offices), using data from 173 countries, spanning 29 years (1980-2008). Crucially, the longitudinal nature of the data makes it possible to control for country fixed effects, reinforcing the causal interpretation of our estimates.

## 2. Study data and methods

### *2.1 Data Source and Measures*

We combine data from several sources, for the period 1980-2008, over which our outcome variables of interest are available. Data on NCD risk factors are taken from the Global Burden of Metabolic Risk Factors Project of the Chronic Diseases Collaborating Group at Imperial College London (2014). In particular we use age-standardized country-level average BMI, TC and SBP levels and diabetes prevalence, for adults aged 20 years and older. Briefly, the data have been estimated on the basis of a large number of surveys, articles and epidemiological studies (Danaei et al., 2011a; Danaei et al., 2011b; Finucane et al., 2011). Although as much effort as possible was made to obtain the actual estimates from the epidemiological literature, in a minority of cases the data was unavailable. Such missing data was modelled as a function of time-varying, country-level economic, demographic and epidemiological characteristics. Furthermore, in order to reduce the impact of short-term fluctuations in the predictor variables over time, their weighted averages were used, with decreasing weights for observation more distant in the past. The prediction Bayesian models were fit using Markov Chain Monte Carlo algorithm.

This dataset was recently used to study, for example, the association between market deregulation, fast food consumption and BMI (De Vogli et al., 2014a), and to estimate the "ideal" GDP per capita level at which the economic activity is sustainable in terms of $CO_2$ emissions, and healthy BMI levels (Egger et al., 2012). The data was also recently used to estimate population-level associations between nutrient intake and average BMI, glucose and cholesterol levels (Dave et al., 2016). Using these data, Doytch et al (2016) found that both caloric intake and physical inactivity can partly explain the impact of GDP per capita and labour force participation on average BMI.

Data on the proportion of people living in urban areas (i.e. the urbanization rate) are from the World Bank's World Development Indicators (WDI)[1]. This data is originally collected by the national statistical offices, but is further smoothed by the United Nations Populations Division. Specifically, "percentages urban are the numbers of persons residing in an area defined as "urban" per 100 total population"[2]. Additional control variables are from the WDI (population growth rate (annual, %); population density (per square km of land area); services value added (as a % of GDP); logarithm of the GDP per capita (constant 2005 US$); proportion of the population aged 16-64 years; proportion of the population who are female), FAOSTAT[3] (food supply, kcal/capita/day; fat supply, g/capita/day).

### *2.2 Analytic Methods*

In our empirical analysis we include control variables that account for countries' differential propensities to urbanize, and which also influence NCD risk factors and conditions. First, we control for both the population growth rate and population density, as they may be associated with both urbanization (Canning, 2011) and average population health (Hinrichsen & Robey, 2000). We also control for two other potential correlates of urbanization- proportion of population who are female, as well as proportion of population of working age (aged 15-64 years). We also control for the level of economic development, either by splitting countries into income groups (as described below), or by controlling for the gross domestic product (GDP) per capita in the pooled models. In addition, following Glaeser (2014), we control for agricultural productivity measured as cereal yield in kilograms per hectare times hectares per capita, as this was shown to be a driver of urbanization, both theoretically and empirically (Glaeser, 2014). Second, we account for all relevant time-invariant, country-level factors that may affect both NCDs and urbanization, such as - among

[1] http://data.worldbank.org/data-catalog/world-development-indicators
[2] http://data.worldbank.org/indicator/SP.URB.TOTL.IN.ZS
[3] http://faostat.fao.org/

others - geographical and institutional characteristics, by controlling for country fixed effects. Third, we include a general time trend, as well as ten region-specific[4] time trends to control for any common regional factors that may be changing over time. Forth, to account for the possibility that the outcome variable may simultaneously affect the independent variable of interest, we lag urbanization by one period in all specifications.

More formally, our main equation is as follows:

$$Y_{it}^{j} = \alpha + \beta_1 X_{it-1} + \mathbf{Z_{it}}'\beta_2 + \alpha_i + \varepsilon_{it} \qquad (1)$$

Where $Y_{it}^{j}$ is one of the four outcome variables *j* associated with country *i* at time *t*; $X_{it-1}$ is lagged urbanization; $\mathbf{Z_{it}}$ is the vector of control variables as described above, including general and region-specific time trends, with the associated parameter vector $\beta_2$; $\alpha_i$ are country fixed effects, possibly correlated with X and **Z**, and $\varepsilon_{it}$ is an error term. Due to the fact that $\varepsilon_{it}$ might exhibit serial correlation, even conditional on time trends, we estimate cluster-robust standard errors (clustered at the country level).

We estimate model (1) by means of a panel fixed-effect estimator that is consistent (although not necessarily the most efficient), conditional on the assumption of strict exogeneity of $\varepsilon_{it}$. Our main focus is on the urbanization parameter $\beta_1$.

In the analysis by income level, countries are split into two groups: 1) high income countries (HICs), and 2) low and middle income countries (LMICs), using year-specific thresholds applied to Gross National Income per capita (current Atlas US$), estimated by the World Bank Atlas method[5].

Urbanization does not only influence lifestyles, but also supports economic development and accompanies the structural change from agriculture to manufacturing. For

[4] The regions are: Sub-Saharan Africa; Latin America and Caribbean; East Asia; Mediterranean and North Africa; Eastern and Southern Europe; Former Soviet Union; North America; Pacific; South Asia; Western Europe.

