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Exploring Differences in the Rate of Type 2 Diabetes Among American Cities: How Urbanization Continues to Challenge the Traditional Epidemiological View

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Abstract: As the world's largest urban regions continue to expand, a concomitant rise in non-communicable diseases, particularly type 2 diabetes, poses an increasingly ominous challenge to experts in the field of public health. Given that the majority of the world's population (54%) resides in urban areas, a figure likely to reach two-thirds by 2050, this issue presents serious implications for medical practitioners as well as policymakers seeking to manage long-term healthcare costs while sustaining historic increases in life expectancy. To explore how these trends are continuing to affect the United States, a multiple regression analysis was conducted using data provided by the Centers for Disease Control and Prevention (CDC) through their initiative, *500 Cities: Local Data for Better Health*. The regression models revealed that larger cities reported significantly higher rates of type 2 diabetes even after controlling for variables that have been perennially linked to disease onset (e.g., levels of obesity, sedentary behavior). Implications are discussed, most notably the argument for moving beyond the 'food desert' paradigm when identifying and explaining which characteristics of larger cities place their residents at increased risk. This approach could help reveal opportunities for intervention that may not have garnered sufficient attention in the extant literature.

Keywords: urbanization; type 2 diabetes; epidemiology

1. Introduction

Academics and government officials alike have been increasing their exhortations with respect to the public health crises that have emerged amidst the breathtaking pace of recent economic and urban development, most notably in South Asia (e.g., India) and China [1–3]. Both the United Nations and the World Health Organization have launched initiatives to address the alarming rise of chronic medical conditions as agrarian landscapes cede to urban sprawl [4]. Although these consequences vary (for example, when comparing developing countries with those that are industrialized), a phenomenon that has proven consistent worldwide is the notable increase in non-communicable diseases (NCDs), prominent among these being type 2 diabetes [5]. Researchers have long documented how urbanization and economic development often engender considerable changes in lifestyle, particularly with respect to eating habits, level of physical activity and work patterns [6–9]. Consequently, it is perhaps not surprising that approximately two-thirds of all individuals stricken with type 2 diabetes reside in or around urban areas, according to the International Diabetes Federation [10].

As mentioned, researchers have drawn distinctions between the types of urbanization occurring in poorer countries (e.g., Bangladesh) versus those in the developed world. For example, in Dhaka (the capital of Bangladesh), the scale of its sprawl leaves its nearly nine million residents vulnerable to more acute crises, such as food shortages and malnutrition [11]. Other conditions that preceded urbanization, such as tuberculosis and malaria, are curiously rising in the developing world even as it

suffers notable increases in obesity and diabetes, a phenomenon that has been described as a “double burden” for the health care communities in these beleaguered regions [12]. Certainly, the nature and context of urbanization is fundamental to understanding its role in disease onset.

As Gassasse et al. state, it is not urbanization per se but the type of urban growth that affects rates of type 2 diabetes in any particular country [5]. Indeed, given the disconcerting figures in the public health literature, it is easy to lose sight of the longstanding benefits urban residents have enjoyed, particularly in the developed world. For example, between 1969 and 2009, life expectancy in the United States increased significantly more for those living in urban areas compared to residents from rural environs [13]. However, by definition, the gains observed when examining the urban–rural divide do not directly address the escalation in type 2 diabetes (among other chronic diseases) seen in American cities during the same time span. One American city that warrants attention and provides a useful context and rationale for this study is the nation’s fifth largest: Houston, Texas. Even with its considerable size, the city of Houston, as well as the surrounding metropolitan region, is still growing at a robust pace [14]. Houston also has one of the highest rates of obesity (34.4%) and diabetes (13.5%) in the nation [15]. It is estimated that by 2040, 1 in 5 residents will be diabetic [16]. In this light, Houston provides a template for understanding how certain cities can impact disease rates not only by their sheer size, but by the nature of their rapid (and ongoing) growth as it occurs in an already sprawling urbanized landscape. Although seen as a sign of commercial and civic vitality, this type of expansion also brings a measure of instability, particularly for residents who are economically vulnerable. Their sense of social disruption and displacement continues to receive attention from researchers documenting the ramifications of gentrification [17]. For neighborhoods in the midst of transformation, longtime residents may feel increasingly alienated as “existing services change, move, or are shut down—with concrete consequences for those [who are] relying on them.” [18] (para. 1). Access to basic amenities, a longstanding logistical conundrum for many citizens within America’s largest urban areas, may prove even more difficult amidst rapid economic growth and development. Indeed, researchers continue to document the pernicious effects of “food deserts” for the poorest residents in major American cities despite their ongoing revitalization [19].

There are other challenges, however, for residents of large and expanding urban environments regardless of their socioeconomic status. This second issue is represented by a construct that has only recently gained visibility and attention from social scientists; a phenomenon known as *time poverty*. According to Williams, Masuda and Tallis, “Time is a scarce resource that individuals and households must allocate to produce goods, obtain services, and pursue rest and relaxation. Time poverty has been proposed as a complement to income poverty, yet it remains a relatively unknown measure in both policy and research spheres.” [20] (p. 265). In other words, too little is known about the negative effects (e.g., health-wise) for someone who is *time-poor*, a condition that can coincide with financial strain but may prove just as noxious for the gainfully employed. Even as researchers continue to reveal the deleterious effects of longer commutes, the implications regarding the drain on employees’ time, particularly when combined with longer working hours, warrants further examination [21,22].

