PLOS COMPLEX SYSTEMS

RESEARCH ARTICLE

Investigating the link between impulsivity and obesity through urban scaling laws

Tian Gan[1,2], **Rayan Succar**[1,2], **Simone Macrì**[3*], **Maurizio Porfiri**[1,2,4*]

1 Department of Mechanical and Aerospace Engineering, Tandon School of Engineering, New York University, Brooklyn, New York, United States of America, **2** Center for Urban Science and Progress, Tandon School of Engineering, New York University, Brooklyn, New York, United States of America, **3** Centre for Behavioural Sciences and Mental Health, Istituto Superiore di Sanità, Rome, Italy, **4** Department of Biomedical Engineering, Tandon School of Engineering, New York University, Brooklyn, New York, United States of America

* simone.macri@iss.it (SM); mporfiri@nyu.edu (MP)

Citation: Gan T, Succar R, Macrì S, Porfiri M. (2025) Investigating the link between impulsivity and obesity through urban scaling laws. PLoS Complex Sys 2(5): e0000046. https://doi.org/10.1371/journal.pcsy.0000046

Editor: Marcos Oliveira, University of Exeter Faculty of Environment Science and Economy, UNITED KINGDOM OF GREAT BRITAIN AND NORTHERN IRELAND

Received: October 12, 2024

Accepted: April 3, 2025

Published: May 15, 2025

Data availability statement: All data is available at https://github.com/dynamicalsystemslaboratory/Impulsivity_Obesity.git.

Funding: This work was supported by the National Science Foundation (ECCS-1928614, DUE-2129076, and EF-2222418 to M.P.) and the European Union's Horizon 2020 research and innovation programme (847879 to S.M.). The funders had no role in study design, data

Abstract

Impulsivity has been proposed as a key driver of obesity. However, evidence linking impulsivity and obesity has relied on the study of individual factors, with limited account for the urban attributes of obesogenic environments. Here, we investigate the relationship between obesity and impulsivity through urban scaling and causal discovery. For 915 cities in the United States of America, we study the prevalence of obesity in adults, attention deficit hyperactivity disorder (ADHD) in children, and relevant urban features. We observe sublinear scaling of obesity and ADHD with population size, these disorders being less prevalent in larger cities. By applying a causal discovery tool to the deviations of cities from the urban scaling laws, we identify an influence of ADHD on obesity, moderated by lifestyle. The strength of these associations is confirmed by individual-level data on a cohort of 19,333 children, wherein we observe that ADHD modulates obesity both directly and indirectly.

Author summary

Is a lack of impulse control related to weight gain? Extant epidemiological, clinical, and preclinical research has provided a positive answer to this question and helped detail some of the fundamental mechanisms underlying this association. Yet, these studies assume that we all live in an identical city, thereby neglecting the specific role of urban features on the link between impulsivity and obesity. Here, we explore this link through the lens of urban scaling laws, putting forward a causality framework for urban research, inspired by Judea Pearl's work. Our analysis reveals that urban lifestyle modulates the relationship between obesity and impulsivity. These findings underscore the importance of city-level interventions in mitigating the impact of impulsivity disorders on the obesity epidemic.

collection and analysis, decision to publish, or preparation of the manuscript.

Competing interests: The authors have declared that no competing interests exist.

Introduction

The escalating prevalence of obesity is a dire global health priority [1,2]. For example, in the United States of America (USA), the societal and personal costs of obesity are substantial, with a projected increase in prevalence from 15% in 2010 to 26% by 2030 [3]. Its etiology involves multiple factors, such as behavior, genetics, age, diet, and environment [4–6].

Among the candidate factors modulating overweight and obesity, impulsivity – the tendency towards unplanned decision-making regardless of potential consequences [7] – has been proposed as a pivotal one [8]. Impulsivity constitutes a fundamental and evolutionarily adaptive phenomenon [9] through which individuals may identify advantageous strategies under time pressure, have a better performance than non-impulsive subjects when rewards are abundant, or obtain some benefits in field such as entrepreneurship, literature and arts [10]. Despite its adaptive meaning, excess impulsivity may often lead to inaccurate choices and potentially negative outcomes. Within this framework, impulsivity extends to food choices (emotional and overeating) that lead to overweight and obesity [11]. Several lines of evidence support this proposition. A comprehensive epidemiological investigation reported a robust association between impulsivity and weight status [12]. Meta-analyses strengthen this finding by identifying a direct association between pathological impulsivity, in the form of attention deficit hyperactivity disorder (ADHD), and obesity [13,14]. Laboratory studies also revealed that higher impulsivity correlates with overeating in healthy individuals [15]. Genome-wide association studies identified a genetic overlap between ADHD and obesity [16]. Last, anti-ADHD drugs were shown to reduce obesity [17]. Therefore, in the present study, to operationalize the study of the association between pathological impulsivity and aberrant weight gain, we collected data on ADHD and obesity.