[5] http://data.worldbank.org/about/country-and-lending-groups

most – though by no means for all – individuals the latter imply a significant increase in income that, in turn, stimulates greater investment in education. Since income and education have been shown to be key determinants of health (Pritchett & Summers, 1996), it turns out that urbanization can influence health both directly, e.g. by means of its influence on lifestyles, and indirectly, via its effect on economic development, as well as through a range of other potential channels. In this paper we estimate both the total effect of urbanization (i.e. the combination of the direct and the indirect effects), as well as account for its impact through two potential mediators, trying to shed light on the current debate on the relative importance of energy-expending vs energy-consuming factors in explaining the growing obesity burden around the world (Cutler et al., 2003; Finkelstein et al., 2005). Specifically, as urbanization may be linked with health through its effect on diets (Popkin, 1999), we estimate the effect of urbanization on total calorie supply per capita, as well as on fat supply per capita.

## 3. Results

### *3.1 Descriptive statistics*

We start by describing trends in the NCD risk factors/conditions of interest, by geographical regions (with regional averages weighted by country population size)[6]. Table 1 shows that average TC levels have been decreasing in almost all regions (except East Asia), if to varying degrees. Over most of the observation period, Western Europe and North America had the highest and SSA the lowest level, as expected. Similarly, in most regions, especially those composed of mostly richer countries, mean SBP has declined, with the notable exception of Africa and South Asia. In the late 1990s, the SBP trend flattened out in the former Soviet Union (FSU), following a period of sustained decline. In contrast, diabetes

[6] We also show a set of extended descriptive statistics in the Annex, Table A1.

prevalence has increased in all regions throughout the observation period. North America and Middle East and North Africa (MENA) had the highest mean prevalence, while SSA and East Asia displayed lower levels. Western Europe had the lowest diabetes prevalence of all regions.

On the other hand, average BMI has been increasing in all regions, if to varying degrees. By the mid-1990s, North America had already reached the highest average BMI value, although the Middle East and North Africa (MENA) region has been catching up gradually. Finally, the proportion of people living in urban areas increased in all but two regions – the FSU and the Pacific. East Asia stands out as the region with the fastest urbanization growth rate.

Table 1: Trends in outcome and main explanatory variables, by region

| | 1980 | 1985 | 1990 | 1995 | 2000 | 2005 |
|---|---|---|---|---|---|---|
| | | | *Mean total cholesterol, mmol/l* | | | |
| SSA | 4.48 | 4.38 | 4.29 | 4.21 | 4.17 | 4.16 |
| Americas | 4.95 | 4.91 | 4.87 | 4.86 | 4.85 | 4.80 |
| East Asia | 4.40 | 4.38 | 4.39 | 4.43 | 4.50 | 4.58 |
| MENA | 4.93 | 4.91 | 4.88 | 4.86 | 4.86 | 4.84 |
| ESA | 5.23 | 5.20 | 5.17 | 5.10 | 5.04 | 4.97 |
| Former Soviet Union | 5.54 | 5.44 | 5.33 | 5.13 | 4.95 | 4.87 |
| North America | 5.52 | 5.43 | 5.35 | 5.27 | 5.21 | 5.14 |
| Pacific | 5.56 | 5.47 | 5.38 | 5.29 | 5.19 | 5.09 |
| South Asia | 4.57 | 4.57 | 4.56 | 4.54 | 4.51 | 4.45 |
| Western Europe | 5.87 | 5.77 | 5.68 | 5.59 | 5.48 | 5.39 |
| | | | *Mean SBP, mmHg* | | | |
| SSA | 129.3 | 129.0 | 129.1 | 129.3 | 129.8 | 131.1 |
| Americas | 131.6 | 130.3 | 129.0 | 127.5 | 126.7 | 126.8 |
| East Asia | 126.7 | 124.9 | 124.2 | 124.1 | 124.5 | 125.3 |
| MENA | 129.1 | 128.3 | 127.5 | 126.9 | 126.8 | 127.0 |
| ESA | 133.1 | 132.6 | 132.1 | 130.8 | 129.8 | 129.1 |
| Former Soviet Union | 134.8 | 134.5 | 133.4 | 131.4 | 130.3 | 130.6 |
| North America | 128.4 | 126.6 | 125.2 | 123.8 | 122.8 | 122.1 |
| Pacific | 128.5 | 127.3 | 126.7 | 125.9 | 124.9 | 123.5 |
| South Asia | 121.9 | 123.0 | 124.1 | 124.6 | 124.8 | 124.6 |
| Western Europe | 135.8 | 133.8 | 132.8 | 131.8 | 130.1 | 128.3 |
| | | | *Diabetes, %* | | | |
| SSA | 7.3 | 7.3 | 7.5 | 7.8 | 8.1 | 8.4 |
| Americas | 7.7 | 7.8 | 8.1 | 8.5 | 8.8 | 9.4 |
| East Asia | 8.5 | 8.0 | 7.6 | 7.7 | 8.0 | 8.3 |
| MENA | 8.8 | 9.2 | 9.7 | 10.3 | 10.9 | 11.1 |
| ESA | 8.5 | 8.7 | 9.0 | 9.1 | 9.3 | 9.3 |
| Former Soviet Union | 9.8 | 9.7 | 9.7 | 9.7 | 9.9 | 10.3 |
| North America | 6.4 | 7.1 | 8.1 | 8.9 | 9.8 | 10.8 |
| Pacific | 6.2 | 6.4 | 6.8 | 7.6 | 8.5 | 9.5 |