The last tenet to consider regarding the health risks for residents of large and expanding urbanized regions is the *sedentary nature of work*, specifically the kind of prolonged sitting that countless employees endure in their offices and meeting spaces. In an eight-year longitudinal study of U.S. workers, researchers discovered that time spent sitting in an office was significantly associated with a higher Body Mass Index (BMI), [23]. (This effect was seen only for male workers, however.) Other investigations have similarly revealed the ramifications of extensive sitting within the modern workplace [24,25]. When calculating the amount of time most workers sit, including their commute (often by automobile), the following findings from Vallance et al. [26], citing work from Biswas et al. [27], are particularly striking,

“... high volumes of sitting (e.g., >8 hours/day) have been found to be associated with adverse health outcomes. The strongest risk estimates have been observed for type 2 diabetes, with the most recent meta-analysis reporting a hazard ratio of 1.91 (95% confidence interval [CI] = 1.64, 2.22).” [26] (p.1478)

Clearly, more investigations are necessary to understand how these factors, whether they are well documented (e.g., food deserts, sedentary behavior) or just emerging into the scholarly realm (i.e., time poverty), contribute to the prevalence of chronic diseases such as type 2 diabetes in large urban environments. Specifically, how do these (and other) factors influence the ability of residents in larger cities to effectively manage and maintain their health? What points of intervention are being overlooked or obscured by the conventional view of how people become diabetic? Addressing these questions may facilitate more productive discussions among healthcare providers, urban planners and government officials, both in the U.S. and abroad, as this formidable epidemiological challenge invariably becomes more daunting.

2. Materials and Methods

2.1. Sample and Dataset

The sample and dataset used for this analysis were derived from an initiative entitled *500 Cities: Local Data for Better Health*. This project was created in 2015 through a partnership between the Centers for Disease Control and Prevention (including the CDC Foundation) and the Robert Wood Johnson Foundation. Researchers collected information on 27 health-related variables from the 497 largest cities in the United States. The other three cities in the dataset were chosen to ensure that all 50 states were represented. (Each city, Burlington, Vermont, Charleston, West Virginia and Cheyenne, Wyoming, is the largest in each state.) The 27 variables covered three general categories: 1. *unhealthy behaviors* (5 variables), 2. *prevention measures* (9 variables), and 3. *health outcomes* (13 variables). (See Appendix A for the list of variables employed in this study. The complete list can be retrieved from <https://www.cdc.gov/500cities/measure-definitions.htm>.) According to its website, the project utilized the CDC's Behavioral Risk Factor Surveillance System, the 2010 U.S. Census and the American Community Survey, as its primary data sources. The website also makes the dataset available to the public (via download). The files downloaded from the website did not contain any missing values or incomplete information, nor were there any anomalies or errors apparent within the dataset.

2.2. Data Transformation

Before calculating any inferential statistics, two forms of data transformation were performed on the distribution of population sizes for the 500 cities (i.e., subjects) given that it was highly skewed (see Results section). The first approach involved converting each city's population size to its $\log_{10}$ value (a process known as log transformation.) A $\log_{10}$ value represents the number of times the number 10 is multiplied exponentially in order to equal the original figure, (e.g., the $\log_{10}$ value of $1000 = 3$, since $10^3 = 1000$). This technique is commonly used to transform positively skewed distributions since it effectively reduces extremely high scores [28].

The second data transformation involved segmenting the 500 cities into groups of 50 to form 10 population 'tiers', essentially converting population size from a continuous variable into an ordinal variable. The 50 largest cities were ranked in the top or highest population 'tier', with the next 50 (in terms of size) being ranked in the second tier, and so forth. As a result, two regression analyses were conducted in this study, the first using population as a continuous variable (via log transformation) and the second analysis framing city size in terms of levels or tiers. This second data transformation was not conducted simply to address excess skewness, however. It was also performed in recognition of the considerable similarity between certain cities despite stark differences in the size of their populations.

For example, cities such as New York and Boston share several traits that influence the daily lives of their citizens, specifically, an intense urban core, a comprehensive mass transit system and considerable traffic congestion. Other similarities include the nature of employment and their

educational institutions, as well as diversity in their cultural landscapes. Yet the population difference between the two cities are vast, with New York having nearly 8.2 million residents compared to approximately 620,000 living in Boston, according to the 2010 U.S. Census. Indeed, in April of 2018, the New York Times published an article entitled “What is Your City’s Twin?”, which noted the strong economic similarities (particularly regarding local job markets) between cities such as Dallas and Atlanta (approx. 1.2 million residents vs. 423,000), as well as Los Angeles and Miami (approx. 3.8 million vs. 400,000), despite considerable differences in their population size [29].

These similarities would likely be underestimated, or possibly ignored, should city size be viewed exclusively in terms of raw population figures. Instead, the tier system used in this analysis creates segments (of 50 cities each) that can capture the shared pertinent traits between New York and Washington, DC, for example, (both being members of ‘Tier 1’) while remaining small enough to suitably incorporate the role of the actual population count in determining a city’s size. Overall, both forms of data transformation employed in this study (log transformation and ordinal/ranking/tier) are preferable to viewing the largest cities in the U.S. (i.e., New York, Los Angeles, Chicago, Houston, Philadelphia) as so-called ‘outliers’. Indeed, any comprehensive investigation into the relationship between urbanization and the onset of chronic diseases such as type 2 diabetes would be highly degraded in terms of its external validity if these cities were excluded from the analysis.

2.3. Analysis

As mentioned, the 27 health-related variables measured in the dataset represent the following categories: (1) *unhealthy behaviors*, (2) *prevention measures* and (3) *health outcomes*. This categorization, through its implicit sequencing of behaviors that either increase or reduce health risks (i.e., groups 1 and 2), followed by their ramifications (i.e., group 3), was particularly useful for exploring possible antecedents to disease onset via multiple regression. In terms of how variables were chosen, this analysis was guided by the prevailing literature involved with the medical and behavioral precursors to type 2 diabetes (e.g., [30,31]). The outcome variable (prevalence of type 2 diabetes) is defined by this dataset as the ‘percentage of respondents aged 18 and older in a given municipality who were ever diagnosed with this disease by a physician, nurse or other health care professional’ (see Appendix A).

A stepwise selection method, using the Schwarz Bayesian Information Criterion, was utilized to test the viability of city size as an explanatory variable within the regression models. In stepwise regression, variables may be added or subtracted based on their ability to uniquely explain any variance in the outcome variable whenever a new, potential explanatory variable is introduced into the model [32]. The Schwarz Bayesian Information Criterion is often employed to prevent the regression models from possessing an excessive number of explanatory variables, a common precursor to a condition known as “overfitting”. A regression model is considered to be “overfitted” if it is too closely tied to the sample or ‘test’ data. When overfitted, the model often fails to provide the same degree of predictive value for any new observations compared to its viability when working only with the original dataset.

3. Results

Descriptive statistics for the variables included in the final regression models are provided in Table 1. As mentioned, the definitions for these variables are listed in Appendix A.