The multilevel investigation of obesity and impulsivity points towards a dynamic process in which genetic predispositions and environmental factors act in synergy to affect physical and mental health. Individual factors alone cannot explain obesity prevalence. Previous evidence supports the notion that obesity is the resultant of an intricate combination of genetic and environmental factors. The heuristic value of this information primarily pertains to approaches aimed at normalizing body weight at an individual level (microscale). Yet, obesity can also be considered a global phenomenon, to which macroscale factors (*e.g.,* obesogenic environments) may play a substantial role and for which large-scale prevention/mitigation strategies should be devised and implemented. In order to manage obesity via macroscale approaches, we need to first understand whether and how factors such as culture, social economy, and environment influence the onset of this global pandemic [18]. The urban attributes of obesogenic environments include opportunities for physical activity, access to medication, access to healthy food, and social ties [19–22]. These attributes may vary greatly in different countries, thereby shaping widely different landscapes of obesity worldwide. For example, comparing more urbanised to less urbanised areas, obesity is more prevalent in the latter for China [23] and in the former for USA [24]. Our work aims at identifying whether specific features (*e.g.*, presence of mental healthcare professionals, access to physical activity, college education, and food insecurity) of given environments act as moderators of obesity. Importantly, we focus on candidate moderators that are theoretically modifiable through large-scale approaches.

We study the relationship between obesity and impulsivity through the lens of urban science – the multi/transdisciplinary field that unveils the fundamental mechanisms underpinning urbanisation [25]. The growth in the availability of geo-localized open data about health and urban living, along with recent advances in data science, enables targeted investigations into "the relation of the urban living environment to brain, behavior and mental health [carrying] great promise for effective prevention and targeted early interventions, which will benefit millions." [26]. Urban science harnesses the power of large-scale observational urban data at a population level and integrates them with state-of-art data science techniques towards interpretable models [26–28].

We focus on urban scaling laws, which have been instrumental to understand the effects of urbanisation on infrastructural and socio-economic features of cities, from CO_2 emissions [29] to criminality [30]. Urban scaling laws quantify how specific urban features vary with population size, similar to allometric scaling in biology that describes how certain characteristics of living organisms change with size [31]. Recent work has also highlighted the value of urban scaling in the study of communicable and non-communicable diseases [32]. For example, Cruz et al. [33] reported superlinear (sublinear) scaling of COVID-19 cases (death) in the USA. Rocha et al. [34] determined sublinear scaling of obesity with population size in both Sweden and the USA, so that obesity is proportionally less prevalent in larger cities. Stier et al.[35] found that depression scales sublinearly with population size in USA cities, defeating the common belief that cities are detrimental to mental health. We apply urban scaling to study how the prevalence of obesity and impulsivity vary with respect to the city population. In the analysis, we also address scaling behavior of lifestyle, education, and access to healthy nutritional diet and mental health care, which have been proposed as potential modulators of obesity, impulsivity, and their association [36–38].

Urban scaling provides important insight into the relationship between the population of a city and the prevalence of given disorders. Yet, it is not meant to identify potentially causal associations. To overcome this limitation, in a recent effort, we devised a new methodology to discover causal associations between urban features, demonstrated in the context of firearm ownership, violence, and accessibility [39]. The methodology takes as input scale-adjusted metropolitan indicators (SAMIs) – the difference between a given city's observed trait and the expected trait based on urban scaling laws [40] – and it outputs a directed acyclic graph (DAG) among the selected features, encapsulating directional interactions [41]. We leverage this methodology in the study of the link between impulsivity and obesity, contributing to the "conceptual framework for future urban mental health research that uses a complexity science approach" laid out by [42]. Specifically, we investigate the link between obesity and impulsivity (ADHD) in 915 cities in the USA, accounting for lifestyle, education, and access to healthy nutritional diet and mental health care. While our primary aim was to identify potential causal associations between macroscale modifiable factors and obesity, we also sought to test the validity of our findings using data from a well-defined population with individual-level information on education, physical activity, mental health, and food insecurity. To this end, we assessed the robustness of the observed associations at the individual level, by conducting an analogous analysis on a cohort of 19,333 individuals for which matched data on the aforementioned variables were available. Importantly, we acknowledge that the datasets used in this study differ significantly, having been collected through distinct methodologies. While such differences may result in differential outcomes, the identification of common patterns across independent datasets may strengthen the generalizability of our findings.