| | | | | | | |
|---|---|---|---|---|---|---|
| South Asia | 7.6 | 7.8 | 8.1 | 8.5 | 9.6 | 10.5 |
| Western Europe | 6.5 | 6.2 | 6.2 | 6.4 | 6.5 | 6.9 |
| | | | *Mean BMI, kg/m$^2$* | | | |
| SSA | 21.07 | 21.26 | 21.48 | 21.74 | 22.05 | 22.45 |
| Americas | 23.59 | 23.98 | 24.39 | 24.85 | 25.34 | 25.84 |
| East Asia | 21.37 | 21.53 | 21.74 | 22.04 | 22.36 | 22.61 |
| MENA | 24.18 | 24.70 | 25.13 | 25.61 | 26.19 | 26.75 |
| ESA | 25.25 | 25.39 | 25.61 | 25.71 | 26.00 | 26.37 |
| Former Soviet Union | 25.65 | 25.64 | 25.63 | 25.50 | 25.44 | 25.85 |
| North America | 25.05 | 25.67 | 26.25 | 26.86 | 27.41 | 27.96 |
| Pacific | 23.79 | 24.30 | 24.89 | 25.47 | 25.99 | 26.53 |
| South Asia | 20.75 | 20.69 | 20.68 | 20.71 | 20.96 | 21.14 |
| Western Europe | 24.82 | 24.97 | 25.20 | 25.46 | 25.74 | 26.03 |
| | | | *Urban, %* | | | |
| SSA | 22.3 | 24.6 | 27.1 | 29.1 | 30.9 | 33.0 |
| Americas | 63.8 | 67.2 | 70.3 | 72.9 | 75.4 | 77.1 |
| East Asia | 26.6 | 29.7 | 32.9 | 36.6 | 40.6 | 46.0 |
| MENA | 49.6 | 52.6 | 54.8 | 56.6 | 58.5 | 60.6 |
| ESA | 53.3 | 56.9 | 59.6 | 60.9 | 61.9 | 63.4 |
| Former Soviet Union | 62.7 | 64.6 | 65.6 | 65.0 | 64.6 | 64.3 |
| North America | 72.3 | 73.4 | 74.5 | 76.3 | 78.0 | 79.0 |
| Pacific | 70.0 | 69.3 | 69.2 | 69.1 | 69.1 | 69.1 |
| South Asia | 22.3 | 23.7 | 25.0 | 26.2 | 27.4 | 29.1 |
| Western Europe | 72.1 | 72.6 | 73.2 | 73.9 | 74.5 | 75.5 |

Note: for each country, an average value for each of our three outcome variables is estimated for total population (taking into account information on % of males/females in the population), which is then averaged to give the NCD risk factor prevalence by region, weighted by country population sizes. SSA: Sub-Saharan Africa; ESA: Eastern and Southern Europe; MENA: Middle East and North Africa.

The next set of figures provides an initial glance at the shape of the bivariate relationships between urbanization and each of the outcome variables by income group, as well as showing how this relationship has changed over time. What is quite striking is that in figures 1 and 2, and to a smaller extent in figure 3, the relationship between urbanization and the NCD risk factors has been clearly positive in LMICs. On the other hand, the direction of the association was much less pronounced in the sample of HICs. This was not the case for SBP- the association was equally ambiguous in both HICs and LMICs (Figure 4). Also, average BMI levels and diabetes prevalence have tended to increase over time for a given urbanization rate, although this again was generally more true in the LMICs sample.

Figure 1. Average BMI (kg/m$^2$) by % living in urban areas, time period and income group

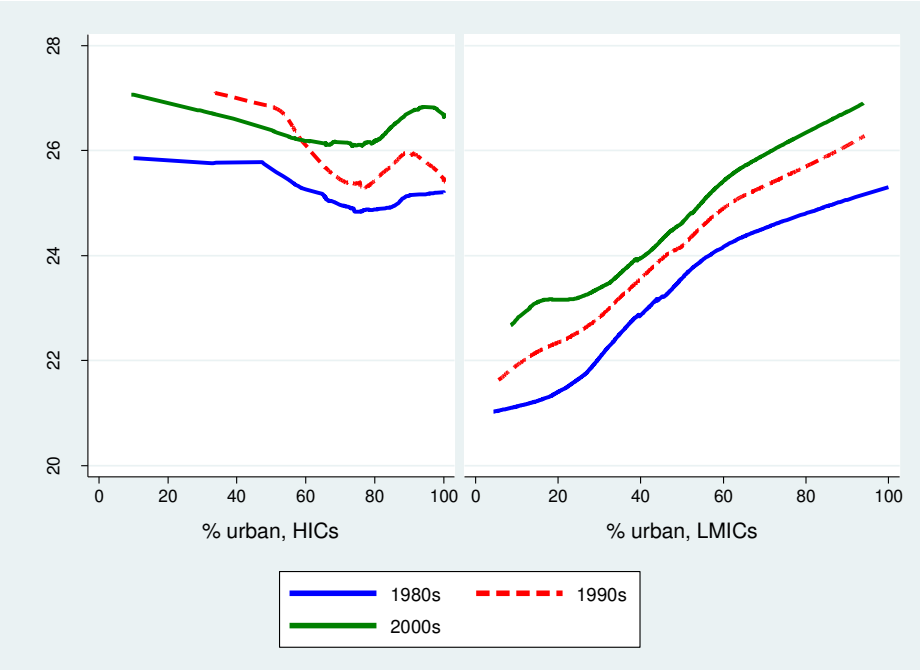



Figure 2. Average total cholesterol (mmol/l) by % living in urban areas, time period and income group