Table 1. Descriptive statistics.

Variable	N	Mean	SD	Min	Max
Size of city population	500	206,041.62	457,481.05	42,417	8,175,133
Size of city population (log transformed)	500	5.13	0.31	4.43	6.91
Lacking access to health insurance (pct. of city pop.)	500	16.47	6.45	4.4	47.6
Obesity rate (pct. of city pop.)	500	29.3	5.86	14.7	45.2
Lacking physical activity during leisure time (pct. of city pop.)	500	25.86	6.25	13	44.8
Prevalence of type 2 diabetes (pct. of city pop.)	500	10.25	2.47	5.5	18.4

The levels of skewness and kurtosis for the variable *size of city population* were 12.29 and 194.29 respectively before a log transformation was performed on its distribution. After the data transformation, the level of skewness was 1.82, with a kurtosis level of 4.32. The figures for the remaining four variables represent percentages. For example, the average or mean percentage of city residents who reported lacking access to health insurance was 16.47%. The average or mean obesity rate among the 500 cities was 29.3%, etc.

As shown in Table 2, city size, as determined by population, was significantly correlated with the prevalence of type 2 diabetes after controlling for other variables that have been established in the extant literature as precursors to this disease, (i.e., obesity, lack of physical activity, lacking access to health insurance). All four variables in the regression model were found to be statistically significant with a *p*-value less than 0.001. The adjusted R-squared figure indicates that this model explains a healthy majority of the variance (80.6%) with respect to the outcome variable, the prevalence of type 2 diabetes. Additionally, neither the Variance Inflation Factor nor the Tolerance Figure indicates any serious risk of multicollinearity among the explanatory variables. [(VIF, Tolerance) for City Size: (1.02, 0.98), Obesity: (3.56, 0.28), Lacking Physical Activity: (4.7, 0.22) and Lacking Access to Insurance (2.04, 0.49).]

Table 2. Effects of insurance access, obesity, sedentary behavior (during leisure time) and size of city population on the rate of type 2 diabetes.

	Rate of Type 2 Diabetes	95% Confidence Intervals
Lacking access to health insurance	0.139 ** (0.01) [12.91]	(0.118, 0.16)
Obesity Rate	0.08 ** (0.016) [5.14]	(0.05, 0.112)
Lacking Physical Activity During Leisure Time	0.166 ** (0.017) [9.8]	(0.132, 0.199)
Size of City Population (log transformed)	0.71 ** (0.159) [4.46]	(0.398, 1.023)
Constant/Intercept	−2.34 * (0.838) [−2.79]	(−3.98, −0.691)

Note: Standard errors appear in parentheses, t-scores appear in brackets. ** *p* < 0.001, * *p* < 0.01. Observations: 500. Adj. R-Squared: 0.806 **. F(4, 495): 571.69.

As mentioned in the Methods section, a second regression analysis, (with results listed in Table 3), was conducted with *size of city population* being measured through a ‘tier’ system (i.e., as an ordinal variable.) The variable was reverse scored during the analysis. For example, the highest (or first) tier, which contained the 50 largest cities, was given a score of 10, with the second tier given a score of 9, and so forth.

Similar to the first regression analysis, city size (as measured by population tier) was significantly correlated with the prevalence of type 2 diabetes when controlling for all other pertinent variables. Also, each of the four variables in the regression model were similarly found to be statistically significant with a *p*-value less than 0.001. This second regression model explained marginally less variance in the outcome variable than the first model (adjusted R-squared = 80.4%), while showing no strong indications of multicollinearity among the predictive variables. [(VIF, Tolerance) for City Size: (1.02, 0.98), Obesity: (3.57, 0.28), Lacking Physical Activity: (4.7, 0.21) and Lacking Access to Insurance (2.04, 0.49).]

Table 3. Effects of insurance access, obesity, sedentary behavior (during leisure time) and size of city population (as ordinal variable) on the rate of type 2 diabetes.

	Rate of Type 2 Diabetes	95% Confidence Intervals
Lacking access to health insurance	0.14 ** (0.01) [12.86]	(0.118, 0.161)
Obesity Rate	0.078 ** (0.016) [4.91]	(0.047, 0.109)
Lacking Physical Activity During Leisure Time	0.169 ** (0.017) [9.92]	(0.135, 0.202)
Size of City Population (as ordinal variable)	0.066 ** (0.017) [3.82]	(0.032, 0.1)
Constant/Intercept	0.951 ** (0.261) [3.64]	(0.438, 1.47)

Note: Standard errors appear in parentheses, t-scores appear in brackets. ** $p < 0.001$. Observations: 500. Adj. R-Squared: 0.804 **. F(4, 495): 511.02.

4. Discussion

The results from the regression models clearly indicate that larger cities are associated with significantly higher rates of type 2 diabetes even after controlling for the effects of obesity, sedentary behavior (during leisure time) and access to health insurance. The models explain a large portion (>80%) of the variance, without any robust indication or discernible risk of multicollinearity among the predictors. Given that there were negligible differences between the two models, it is tempting to choose the regression equation that maintained city size as a continuous variable, rather than adhering to an ordinal/ranking system that was less sensitive to measuring population (which was the attribute used to indicate a city's size). However, the levels of skewness (1.82) and kurtosis (4.32) in the distribution for this variable, even after performing a log transformation, were relatively high. Therefore, the second model (using the tier/ranking system) can be viewed as a viable alternative since it provides nearly the same predictive value without similar complications. The second model also validates the concept that some cities may ultimately be quite similar along several key dimensions despite notable differences in the size of their respective populations.

Ultimately, both of these models provide evidence for those who wish to modulate or adjust the traditional paradigm for understanding how and why residents in larger cities report significantly higher rates of type 2 diabetes. The following points (two of which involve concepts that were initially broached in the Background section) are worth considering as part of a more comprehensive and critical analysis of the relationship between urbanized environments and the risk of disease onset.