Results

Our approach entails a multilevel analysis that spanned from general trends at a city level to granular cause-effect relationships at an individual level. Specifically, our study unfolds along three sequential methodological steps performed on publicly available data, see Tables 1 and 2. First, through urban scaling, we investigated the prevalence of obesity, ADHD, and salient urban features as a function of the size of a city (collating data from cities in the USA,

Table 1. City-level dataset. List of variables considered in the current study for city-level analyses, together with their acronyms, the levels attainable by each variable, and data source.

Variable	Level	Data source
Prevalence of obesity (*OB*)	Number of adults reporting BMI ≥ 30 kg/m^2	2015 County Health Rankings [65]
Prevalence of ADHD (*ADHD*)	Number of ADHD children	Zgodic et al. [66]
Access to mental health providers (*MHP*)	Number of mental health providers located within a city	2015 County Health Rankings [65]
Prevalence of physical inactivity (*PA*)	Number of adults aged 20 and over reporting no leisure-time physical activity	2015 County Health Rankings [65]
Prevalence of food insecurity (*FI*)	Number of adults who lack adequate access to food	2015 County Health Rankings [65]
Prevalence of college education (*CE*)	Number of adults aged 25-44 with some post-secondary education	2015 County Health Rankings [65]

https://doi.org/10.1371/journal.pcsy.0000046.t001

Table 2. Individual-level dataset. List of variables considered in the current study for individual-level analyses, together with their acronyms, the levels attainable by each variable. This dataset is obtained from the 2021 National Survey of Children's Health [68], a nationwide survey that screens households with children under 18 years of age.

Variable	Level
BMI (*BMI*)	1 = "Underweight"
	2 = "Healthy weight"
	3 = "Overweight"
	4 = "Obese"
ADHD severity (*SEV*)	1 = "Does not currently have ADHD"
	2 = "Current ADHD rated mild"
	3 = "Current ADHD rated moderate or severe"
Physical activity (*PA*)	1 = "0 days"
	2 = "1-3 days"
	3 = "4-6 days"
	4 = "7 days"
Food insufficiency (*FINS*)	1 = "Could always afford to eat good nutritious meals"
	2 = "Could afford enough to eat but not always the kinds of food should eat"
	3 = "Sometimes could not afford enough to eat"
	4 = "Often could not afford enough to eat"
Adult education (*EDU*)	1 = "Less than high school"
	2 = "High school or GED"
	3 = "Some college or technical school"
	4 = "College degree or higher"
Use of mental health care services (*UMH*)	1 = "Not received mental health care"
	2 = "Received mental health care"

https://doi.org/10.1371/journal.pcsy.0000046.t002

ranging from 10,339 to 19,220,403 residents). Second, we studied associations among the SAMIs of these variables through a DAG. Last, we tested the strength of these associations through the analysis of an independent cohort, for which data on severity of ADHD diagnoses, body mass index (BMI), and salient features analogous to those chosen for the city-level analysis were available.

City-level analysis: Urban scaling

Urban scaling laws help identify nuanced relationships between population size and city-specific features, from gross domestic product to criminality [43]. To identify city-level moderators of the potential relationship between ADHD and obesity, we first conducted urban scaling analysis on 915 cities with respect to the following six features: prevalence of adult obesity, prevalence of leisure-time physical inactivity, access to mental health providers, college education, prevalence of food insecurity among children, and prevalence of ADHD among children (see Table 1 and Methods). For each of the features, we fitted an urban scaling law of the form

$$Y_i = Y_0 N_i^{\beta} e^{\xi_i}, \tag{1}$$

where i identifies a city, Y_i is the urban feature under investigation, N_i is the city population, β is the scaling exponent, Y_0 represents the common baseline, and ξ_i encapsulates the deviation of the city feature from the common scaling law (so-called scale-adjusted metropolitan indicators, SAMIs [44]). For logarithmically transformed data, the scaling law reduces to a linear model which can be simply fit as a linear regression (see Methods). The slope of the linear model provides the scaling exponent β, where $\beta = 1, \beta > 1$, and $\beta < 1$ describe, respectively, linear, superlinear, and sublinear behaviors.