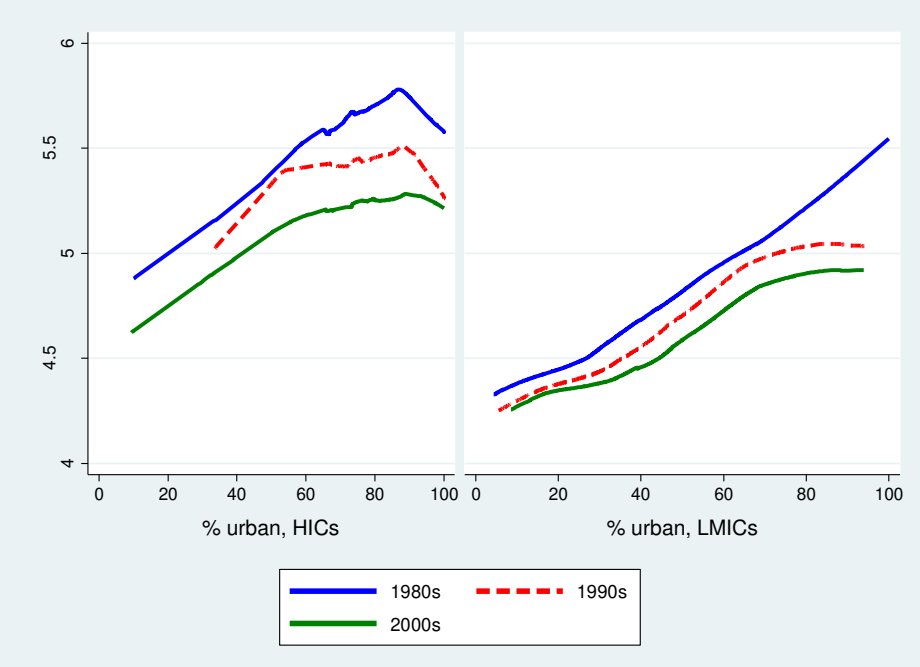


Figure 3. Average diabetes prevalence by % living in urban areas, time period and income group

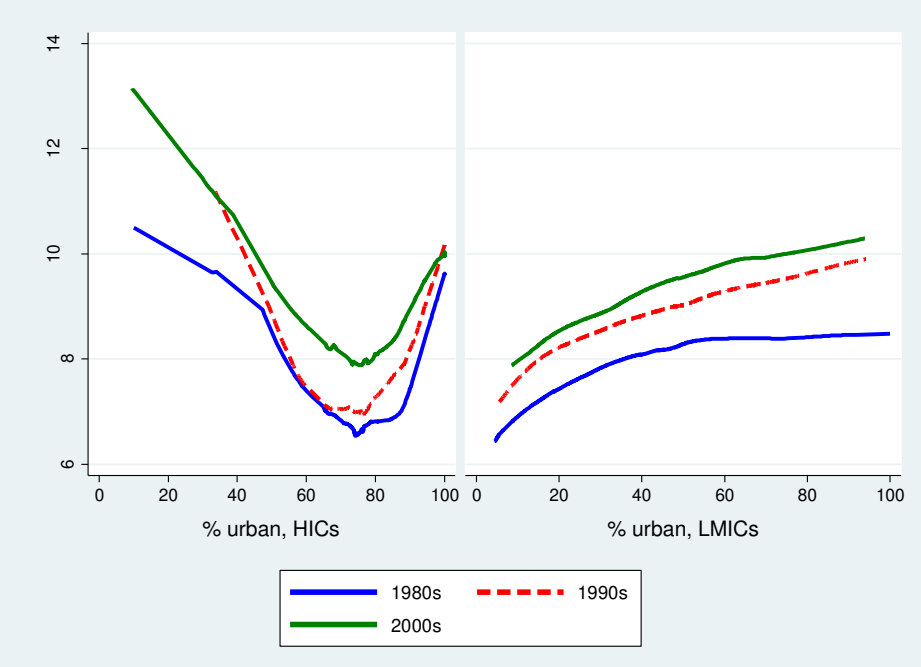
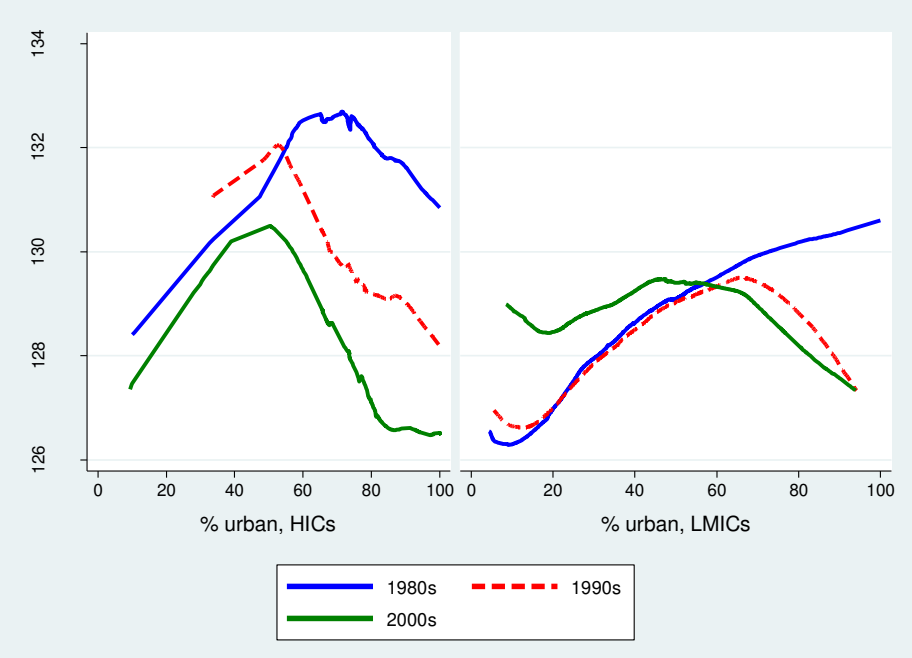



Figure 4. Average SBP (mmHg) by % living in urban areas, time period and income group



### *3.2 Regression results*

Results are reported separately for each outcome variable and by gender (Table 2). All models include controls, country fixed effects, general and region-specific time trends. In the pooled sample including all countries, urbanization is significantly positively related to mean BMI and average total cholesterol. In addition, it is significantly positively related to diabetes prevalence among women, and negatively- to average SBP among men. . While the association with BMI is positive and significant for both men and women, it is somewhat stronger for the latter: each p.p. increase in the proportion of people living in urban areas is related to an increase in BMI of about 0.024 among women, and by 0.016 among men. Likewise, the association is stronger for women when the outcome is diabetes: each p.p. increase in the extent of urbanization is predicted to lead to an almost 0.034 p.p. greater diabetes prevalence in this group. We note that the evidence presented here is consistent with bivariate relationships shown in Figures 1-4.