4.1. The Sedentary Nature of Work

It is important to note that the data provided by the CDC in this study examined the nature of residents' physical activity *during their leisure time* without similar consideration for the implications of extensive sitting or other sedentary behaviors during working hours (see Limitations section). As mentioned, increases in both commuting time (particularly by automobile) as well as the working day have garnered more attention from researchers regarding their potentially negative health effects. Indeed, the meta-analysis from Biswas et al. (cited by Vallance et al.) which revealed a strong association between extensive sitting (>8 hrs. per day) and the risk of developing type 2 diabetes, appears even more compelling given the results from the regression models employed in this analysis.

It is highly plausible that traditional modes of exercise sometimes fail to ameliorate the negative health consequences that arise from the type of sedentary behavior that has proven to be a hallmark of urban occupational life. Indeed, even after accounting for the effects of obesity, as well as access to health care (via insurance), the significantly higher rates of type 2 diabetes among residents of larger cities strongly suggests that their workplace (or work-oriented) behavior poses a greater risk compared to those commuting and working in notably smaller environs.

4.2. Diabetes without Obesity

As listed on the *500 Cities* website, residents (aged 18 and older) are considered obese if their Body Mass Index (BMI) is 30 or greater (See Appendix A). Although CDC researchers acknowledge that simply being overweight, rather than being clinically obese, increases the risk of developing type 2 diabetes, this variable (i.e., being overweight) was not measured. (This does not represent a limitation with the *500 Cities* dataset per se. However, this observation is mentioned in the Limitations section.) In any event, given that the regression models in this study controlled for the effects of sedentary behavior during leisure time, obesity rates and access to health insurance, it becomes all the more prudent to isolate those factors which may be contributing to a (possibly) higher prevalence of residents that are overweight (but not obese) in larger cities. A more conventional epidemiological paradigm may attempt to explain this phenomenon as a result of the aforementioned “food deserts” in conjunction with the ‘Standard American Diet’ (SAD). However, the results in this study demonstrated that small-to medium-sized cities, which generally do not contain any discernible food deserts, still exhibit higher rates of type 2 diabetes compared to the smallest cities. Conversely, the Standard American Diet is not confined to only the largest urban populations [33]. Therefore, the (possibly) greater presence of overweight, diabetic residents living in larger urbanized environments may reinforce two tenets proposed when framing this analysis; more work-related sedentary behavior, in conjunction with time poverty, leaves residents in larger cities at greater risk for becoming overweight and diabetic.

It is also worth noting that some individuals develop type 2 diabetes without becoming overweight or obese [34]. Indeed, researchers have increased their focus on the role of *inflammation* with respect to disease onset, independent of body weight. Studies have identified specific inflammatory “bio-markers” that have been linked to cases of type 2 diabetes after controlling for measures of obesity such as body mass index (BMI) and hip-to-waist ratio, [35,36]. However, given that inflammation remains an integral component in the relationship between obesity and type 2 diabetes [37,38], the task of isolating its effects remains complex and warrants further investigation.

A primary dietary variable involved with the growing literature examining the role of inflammation and the onset of type 2 diabetes has been the intake of sugar [39]. Sugar intake (especially in the form of sugar-sweetened beverages) has also been identified as a causative factor with respect to type 2 diabetes independent of obesity [40]. In an analysis covering 175 countries (utilizing data provided by the United Nations, World Bank, World Health Organization and International Diabetes Federation) researchers revealed that differences in diabetic rates (across countries) that could not be explained by levels of physical activity or obesity rates (including citizens who were simply overweight) were in fact statistically associated with differences in the availability of sugar within a given country’s dietary landscape [41].

A cross-sectional study of the American dietary landscape revealed that excessive levels of sugar intake in the United States were not limited to the consumption of sweetened beverages or fast food, but instead entailed a wide array of ‘ultra-processed’ foods and products [42]. Specifically, researchers determined that ultra-processed foods constituted “more than half of all calories” along with “nearly 90% of all added sugars” found in the American diet ([42], Discussion section, para. 1). Given that this study employed a nationally representative sample, these findings underscore the presence of dietary challenges that are situated well beyond the limited choices that define urban ‘food deserts’. It is also clear that Americans across all socioeconomic strata may be at increased risk for developing type 2 diabetes through sub-optimal diets without necessarily becoming overweight or obese. Therefore, it is quite plausible that higher rates of type 2 diabetes seen in larger cities, after controlling for the effects of obesity, physical activity during leisure time and access to health insurance, are not solely the product of overweight residents who may have been unaccounted for in this analysis.

4.3. Subjects for Future Study

Time poverty. Given the lengthening of commutes, as well as the working day, in many urban occupational settings, residents of larger cities may have significantly less time to engage in a myriad of behaviors associated with healthier, more productive lifestyles (as argued by Williams, Masuda and Tallis). In this light, it is plausible to posit that residents who can be classified as ‘time-poor’ are less likely to schedule routine visits to their primary health care providers and/or avail themselves of other health-oriented services despite being covered by insurance. Time poverty can also be viewed as an obstacle to any behavior that qualifies as a form of stress management, whether it involves the rest and relaxation underscored by Williams et al., or other activities that provide similarly salubrious effects (e.g., physical therapy, meditation, arts and entertainment). Considering the precarious nature of the American dietary landscape (as underscored in the previous section), it is pertinent to note that researchers continue to document a strong association between stress and the consumption of foods that are particularly high in sugar and fat [43,44]. Overall, it appears that any future investigation into the relationship between urbanization and the prevalence of type 2 diabetes may significantly benefit from measures that reveal the presence of time constraints that could undermine city residents’ ability to engage in preventative care. Such analyses could also help cultivate health-oriented strategies that successfully operate within time constraints in a manner that has proven elusive for more conventional programs and interventions to date.

Information and communication technology (ICT) as a means of intervention. The proliferation of information and communication technology (ICT) has prompted researchers to devise new frameworks for understanding how the power of innovation can be applied to urban settings as a means of improving residents’ quality of life. Solanas et al. provide a compelling example through their inception of the “Smart Cities” paradigm; an innovative methodology that capitalizes on evolving urban infrastructure to provide healthcare services beyond more traditional, centralized facilities, thereby reaching more residents more consistently, and enhancing the ability of patients to claim more ownership and control over their efforts to stave off disease [45].