Superlinear scaling of a certain feature means that, on average, the feature is more abundant in larger cities, even when considering it on a per capita basis. A well-known example of this phenomenon is city income, which exhibits superlinear scaling so that the income per capita is, on average, higher in larger cities. Conversely, sublinear scaling signifies that a feature is more abundant in smaller cities. For example, road surface area exhibits sublinear scaling, where the road surface area per capita is higher in smaller cities.

Urban scaling analysis for the six urban features of interest is presented in Fig 1. The prevalence of obesity in adults scales sublinearly with city population ($\beta = 0.97$ with 95% confidence interval, CI, [0.96,0.98]), indicating that per-capita prevalence decreases with increasing population, see Fig 1A. Likewise, the prevalence of children with ADHD, and leisure-time physical inactivity, scale sublinearly with population ($\beta = 0.98$ with 95% CI [0.97,0.99]; $\beta = 0.95$ with 95% CI [0.94,0.96], respectively), see Fig 1B and Fig 1C, respectively. While scaling exponents of these features suggest a near-linear relationship [45], when looking at a country like the USA where city populations vary in size by three orders of magnitude, even a 0.05 deviation from linearity could be salient. For example, the scaling exponent of 0.95 for physical inactivity implies that the probability that a person is physical inactive in a large city (10^8 residents) is 70% of the probability in a small city (10^5 residents). Conversely, the relationship between access to mental health providers and population exhibits superlinear scaling ($\beta = 1.17$ with 95% CI [1.13,1.21]), see Fig 1D. The prevalence of adults with post-secondary education scale superlinearly ($\beta = 1.08$ with 95% CI [1.07,1.08]), see Fig 1E. The prevalence of food-insecurity scales linearly with population ($\beta = 1$ with 95% CI [0.99,1.01]), see Fig 1F.

Cities within a state exhibit significant disparities in various features, as highlighted by the inequality analysis in Fig 2. Over 80% states have a greater than 0.2 Gini coefficient in the prevalence of ADHD in children, leisure-time physical inactivity, and food insecurity. The prevalence of adults with post-secondary education is distributed unequally ($G \geq 0.2$) over all the states. The vast majority of states have inequality in the prevalence of obesity in adults (except Hawaii with $G = 0.172$), and access to mental health providers (except Vermont and Maine with $G = 0.157$ and $G = 0.029$, respectively). These figures provide a critical context for interpreting the heterogeneity across cities within states. This heterogeneity brings challenges to naively combining datasets at different scales or resolutions.