Table 2: Association between urbanization and NCDs, full sample

| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) |
|---|---|---|---|---|---|---|---|---|
| | BMI | | Total cholesterol | | Diabetes | | SBP | |
| | Females | Males | Females | Males | Females | Males | Females | Males |
| Urban, % | 0.024*** | 0.016*** | 0.003** | 0.004*** | 0.034** | 0.025 | -0.025 | -0.065*** |
| S.E. | (0.007) | (0.005) | (0.001) | (0.001) | (0.015) | (0.016) | (0.029) | (0.021) |
| Pop. growth , % | 0.033** | 0.015* | -0.003 | -0.002 | -0.009 | 0.007 | -0.040 | -0.019 |
| S.E. | (0.015) | (0.009) | (0.002) | (0.002) | (0.027) | (0.023) | (0.049) | (0.031) |
| Pop. density | -0.000 | -0.000 | -0.000 | -0.000 | -0.003 | -0.001 | -0.001 | 0.004 |
| S.E. | (0.001) | (0.001) | (0.000) | (0.000) | (0.002) | (0.002) | (0.005) | (0.004) |
| Productivity | 0.000* | 0.000 | 0.000* | 0.000 | 0.000 | -0.000 | 0.001 | 0.000 |
| S.E. | (0.000) | (0.000) | (0.000) | (0.000) | (0.000) | (0.000) | (0.001) | (0.000) |
| CFE | Yes | Yes | Yes | Yes | Yes | Yes | Yes | Yes |
| Observations | 4,245 | 4,245 | 4,245 | 4,245 | 4,245 | 4,245 | 4,245 | 4,245 |
| R-squared | 0.896 | 0.899 | 0.719 | 0.716 | 0.656 | 0.469 | 0.645 | 0.484 |
| N. of countries | 169 | 169 | 169 | 169 | 169 | 169 | 169 | 169 |

Notes: Robust standard errors in parentheses: *** p<0.01, ** p<0.05, * p<0.1. Standard errors are clustered at country level. In all models, urbanization is lagged by one year. All models also control for the log GDP per capita, proportion of the population aged 15-64 years, female proportion of the population, time trend, region-specific time trends and country fixed effects.

As a robustness check, in the Annex (Tables A2.1-A2.3), we compare regression estimates between models where the outcome is defined either with the Global Burden of Diseases, Injuries, and Risk Factors (GBMRF) data, or with the alternative Global Health Observatory (GHO) repository from the WHO[7]. GBMRF estimates were prepared as part of the wider Global Burden of Diseases, Injuries, and Risk Factors study, and as such both are based on Bayesian meta-regression modelling methodology. The GHO estimates were also based on Bayesian methodology, though they relied on inputs from a number of other collaborators and/or data sources. The correlation between GBMRF and GHO measures is very high for BMI (0.96) and for total cholesterol (0.99) and it is weaker for systolic blood pressure (0.87). On the whole, regardless of the outcome variable used, there is very little difference between parameter estimates

Results presented in Table 3 indicate that the association between urbanization and NCD risk factors is almost totally driven by the LMICs group, which is also consistent with the bivariate associations shown in Figures 1, 2 and 3. Indeed, it is always insignificant in the HICs, while the reverse is true for BMI, total cholesterol, diabetes (for both genders) and SBP (for males) among the LMICs. Interestingly, each p.p. increase in urbanization is related to an about 0.05% increase in the prevalence of diabetes in LMICs, while in the full sample we found much weaker association.

Table 3. Association between urbanization and NCDs, by country income group

| | (1) | (2) | (3) | (4) | (6) | (7) | (5) | (8) |
|---|---|---|---|---|---|---|---|---|
| | BMI | | TC | | Diabetes | | SBP | |
| | Females | Males | Females | Males | Males | Females | Females | Males |
| | | | | High income countries | | | | |
| Urban, % | -0.004 | 0.004 | 0.001 | 0.000 | -0.013 | -0.060 | -0.012 | -0.049 |
| S.E. | (0.018) | (0.012) | (0.005) | (0.005) | (0.040) | (0.048) | (0.055) | (0.057) |
| Country fixed effects | Yes | Yes | Yes | Yes | Yes | Yes | Yes | Yes |
| Observations | 846 | 846 | 846 | 846 | 846 | 846 | 846 | 846 |
| R-squared | 0.897 | 0.941 | 0.877 | 0.870 | 0.728 | 0.689 | 0.941 | 0.852 |
| Number of countries | 47 | 47 | 47 | 47 | 47 | 47 | 47 | 47 |
| | | | | Low and middle income countries | | | | |

[7] http://apps.who.int/gho/data/node.main.A867?lang=en

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Urban, % | 0.026*** | 0.020*** | 0.003* | 0.004*** | 0.052*** | 0.047*** | -0.048 | -0.08*** |
| S.E. | (0.008) | (0.006) | (0.002) | (0.001) | (0.017) | (0.018) | (0.033) | (0.025) |
| CFE | Yes | Yes | Yes | Yes | Yes | Yes | Yes | Yes |
| Observations | 3,232 | 3,232 | 3,232 | 3,232 | 3,232 | 3,232 | 3,232 | 3,232 |
| R-squared | 0.899 | 0.866 | 0.705 | 0.624 | 0.706 | 0.453 | 0.601 | 0.241 |
| Number of countries | 146 | 146 | 146 | 146 | 146 | 146 | 146 | 146 |

Notes: Robust standard errors in parentheses: *** p<0.01, ** p<0.05, * p<0.1. Standard errors are clustered at country level. In all models, urbanization is lagged by one year. All models also control for population growth rate, population density, productivity, proportion of the population aged 15-64 years, female proportion of the population, time trend, region-specific time trends and country fixed effects.