Through this paradigm, the authors highlight the ability of ICT to facilitate “a variety of health-related tasks, including communication between patients, doctors and carers; distant provision of care; remote support to electronic diagnostic medical records; [and] medication adherence control” in a fashion that “can significantly contribute to the reduction of management costs and increased efficiency.” [45] (p.75) Interventions using ICT appear particularly well suited to addressing the etiology of type 2 diabetes, specifically through its ability to closely monitor residents’ lifestyles and health status in a relatively unobtrusive fashion. The reduced costs and improved efficiency of such interventions could also broaden access for residents, including those who currently lack health insurance (often due to financial constraints). Easy and continual access through ICT may also serve to circumvent persistent time constraints (i.e., the logistical barriers that arise for urban residents who could be classified as ‘time-poor’). Clearly, future investigations could benefit from including information technology as a treatment modality

that can be integrated into various urban settings. Solanas et al. highlight the use of sensors that can be strategically placed throughout cities to provide their residents with a bevy of pertinent (i.e., health-related) variables to help gauge and possibly modify their daily routines (e.g., alerting residents to the presence of allergens, smog/air pollution, noxious traffic conditions, etc.) [45]. Investigators considering expanding on these initiatives can be encouraged by the emerging literature supporting the use of ICT to develop more effective initiatives to combat the onset of type 2 diabetes.

For example, in a randomized controlled trial, Block et al. tested the effectiveness of a behavioral intervention program, designed for pre-diabetic subjects, that targeted an array of health-related behaviors (ranging from exercise and diet to stress management and sleep.) [46]. Through a fully automated system, subjects received their individually tailored plans, derived by algorithm, via the Internet (e.g., receiving weekly e-mails and accessing individualized web pages that tracked their participation). The program used mobile phone applications and automated phone calls (using interactive voice response (IVR) technology) to provide additional support. Participants in the intervention group showed significant improvement across an array of clinical measures, such as Body Mass Index (BMI), waist circumference, and TG/HDL (triglyceride to hi-density lipoprotein cholesterol) ratio. The authors underscored that this program's effectiveness, combined with its affordability, made such interventions highly scalable; a vital attribute given that 1 in 3 (i.e., 86 million) adults in the US can be classified as pre-diabetic. Another investigation similarly reported positive results in terms of clinical success and cost effectiveness for ICT-based interventions designed to augment the Diabetes Prevention Recognition Program created by the Centers for Disease Control and Prevention [47].

A meta-analysis covering 32 different intervention trials revealed that those employing information technology were significantly more likely to lower patients' levels of hemoglobin A_{1c}, a key indicator of glycemic control [48]. Researchers also noted generally positive results in a systematic review of health interventions conducted in developing countries that exclusively used mobile phones [49]. However, the authors cautioned their analysis was based on a fairly small number of studies with sufficient methodological rigor, and included interventions for diseases besides diabetes (e.g., asthma).

Overall, the inclusion of ICT in any future investigation not only holds tremendous promise for addressing the financial and logistical constraints that can impede or stymie urban residents seeking routine care, it recognizes the sea change that Solanas et al. and others have cited with respect to an increasingly de-centralized, data-driven and user-oriented healthcare system. Indeed, the burgeoning field of healthcare informatics appears ideally suited for providing more personalized and user-oriented treatment plans. Systems that continually adapt to an individual's lifestyle, whether at home or in the workplace, will invariably be more sensitive to the types of health changes (which are often quite subtle) that indicate an elevated risk for developing type 2 diabetes, among other chronic illnesses. Employers may also be keen to utilize this technology on-site to address the potentially deleterious effects from the sedentary nature of work, limited dietary options and/or occupational stress. Although social commentators may lament the near ubiquity of ICT, particularly as it relates to privacy concerns, the emerging networks that enable more communication and interaction with medical professionals outside of the traditional "brick-and-mortar" apparatus remain a largely untapped, and potentially vital, resource.

4.4. Limitations

As mentioned, two key variables from the *500 Cities* dataset were operationalized in a manner that may have prevented a more comprehensive understanding of their influence over the prevalence of type 2 diabetes. First, sedentary behavior was only ascertained based on how residents reported spending their leisure time (rather than including their working day). In addition, as stated on the *500 Cities* website, a resident's level of physical activity was measured by a simple "yes/no" response to the following question: "During the past month, other than your regular job, did you participate in any physical activities or exercises such as running, calisthenics, golf, gardening or walking for exercise?" This question is rather diffuse and lacks the necessary precision to accurately determine the level of physical activity of

residents that responded in the affirmative. It is highly plausible that an inordinate amount of variance exists among respondents who reported being physically active, thereby undermining the ability of the dataset to illustrate how physical activity can help stave off disease.

The second variable, as mentioned in the Discussion section, involves measuring obesity rates (i.e., the percentages of residents with a BMI ≥ 30) without a similar accounting of residents who are overweight, (BMI ≥ 25). This occurred despite the CDC acknowledging on the *500 Cities* website that both conditions have been identified as precursors to type 2 diabetes. Although this limitation may be mitigated when considering the methodology employed in this analysis (see Discussion section), any future studies in this area would optimally contain data that documents the percentage of residents that are overweight, as well as obese, in a given municipality.

Another limitation can be noted when considering the measures used to ascertain residents' ability to access health insurance. Given the presence of Medicare, there is a natural ceiling with respect to the age of adults who are unable to gain access to insurance. Consequently, this variable was measured among residents ages 18 to 64, with the remaining variables being measured among all residents 18 and older. Rather than reflecting a limitation within the *500 Cities* dataset, this difference poses more of a statistical dilemma, however minor, for the regression models employed in this analysis. Despite this difference among measures, the results garnered in this study still provide robust evidence that shows cities with a greater percentage of residents lacking access to health insurance suffering significantly higher rates of type 2 diabetes, even after accounting for all other pertinent variables. This observation is likely bolstered by the presence of longtime residents whose health was negatively affected by their inability to access health insurance before they became eligible for Medicare.

Indeed, the subject of age reveals a broader limitation given the general lack of demographic information provided by this dataset. The opportunity to explore differences in key variables by gender, ethnicity, education level or income would have provided more opportunities for analysis, and yielded a more intricate portrait of the challenges faced with respect to particular population segments. The inclusion of age (to some degree) remains an exception, but also underscores how the information gathered by this project involved only adults, a lamentable point considering longstanding concerns involving childhood obesity among other related challenges regarding the prevalence of type 2 diabetes.