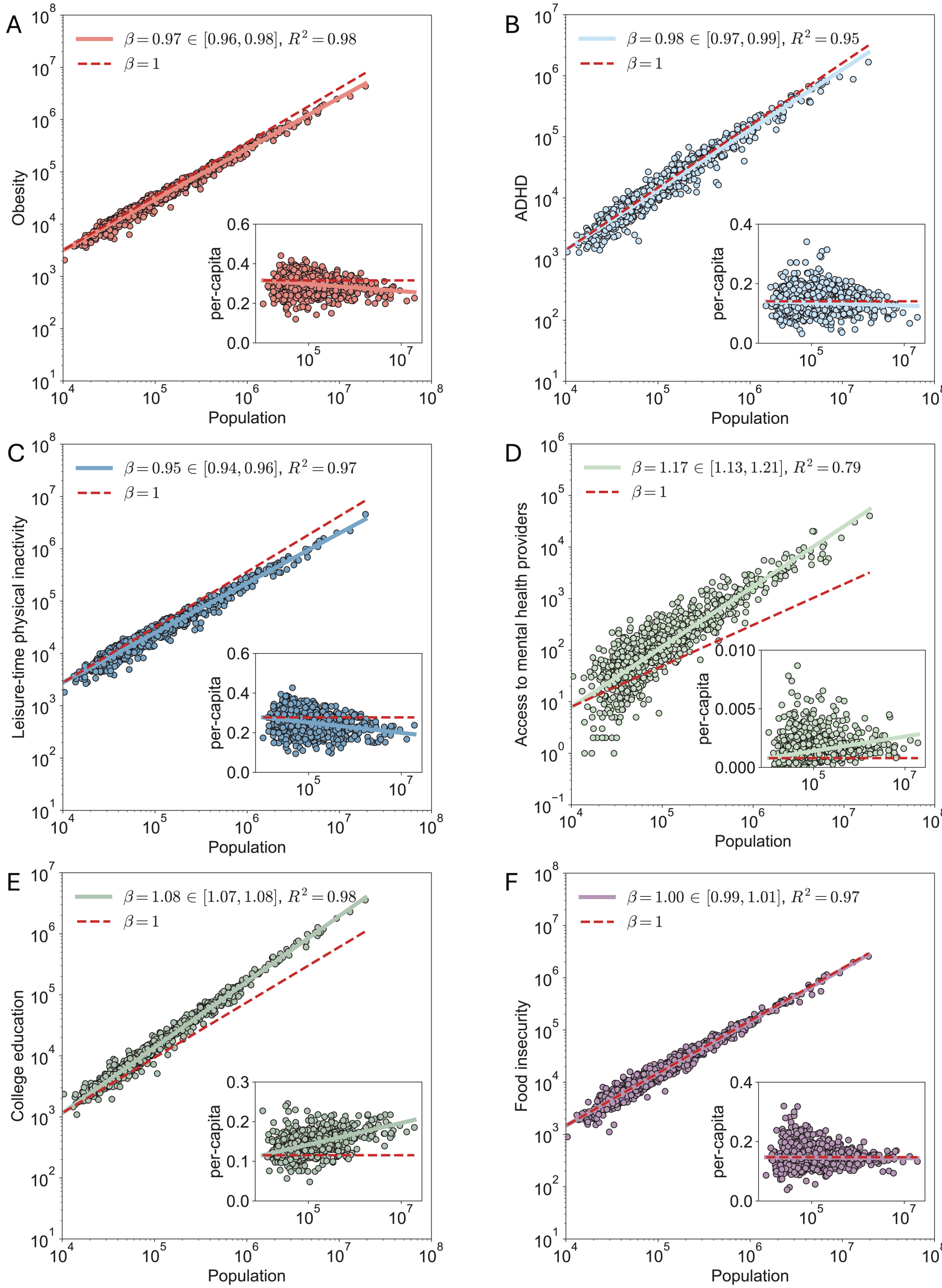

Fig 1. Urban scaling results for 915 cities in the United States of America. As a function of the city population, dots identify the number of (A) adults living with obesity in 2015, (B) children with a diagnosis of ADHD in 2016-2018, (C) adults who reported no leisure-time physical activity in 2015, (D) mental health providers in 2015, (E) adults with some post-secondary education in 2015, and (F) food-insecurity in 2015. The dashed line in each plot represents linear scaling and insets show corresponding per-capita metrics.