In the Annex, Table A4, we consider a further subdivision between middle and low-income countries. Results suggest that the association continues to be strong among the middle income countries, while becoming slightly weaker in the low income countries (although it continues to remain significant there for BMI and diabetes, despite large reduction in the sample size).

To further investigate the potentially heterogeneous urbanization effect, we group countries according to the intensity of their urbanization process. We find that in the three regions that underwent the fastest process of urbanization (as per Table 5) - East Asia, Americas and Sub Saharan Africa - urbanization was related to a greater impact on BMI and diabetes than in the remaining regions, while the reverse was true for TC. No clear pattern emerged for SBP (see tables A3.1-A3.4 in the Annex).

Finally, we conduct a tentative pathway analysis of the association between urbanization and health outcomes. A rather widespread approach to this is to use the baseline model as a starting point, subsequently adding new potential mediators and thereby assessing how the association between main variables of interest changes. However, as was shown elsewhere (Angrist & Pischke, 2008; Bullock & Ha, 2011) and is often under-appreciated, doing so can produce biased estimates, as the *ceteris paribus* assumption will be violated.[8] Therefore, we will follow a different approach, which despite not providing a definite answer

[8] To directly study the effects of mediators, we would require appropriate instrumental variables for each potential mediators, which unfortunately are not available.

as to how much a certain variable may mediate the association of interest, should at least provide some indication of whether the variable in question is (or is not) indeed a mediator.

Specifically, we select two potential mediators related to energy availability, based on the available medical evidence that links them with NCD risk factors: food supply (kcal/capita/day) and fat supply (g/capita/day)). This choice was based on the literature showing that both population and individual-level energy intake is positively related to average BMI and overweight (Dave et al., 2016; Doytch et al., 2016; Goryakin & Suhrcke, 2014) and diabetes (Pan et al., 1997), although its relationship with average total cholesterol is more nuanced, with "bad" LDL cholesterol levels in the blood being positively related to only some kinds of fats, e.g. trans fat and saturated fat consumption (Mensink & Katan, 1990). Similarly, total cholesterol levels also appears to be positively related to saturated fat consumption (Clarke et al., 1997).

We estimate the effect of urbanization on these mediators, denoted $M^j_{it}$ , by regressing each of them on lagged urbanization (equation 2), the same controls as in equation (1) including country fixed effects and time trends. The results are shown in Table 4 below.

$$M_{it}^{j} = \alpha + \beta_1 X_{it-1} + \mathbf{Z_{it}}'\beta_2 + \alpha_i + \varepsilon_{it} \qquad (2)$$

Table 4. The association of urbanization with potential mediators related to energy availability

| | (1) | (2) |
|---|---|---|
| | Food supply, kcal/capita/day | Fat, g/capita/day |
| Urban, % | 5.784* | 0.307** |
| | (3.062) | (0.137) |
| CFE | Yes | Yes |
| Observations | 3,992 | 3,992 |
| R-squared | 0.324 | 0.346 |
| Number of countries | 159 | 159 |

Notes: Robust standard errors in parentheses: *** p<0.01, ** p<0.05, * p<0.1. Standard errors are clustered at country level. In all models, urbanization is lagged by one year. All models also control for population growth rate, population density, productivity, the log GDP per capita, proportion of the population aged 15-64 years, female proportion of the population, time trend, region-specific time trends and country fixed effects.

We find that both energy supply-related variables are significantly related to urbanization. These findings suggest that part of the effect of urbanization on NCDs risk factors is potentially mediated by the higher calories intake and nutritional habits that are likely to emerge in urban environments.

The question also remains about whether the effect of urbanization is constant, or whether it might also depend on the prevalence of a risk factor in a given country. To check this possibility, we augmented model (1) by the interaction between urbanization and the initial level of the risk factor $Y_{i0}$. The initial level of the risk factor $Y_{i0}$ is predetermined compared to the current level of urbanization and country fixed effects will capture those predetermined factors that might have produced the co-evolution of urbanization and risk factors before and up to time 0. The results (shown in Appendix, Table A5) suggest that the effect of urbanization on all outcomes (except diabetes) declines with a greater baseline prevalence of these risk factors.

## 4. Discussion

In our analysis, urbanization turned out to be a consistent driver of at least two NCD-related risk factors: BMI and TC. However, its effect is comparatively modest: only if we hypothetically decreased urbanization from its sample maximum (100 percent) to its minimum (5 percent), then the implied reduction in the NCD risk factors would be of an appreciable magnitude. Specifically, the corresponding average reduction in TC would be 0.40 mmol/L, which is about equal to one standard deviation in this variable; the reduction in BMI of 2.3 kg/m$^2$; and the reduction in diabetes prevalence of 3.2 p.p. (only among women). The latter two effects would be comparable to those obtainable by means of certain diet and exercises interventions (Miller et al., 1997) or some well-known lifestyle modification

programs[9] (Group, 2002). More realistically, if we considered a variation in the urbanization rate equivalent to that which occurred between 1985 and 2005, then the changes in the risk factors predicted by model (2) is of the magnitudes reported in Table 5, for each region.[10]

Table 5. Predicted changes in the risk factors explained by a variation in urbanization rates equivalent to that observed between 1985 and 2005. By region.