Although demographic variables such as income were not included in this dataset, the degree to which residents reported having access to health insurance can provide some insight into their financial status. According to the Kaiser Family Foundation, the high cost of coverage is one of the primary reasons for being uninsured [50]. However, the question ascertaining whether residents were covered by health insurance may suffer the same methodological pitfall as the item measuring levels of physical activity during leisure time. A simple "yes/no" response to being asked whether a given individual has current health insurance fails to take into account the vast array of plans and services (and their associated costs) provided by the insurer. Indeed, even with such transformative legislation as the Affordable Care Act (ACA), which provided coverage for millions of U.S. residents who were previously uninsured, large disparities remain among the various plans, particularly with respect to the size of deductibles, among other burdens incurred by patients.

Lastly, it is important to consider that the regression models employed in this analysis represent only two out of potentially several models that could be constructed in a methodologically sound and meaningful fashion using this dataset. It is conceivable that other models (which ostensibly would be just as statistically viable with comparable levels of predictive power) would not similarly highlight the relevance of city size as an independent variable as it relates to the prevalence of type 2 diabetes. However, rather than viewing this exclusively as a potential limitation, it is important to acknowledge that the model used in this study revealed a more profound phenomenon which would likely be captured by *any* sound regression model regardless of its emphasis (if any) on city size; specifically, the preponderance of variance in the prevalence of type 2 diabetes that remains unexplained in larger urbanized environments even after accounting and controlling for variables (e.g., obesity) that have been traditionally linked to disease onset.

5. Conclusions

As rates of type 2 diabetes continue to rise, so too the demand for more innovative strategies to combat this pervasive, and seemingly intractable, public health conundrum. The findings from this study strongly suggest that urbanized environments are contributing to this crisis in a fashion that cannot be readily explained by the conventional epidemiological paradigms, particularly those that embrace a somewhat myopic methodological approach that is unduly preoccupied with the presence of food deserts and the excessive caloric content of the 'Standard American Diet'. However, it is equally important to consider that the points of intervention proposed in this study represent a pragmatic, rather than radical, re-assessment of how diet, exercise and healthcare can be successfully managed in the American urban landscape of the 21st century. For example, it is abundantly clear that the sedentary nature of work deserves far greater scrutiny. It is also apparent that having access to health insurance does not ensure that all appropriate medical services are similarly accessed and utilized by the insured. Indeed, simply asking whether a given individual has access to health insurance (i.e., framing this as a dichotomous rather than continuous variable) is probably unwise, as there is considerable variance in the type and levels of care afforded to residents based on their respective carriers and insurance plans. Finally, there is likely little to be gained from any public health initiative that does not directly address the potentially pernicious effects of *time poverty*; a condition that is likely endured by a preponderance of urban residents regardless of their socioeconomic status. Even as technology designed to work within constricted schedules continues to evolve, individuals who spend a disproportionate amount of their waking hours meeting the demands of urban life, be they financial or logistical, may still be less likely to engage in personal and professional care regardless of the time-saving devices at their disposal. Reframing time as a resource on par with income may instead foster strategies that seek to complement rather than defer to innovation. In essence, allowing individuals more time to achieve their health objectives may prove more successful and sustainable than encouraging their use of more technology to cope with an increasingly demanding lifestyle.

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Appendix A

Table A1. Definitions of variables examined in regression analyses *.

Variable	Definition as Listed on 500 Cities Website	Respondents *	Numerator *	Denominator *
Lacking physical activity during leisure time	No leisure-time physical activity among adults aged 18 years and older	Resident adults aged 18 years and older	Respondents who answered no to the following question: "During the past month, other than your regular job, did you participate in any physical activities or exercises such as running, calisthenics, golf, gardening, or walking for exercise?"	Number of adults aged ≥ 18 years who reported any or no physical activity in the past month (excluding those who refused to answer, had a missing answer, or answered "don't know/not sure").
Obesity	Obesity among adults aged 18 years and older	Resident adults aged 18 years and older	Respondents aged ≥ 18 years who have a body mass index (BMI) ≥ 30.0 kg/m² calculated from self-reported weight and height. Excluding the following: • Height: data from respondents measuring <3 ft or ≥ 8 ft • Weight: data from respondents weighing <50 lbs or ≥ 650 lbs • BMI: data from respondents with BMI <12 kg/m² ≥ 100 kg/m² • Pregnant women	Respondents aged ≥ 18 years for whom BMI can be calculated from their self-reported weight and height (excluding unknowns, refusals to provide weight or height and exclusions listed below): • Height: data from respondents measuring <3 ft or ≥ 8 ft • Weight: data from respondents weighing <50 lbs or ≥ 650 lbs • BMI: data from respondents with BMI <12 kg/m² ≥ 100 kg/m² • Pregnant women
Lacking access to health insurance	Current lack of health insurance among adults aged 18–64 years	Resident adults aged 18–64 years.	Respondents aged 18–64 years who report having no current health insurance coverage.	Respondents aged 18–64 years who report having current health insurance or having no current health insurance (excluding those who refused to answer, had a missing answer, or answered "don't know/not sure").
Type 2 Diabetes	Diagnosed diabetes among adults aged 18 and older	Resident adults aged 18 years and older	Respondents aged ≥ 18 years who report ever been told by a doctor, nurse, or other health professional that they have diabetes other than diabetes during pregnancy.	Respondents aged ≥ 18 years who report or do not report ever been told by a doctor, nurse, or other health professional that they have diabetes (excluding those who refused to answer, had a missing answer, or answered "don't know/not sure").

* Definitions and terminology provided by 500 Cities: Local Data for Better Health website. <https://www.cdc.gov/500cities/measure-definitions.htm>.