https://doi.org/10.1371/journal.pcsy.0000046.g001

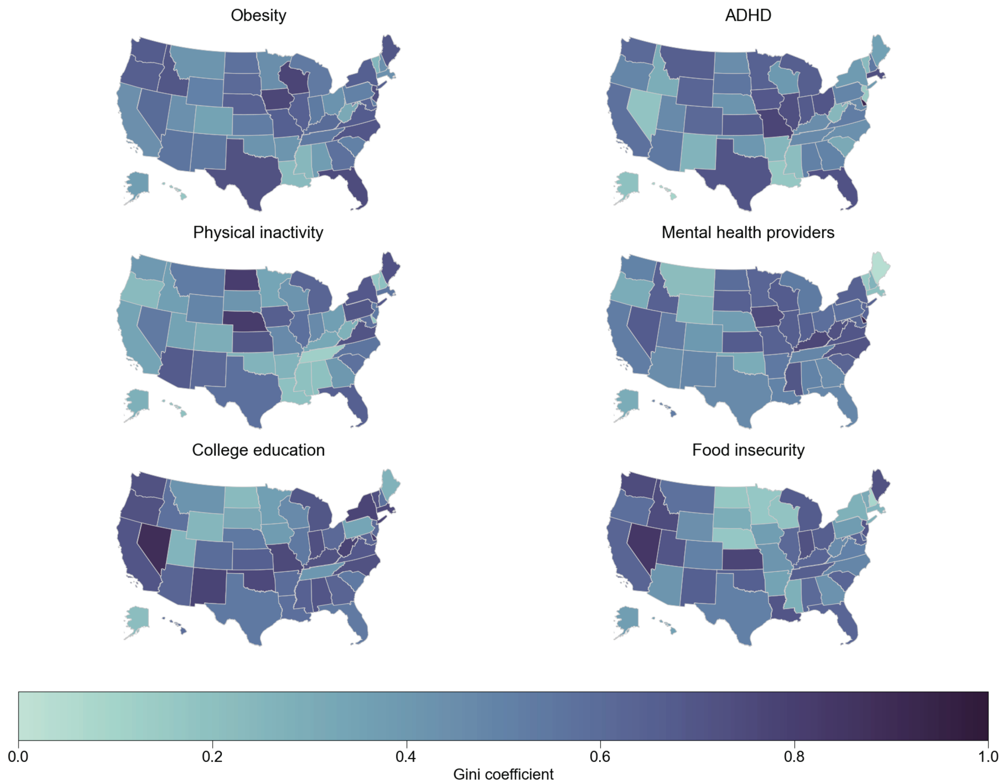

Fig 2. Mappings inequalities for 49 states in the United States (except for Connecticut). Maps among six urban features for 915 cities, measured by Gini coefficients: prevalence of adult obesity, ADHD in children, adult physical inactivity, mental health providers, college education, and food insecurity. The scale of Gini coefficients is from 0 to 1. The USA census shape files [77] were used to create these maps.

https://doi.org/10.1371/journal.pcsy.0000046.g002

City-level analysis: Associations

To identify potentially causal associations between the variables of interest while avoiding spurious relationships, it is first necessary to normalize data with respect to city size. This step is afforded by the use of the SAMIs, which provide a normalized measure for the deviation of the urban feature of a particular city with respect to its nominal value that would be predicted by the corresponding scaling law (see Sect A of S1 Appendix). For example, New York City (the rightmost point on any of the graphs in Fig 1) has a positive residual with respect to leisure-time physical activity and a negative one for obesity. As such, New York City dwellers have comparably fewer opportunities for physical activity during leisure time but, nonetheless, have lower likelihood of suffering from obesity. The SAMIs were then used as the input to the causal analysis, which utilized pairwise and partial correlations between variables for the identification and orientation of associations within Pearl's framework [41] (see Sect B of S1 Appendix for details).

The analysis was performed using the Peter-Clark (PC) algorithm [46], with significance set at 0.050. Given that the SAMIs of the urban features are distributed approximately normally (see Sect B of S1 Appendix), the algorithm computes Fisher's z scores among selected combinations of features, to derive a DAG under the following assumptions: causal Markovianity, faithfulness, lack of hidden variables, and acyclicity (see Methods). The output of the algorithm is shown in Fig 3A (see Sect B of S1 Appendix for details). The observed DAG