| | urbanization, 1985 % | urbanization, 2005 % | Δ urb, p.p. | Δ BMI, kg/m2 | Δ TC, mmol/l | Δ DB, p.p |
|---|---|---|---|---|---|---|
| SSA | 24.6 | 33 | 8.4 | 0.17 | 0.03 | 0.25 |
| Americas | 67.2 | 77.1 | 9.9 | 0.20 | 0.03 | 0.30 |
| East Asia | 29.7 | 46 | 16.3 | 0.33 | 0.06 | 0.49 |
| MENA | 52.6 | 60.6 | 8 | 0.16 | 0.03 | 0.24 |
| ESA | 56.9 | 63.4 | 6.5 | 0.13 | 0.02 | 0.20 |
| Former Soviet Union | 64.6 | 64.3 | -0.3 | -0.01 | 0.00 | -0.01 |
| North America | 73.4 | 79 | 5.6 | 0.11 | 0.02 | 0.17 |
| Pacific | 69.3 | 69.1 | -0.2 | 0.00 | 0.00 | -0.01 |
| South Asia | 23.7 | 29.1 | 5.4 | 0.11 | 0.02 | 0.16 |
| Western Europe | 72.6 | 75.5 | 2.9 | 0.06 | 0.01 | 0.09 |

To illustrate, in the SSA region, the proportion of people living in urban areas increased by about 8.4 p.p. between 1985 and 2005. Holding everything else constant, and based on results in Table 2, the implied change in average BMI in that region, if urbanization increased by the same 8.4 p.p. in the future, would be by about 0.17 kg/m$^2$; the increase in TC would be 0.03 mmol/l; and the proportion of people living with diabetes would increase by about 0.25 p.p. That said, an increase in BMI does not necessarily imply worsening health status, especially in the context of low income countries, where a significant proportion of people is underweight. Also, note that despite generally increasing urbanization rates in most regions between 1985 and 2005, a number of regions experienced declines in the risk factors over the same period (Table 1), suggesting that improvements in health may continue despite more people moving to live in the cities.

[9] In the lifestyle intervention program, participants were expected to achieve a weight reduction of at least 7 percent, through a healthy diet, and at least 150 minutes of moderate intensity exercise per week.

[10] We assume that the effect of urbanization on risk factors is homogenous across regions.

Taken together, our findings suggest that potentially desirable effects of urbanization on selected NCD risk factors (e.g. via improved health services) may be outweighed by certain adverse ones, possibly including less healthful diets, lack of exercise, higher pollution and stress levels that could also result from urbanization. It is notable that urbanization is more strongly (and positively) related to energy-imbalance-related NCD risk factors, i.e. TC, diabetes and BMI, which suggests that people living in cities may tend to get more exposed to energy-dense foods, and may develop a greater preference for calorie-rich "Western" diets (Popkin, 1999, 2001; Popkin et al., 2012; Popkin & Gordon-Larsen, 2004; Yusuf et al., 2001) or may have reduced opportunities for physical activity (Monda et al., 2007; Popkin, 1999; Swinburn et al., 2011). Our preliminary pathway analysis in particular indicates that two variables related to energy intake – total calorie and fat per capita supply – are potentially mediating the association between urbanization and NCD risk factors.. This finding seems particularly salient in view of the strong evidence on the steady increase in caloric intake around the world in the recent decades, and that overweight burden is significantly driven by growing calorie consumption from energy-dense foods (Cutler et al., 2003; Dave et al., 2016; Finkelstein et al., 2005).

The question still remains about why the effect of urbanization on SBP may be weaker than on BMI/TC/diabetes. The main population-level determinants of SBP include salt intake, fruit and vegetable consumption, exercise, adiposity and antihypertensive medications use. Although energy imbalances caused by urbanization are likely to lead to greater diabetes prevalence, BMI and TC levels, they can also affect SBP levels more ambiguously, for example through an increase in the fruit and vegetable consumption likely to be more available in urban areas, or through greater take-up of antihypertensive medications in urbanized health systems. If these behaviours counteract the adverse impact of urbanization on SBP mediated by BMI, then the urbanization-SBP relationship may well be a

priori unclear. In line with this logic, a number of studies have attributed the observed decline in the SBP levels to more intensive management of hypertension with medications, which tends to be much more widespread in the richer countries (which coincidentally are more urbanized) (Danaei et al., 2011a). There has not been much research on the relationship between living in urban areas and fruit and vegetable consumption, but at least two studies found evidence of positive association (Goryakin et al., 2015b; Johansson et al., 1999).

It is also notable that the association between urbanization and three NCDs risk factors is always positive but considerably stronger in the sample of LMICs compared to the HICs. A similar pattern has also been observed by Goryakin and Suhrcke (2014) and Popkin et al. (2012) who found that living in urban areas is positively associated with the probability of being overweight predominantly in the low income countries. We think that such difference between LMIC and HIC countries may be due to a trade-off between the demand for calories and the demand for healthy lifestyles whose balance depends on the level of country income. In the LMICs, living in urban areas is associated with greater demand for calories compared to rural areas (Neuman et al., 2013) due to the better socio-economic conditions generally available in urban areas (Monteiro et al. 2004). As countries get richer, the demand for thinness and for healthier lifestyles increases especially among the urban residents. The latter may outweigh the demand for calories (Goryakin & Suhrcke, 2014).[11]

Our data provide an opportunity of testing this hypothesis by estimating whether the effect of urbanization on the demand for calories is stronger in LMICs compared to HICs. We thus estimate model (2) separately in the LMICs and the HICs focusing on the two energy supply outcomes discussed above. Our results suggest that urbanization is positively associated with equilibrium supply of calories/fats in LMICs but not HICs. This is in line

[11] Monteiro et al (2004), and (Dinsa et al., 2012), suggest that while there is positive association between socioeconomic status and the probability of being overweight in the low income countries, this gradient progressively reverses as country income increases.

with our expectation that living in urban areas in LMICs should be related to higher SES and, in turn, higher SES should be related to greater equilibrium supply and demand for calories there. On the other hand, we should expect that living in urban areas in HICs should lead to greater demand for healthier diet, if the "Monteiro Hypothesis" is correct, and the results in Table 6 also support this.