References

1. Fei, Y.; He, Y.; Sun, L.; Chen, J.; Lou, Q.; Bao, L.; Cha, J. The study of diabetes prevalence and related risk factors in Fuyang, a Chinese county under rapid urbanization. *Int. J. Diabetes Dev. Ctries.* **2016**, *36*, 213–219. [CrossRef]
2. Cheema, A.; Adeloye, D.; Sidhu, S.; Sridhar, D.; Chan, K. Urbanization and prevalence of type 2 diabetes in Southern Asia: A systematic analysis. *J. Glob. Health* **2014**, *4*, 1–20. [CrossRef] [PubMed]
3. Ramachandran, A.; Mary, S.; Yamuna, A.; Murugesan, N.; Snehathatha, C. High prevalence of diabetes and cardiovascular risk factors association with urbanization in India. *Diabetes Care* **2008**, *31*, 893–898. [CrossRef] [PubMed]
4. UN-Habitat—Global Report on Urban Health; Equitable, Healthier Cities for Sustainable Development. Available online: <https://unhabitat.org/books/global-report-on-urban-health-equitable-healthier-cities-for-sustainable-development/> (accessed on 1 October 2018).
5. Gassasse, Z.; Smith, D.; Finer, S.; Gallo, V. Association between urbanization and type 2 diabetes: An ecological study. *BMJ Glob. Health* **2017**, *2*, e000437. [CrossRef]
6. Garden, F.; Jalaludin, B. Impact of urban sprawl on overweight, obesity and physical activity in Sydney, Australia. *J. Urban Health* **2009**, *86*, 19–30. [CrossRef]
7. Oyhenart, E.E.; Castro, L.E.; Forte, L.M.; Sicre, M.L.; Quintero, F.A.; Luis, M.A.; Torres, M.F.; Luna, M.E.; Cesani, M.F.; Orden, A.B. Socio-environmental conditions and nutritional status in urban and rural schoolchildren. *Am. J. Hum. Biol.* **2008**, *20*, 399–405. [CrossRef]
8. Lopez, R. Urban sprawl and risk for being overweight or obese. *Am. J. Public Health* **2004**, *94*, 1574–1579. [CrossRef] [PubMed]
9. Jeffery, R.; Utter, J. The changing environment and population obesity in the United States. *Obes. Res.* **2003**, *11* (Suppl. 10), 12S–22S. [CrossRef]
10. International Diabetes Federation. *Two Thirds of All People with Diabetes Live in Urban Areas*. IDF Diabetes Atlas, 7th ed.; Available online: <https://www.idf.org/e-library/epidemiology-research/diabetes-atlas/13-diabetes-atlas-seventh-edition.html> (accessed on 1 October 2018).
11. Jowell, A.; Zhou, B.; Barry, M. The impact of megacities on health: Preparing for a resilient future. *Lancet Planet. Health* **2017**, *1*, e176–e178. [CrossRef]
12. Prentice, A. The emerging epidemic of obesity in developing countries. *Int. J. Epidemiol.* **2006**, *35*, 93–99. [CrossRef]
13. Singh, G.; Siahpush, M. Widening rural-urban disparities in life expectancy, U.S., 1969–2009. *Am. J. Prev. Med.* **2014**, *46*, e19–e29. [CrossRef] [PubMed]
14. Houston's Population Keeps Popping, but Growth is Slowing. Available online: www.bizjournals.com/houston/news/2018/03/23/houstons-population-keeps-popping-but-growth-is.html (accessed on 10 November 2018).
15. 500 Cities: Local Data for Better Health. Available online: <https://www.cdc.gov/500cities/about.htm> (accessed on 1 August 2018).
16. Dart, T. (11 February 2016). Houston's Health Crisis: By 2040, one in Five Residents Will Be Diabetic. Available online: <https://www.theguardian.com/cities/2016/feb/11/houstonhealth-crisis-diabetes-sugar-cars-diabetic> (accessed on 1 November 2018).
17. Christafore, D.; Leguizamon, S. Neighborhood inequality spillover effect of gentrification. *Pap. Reg. Sci.* **2018**. [CrossRef]
18. Cities Changing Diabetes—Diabetes in Houston. Available online: <http://www.citieschangingdiabetes.com/cities/houston.html> (accessed on 1 October 2018).
19. Ghosh-Dastidar, B.; Cohen, D.; Hunter, G.; Zenk, S.N.; Huang, C.; Beckman, R.; Dubowitz, T. Distance to Store, Food Prices and Obesity in Urban Food Deserts. *Am. J. Prev. Med.* **2014**, *47*, 587–595. [CrossRef]
20. Williams, J.; Masuda, Y.; Tallis, H. A measure whose time has come: Formalizing time poverty. *Soc. Indic. Res.* **2016**, *128*, 265–283. [CrossRef]
21. Stone, A.; Schneider, S. Commuting episodes in the United States: Their correlates with experiential wellbeing from the American Time Use Survey. *Transp. Res. Part F Traffic Psychol. Behav.* **2016**, *42*, 117–124. [CrossRef]
22. Johnson, J.; Lipscomb, J. Long working hours, occupational health and the changing nature of work organization. *Am. J. Ind. Med.* **2006**, *49*, 921–929. [CrossRef] [PubMed]

23. Tin-chi, L.; Courtney, T.; Lombardi, D.; Verma, S. Association between sedentary work and BMI in a U.S. national longitudinal survey. *Am. J. Prev. Med.* **2015**, *49*, e117–e123.
24. Tigbe, W.; Granat, M.; Satter, N.; Jean, M. Time spent in sedentary posture is associated with waist circumference and cardiovascular risk. *Int. J. Obes.* **2017**, *41*, 689–696. [CrossRef] [PubMed]
25. Parry, S.; Straker, L. The contribution of office work to sedentary behaviour and associated risk. *BMC Public Health* **2013**, *13*, 1–10. [CrossRef]
26. Vallance, J.K.; Gardiner, P.A.; Lynch, B.M.; D'Silva, A.; Boyle, T.; Taylor, L.M.; Johnson, S.T.; Buman, M.P.; Owen, N. Evaluating the evidence on sitting, smoking and health: Is sitting really the new smoking? *Am. J. Public Health* **2018**, *108*, 1478–1482. [CrossRef]
27. Biswas, A.; Oh, P.I.; Faulkner, G.E.; Bajaj, R.R.; Silver, M.A.; Mitchell, M.S.; Alter, D.A. Sedentary time and its association with risk for disease incidence, mortality, and hospitalization in adults: A systematic review and meta-analysis. *Ann. Intern. Med.* **2015**, *162*, 123–132. [CrossRef]
28. Field, A. *An Adventure in Statistics: The Reality Enigma*, 1st ed.; Sage Publications: London, UK, 2016.
29. What Is Your City's Twin? Available online: <https://www.nytimes.com/interactive/2018/04/03/upshot/what-is-your-citys-twin.html> (accessed on 1 October 2018).
30. Maggio, C.; Pi-Sunyer, F. Obesity and type 2 diabetes. *Endocrinol. Metab. Clin. N. Am.* **2003**, *32*, 805–822. [CrossRef]
31. Hu, F.; Tricia, Y.; Colditz, G.; Walter, W.; Manson, J. Television Watching and Other Sedentary Behaviors in Relation to Risk of Obesity and Type 2 Diabetes Mellitus in Women. *JAMA* **2003**, *289*, 1785–1791. [CrossRef]
32. Boslaugh, S. *Statistics in a Nutshell*, 2nd ed.; O'Reilly Media: Sebastopol, CA, USA, 2013.
33. Kannan, V.; Veazie, P. Political orientation, political environment and health behaviors in the United States. *Prev. Med.* **2018**, *114*, 95–101. [CrossRef] [PubMed]
34. Mayo Clinic, Type 2 Diabetes. Available online: <https://www.mayoclinic.org/diseases-conditions/type-2-diabetes/symptoms-causes/syc-20351193> (accessed on 1 November 2018).
35. Pickup, J. Inflammation and activated innate immunity in the pathogenesis of type 2 diabetes. *Diabetes Care* **2004**, *27*, 813–823. [CrossRef]
36. Duncan, B.B.; Schmidt, M.I.; Pankow, J.S.; Ballantyne, C.M.; Couper, D.; Vigo, A.; Hoogeveen, R.; Folsom, A.R.; Heiss, G. Low-grade systemic inflammation and the development of type 2 diabetes. The Atherosclerosis Risk in Communities Study. *Diabetes* **2003**, *52*, 1799–1805. [CrossRef] [PubMed]
37. Esser, N.; Legrand-Poels, S.; Piette, J.; Scheen, A.J.; Paquot, N. Inflammation as a link between obesity, metabolic syndrome and type 2 diabetes. *Diabetes Res. Clin. Pract.* **2014**, *105*, 141–150. [CrossRef]
38. Xu, H.; Barnes, G.T.; Yang, Q.; Tan, G.; Yang, D.; Chou, C.; Sole, J.; Nichols, A.; Ross, J.S.; Tartaglia, L.A.; et al. Chronic inflammation in fat plays a crucial role in the development of obesity-related insulin resistance. *J. Clin. Invest.* **2003**, *112*, 1821–1830. [CrossRef]
39. Schulze, M.B.; Hoffmann, K.; Manson, J.E.; Willett, W.W.; Meigs, J.B.; Wiekert, C.; Heidemann, C.; Colditz, G.A.; Hu, F.B. Dietary pattern, inflammation, and incidence of type 2 diabetes in women. *Am. J. Clin. Nutr.* **2005**, *82*, 675–684. [CrossRef]
40. Malik, V.; Hu, F. Sweeteners and risk of obesity and type 2 diabetes: The role of sugar-sweetened beverages. *Curr. Diab. Rep.* **2012**, *12*, 195–203. [CrossRef] [PubMed]
41. Basu, S.; Yoffe, P.; Hills, N.; Lustig, R.H. The relationship of sugar to population-level diabetes prevalence: An econometric analysis of repeated cross-sectional data. *PLoS ONE* **2013**, *8*, e57873. [CrossRef]
42. Steele, E.M.; Baraldi, L.G.; da Costa Louzada, M.L.; Moubarac, J.; Mozaffarian, D.; Monteiro, C.A. Ultra-processed foods and added sugars in the US diet: Evidence from a nationally representative cross-sectional study. *BMJ Open* **2016**, *6*, e009982.
43. Sinha, R.; Jastreboff, A. Stress as a common risk factor for obesity and addiction. *Biol. Psychiatry* **2013**, *73*, 827–835. [CrossRef]
44. Torres, S.; Nowson, C. Relationship between stress, eating behavior and obesity. *Nutrition* **2007**, *23*, 887–894. [CrossRef]
45. Solanas, A.; Patsakis, C.; Conti, M.; Vlachos, I.S.; Ramos, V.; Falcone, F.; Postolache, O.; Perez-Martinez, P.A.; Di Pietro, R.; Perrea, D.N.; et al. Smart health: A context-aware health paradigm within smart cities. *IEEE Commun. Mag.* **2014**, *52*, 74–81. [CrossRef]

46. Block, G.; Azar, K.M.; Romanelli, R.J.; Block, T.J.; Hopkins, D.; Carpenter, H.A.; Dolginsky, M.S.; Hudes, M.L.; Palaniappan, L.P.; Block, C.H. Diabetes prevention and weight loss with a fully automated behavioral intervention by e-mail, web and mobile phone: A randomized control trial among persons with prediabetes. *J. Med. Internet Res.* **2015**, *17*, e240. [[CrossRef](#)]
47. Su, W.; Chen, F.; Dall, T.M.; Iacobucci, W.; Perreault, L. Return on investment for digital behavioral counseling in patients with prediabetes and cardiovascular disease. *Prev. Chronic Dis.* **2016**, *13*, e13. [[CrossRef](#)]
48. Alharbi, N.S.; Alsubki, N.; Jones, S.; Khunti, K.; Munro, N.; de Lusignan, S. Impact of information-technology-based interventions for type 2 diabetes mellitus on glycemic control: A systematic review and meta-analysis. *J. Med. Internet Res.* **2016**, *18*, e310. [[CrossRef](#)]
49. Stephani, V.; Opoku, D.; Quentin, W. A systematic review of randomized controlled trials of mHealth interventions against non-communicable diseases in developing countries. *BMC Public Health* **2016**, *16*, 572. [[CrossRef](#)]
50. Kaiser Family Foundation. Key Facts about the Uninsured Population. Available online: <https://www.kff.org/uninsured/fact-sheet/key-factsabout-the-uninsured-population/> (accessed on 1 November 2018).



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