Table 6. Association of urbanization with energy intake mediators, by income group

| | (1)<br>Food supply, kcal/capita/day<br>LMICs | (2)<br>Food supply, kcal/capita/day<br>HICs | (3)<br>Fat, g/capita/day<br>LMICs | (4)<br>Fat, g/capita/day<br>HICs |
|---|---|---|---|---|
| Urban, % | 5.592** | -10.538** | 0.255* | -0.578** |
| | (2.818) | (4.259) | (0.134) | (0.283) |
| Observations | 3,007 | 823 | 3,007 | 823 |
| R-squared | 0.319 | 0.575 | 0.309 | 0.434 |
| Number of countries | 136 | 44 | 136 | 44 |

Notes: Robust standard errors in parentheses: *** p<0.01, ** p<0.05, * p<0.1. Standard errors are clustered at country level. In all models, urbanization is lagged by one year. All models also control for population growth rate, population density, productivity, proportion of the population aged 15-64 years, female proportion of the population,, time trend, region-specific time trends and country fixed effects.

Several previous studies focussing on LMICs found positive association between urbanization (or at least living in the city) and BMI, overweight and obesity (Delisle et al., 2012; Goryakin et al., 2015a; Goryakin & Suhrcke, 2014; Ambady Ramachandran et al., 2008), consistent with our findings in Figure 1 and Table 3. Likewise, diabetes prevalence was found to be positively related to the extent of urbanization (or living in the city) in earlier work, eg in (Ambady Ramachandran et al., 2008; A Ramachandran et al., 1999), consistent with findings presented in Figure 3 and Table 3. Several studies also found a positive link between urbanization and abnormal cholesterol levels in LMICs (Delisle et al., 2012; Snehalatha & Ramachandran, 2009; Vorster et al., 2000), again consistent with our findings in Figure 2 and Table 3. In a review article, Ibrahim and Damasceno (2012) found that hypertension prevalence was generally higher in urban compared to rural areas in several LMICs, although this was true only for 7 countries, and apparently without adjustment for

potential confounders. On the other hand, in a study by Delisle et al (2012), average SBP of those living in a large city was significantly smaller than of those living in rural areas in Benin, which is consistent with results in Table 3. Likewise, those living in the wealthiest urban areas in South Africa were also found to have lower mean blood pressure then the reference group (Vorster et al., 2000). Our paper adds to this body of evidence by providing a "big picture", based on advanced econometric analysis of several large country level datasets covering the great majority of world countries.

As far as we are aware, similar data as in our present paper was only used in three previously published papers. In the first one, Danaei et al (2013) graphically showed positive bivariate associations between extent of urbanization and average BMI, in 1980 and 2008, for both men and women. Our paper differs in several important respects: 1) we show graphical associations by different time periods and different country income levels, including for average SBP, diabetes and cholesterol levels; 2) we estimate multivariate models, controlling for a range of potential confounders, time trends and country fixed effects and 3) we test for the link between urbanization and two potentially important mediators. In the second paper, (De Vogli et al., 2014b) estimated the impact of market deregulation on average BMI, controlling for the % of population living in urban areas (and several other variables). In their study, however, the country sample was limited to only 25 high income OECD countries; the impact of urbanization was not of primary interest (and therefore their control variables were different from ours); they did not explore the impact of urbanization on other outcomes. Still, they found urbanization to be positively and significantly related to average BMI. In the third, (De Vogli et al., 2014a) estimated the association between country-level economic globalization and average BMI. Again, the impact of urbanization was not of primary interest and thus they used a different set of controls (and in fact the parameter estimates on

urbanization were not presented in the paper), the sample of countries was smaller, and the impact on other outcomes of potential interest was not measured.

In interpreting the results, the limitations of our study need to be borne in mind. Thus, our outcome variables do not always reflect empirically observed data, but instead are often estimates, if carefully derived ones and arguably the best and most comprehensive available, as they are based on a large number of surveys, and related direct and indirect epidemiological evidence (Danaei et al., 2011a; Danaei et al., 2011b; Finucane et al., 2011). However, most of potential measurement error is likely to be country-specific, and thus is wiped out in country fixed effects models. Any remaining measurement error is unlikely to bias the estimates, but will rather reduce their precision. The fact that we still find statistically significant estimates, particularly in LMICs where measurement error concerns are arguably stronger, is reassuring. In addition, in our outcome variables validation exercise, we found that the correlation between GBMRF and GHO indicators was very high, particularly for BMI and TC outcomes, and that regression parameter estimates differed very little, regardless of the source of the outcome variables.

We also acknowledge that it is rarely possible to draw strong causal conclusions based on observational data. However, we at least increase the confidence in the causal nature of the estimated relationship by controlling for country fixed effects, general and region-specific time trends, as well as by lagging the main covariate of interest – urbanization – by one year.

Bearing in mind these caveats, and while the overall magnitude of the effect appears quite modest, urbanization consistently emerges as a relevant driver of NCD risk factors in our study. In this sense we provide a useful complement to the previous accounts, which were largely based on anecdotal or conceptual discussions or more descriptive analysis. In light of the bigger influence of urbanization on NCD risk factors in poorer countries, policy efforts

should be targeted towards preventing and mitigating the adverse side effects of urbanization, in particular in low and middle income countries.

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