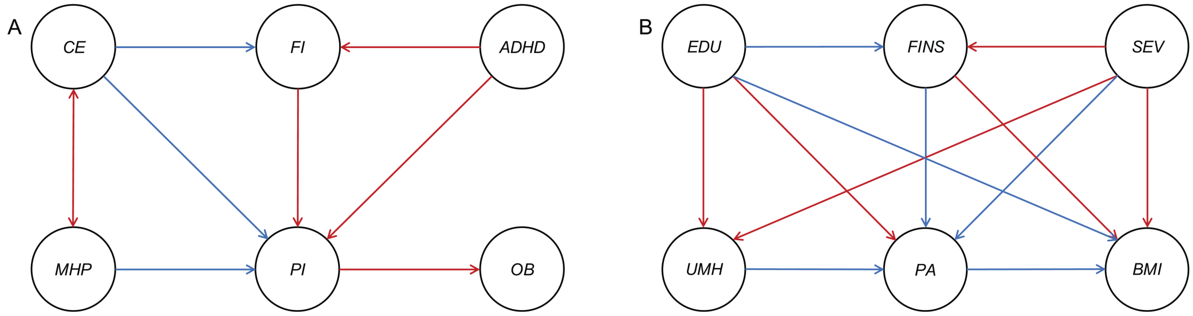

Fig 3. Directed acyclic graphs (DAGs) established from the study city- and individual-level data. (A) DAG among six urban features for 915 cities, measured by Fisher's *z* score: prevalence of adult obesity (*OB*), ADHD in children (*ADHD*), adult physical inactivity (*PI*), mental health providers (*MHP*), food insecurity in children (*FI*), and college education (*CE*). (B) DAG among the six individual features for 19,333 children, measured by Spearman-ranked correlation: body mass index (*BMI*), number of days per week the child does physical activities for over 60 minutes (*PA*), severity of current ADHD conditions (*SEV*), use of mental health care services (*UMH*), food insufficiency (*FINS*), and adult education (*EDU*). Red (blue) arrows indicate positive (negative) association.

https://doi.org/10.1371/journal.pcsy.0000046.g003

suggests that increased physical inactivity during leisure time results in a city populated by a higher number of obese adults. Importantly, we observed that a higher prevalence of children with ADHD is associated with an increased prevalence of food insecurity and physical inactivity. The latter factors are not independent, whereby increased physical inactivity is related to increased food insecurity. At the same time, the presence of mental health providers seems to promote a healthier lifestyle, characterised by a decrement in physical inactivity. Although our analysis fails to provide a directional association, it nonetheless highlights that higher college education correlates with increased access to mental health providers and with reduced prevalence of food insecurity. Despite that casual sufficiency (no hidden variables) is an assumption of the approach, the causal link between physical inactivity and obesity is not prone to hidden variables, that is, there is no unmeasured common cause between the two. Additionally, the causal effect of ADHD on obesity, mitigated by physical inactivity, seems to be robust to the algorithm assumption (see Sect C of S1 Appendix for a detailed sensitivity analysis).

Individual-level analysis: Associations

As a following step, we assessed whether the DAG inferred from city-level data – wherein the single unit of observation is a city and wherein individuals cannot be resolved – was confirmed at the level of an individual. To this aim, we conducted an analogous analysis on a large dataset collected in 19,333 children (see Methods). Different from city-level data, this dataset retained the individual identity of a person and provided all the metrics required for our analysis for each individual. Namely, this dataset comprises categorical information about BMI, the number of days per week the child does physical activity for over 60 minutes, the severity of the ADHD condition, whether the child has received or not mental health care, the highest education of adults in the child's household, and the household's ability to afford food.

Similar to the analysis of city-level data, we applied the PC algorithm, but this time using Spearman-ranked correlations due to the categorical nature of the variables (see Sect B of S1 Appendix). Predictably, the severity of ADHD diagnosis increased the use of mental health care. Similar to city-level data, individual-level data support the view that activity patterns are causally linked to BMI, whereby higher levels of physical activity are conducive to reduced

BMI. The similarity between city- and individual-level observations extended to the link between ADHD and physical activity, whereby a higher severity of ADHD is linked to a reduction in physical activity. Our analysis also strengthens the hypothesis that ADHD may constitute a risk factor for obesity. Thus, besides the indirect link detailed above (city- and individual-level data), we also observed – at an individual level – that increased severity of ADHD is directly linked to higher BMI. Additionally, both analyses indicate that accessibility to adequate food resources may constitute an important node in the aforementioned associations. Last, the city-level findings supporting a direct role of college education in physical activity are reverberated and further detailed at an individual-level. Being reared in a family with a high level of college education protects against increments in BMI directly and indirectly (increasing physical activity and reducing food insufficiency). Adult education in the household also has a positive effect on the extent to which children receive mental health care, which, however, may come at the expense of reduced time available for children to engage in physical activity. Just like the city-level causal graph, the derived DAG seems to be robust to the algorithm assumption (see Sect C of S1 Appendix for a sensitivity analysis).

Discussion

Our urban scaling approach indicated that well-being improves with urbanisation, whereby the prevalence of adult obesity, ADHD in children, food insecurity, and level of adult physical inactivity scale sublinearly with the population size, while access to mental health providers and prevalence of college education scale superlinearly. Within each State, cities are different with respect to any of these features, confirming the inequalities in the United States in lifestyle, education, healthcare, and access to resources [47]. Our formal investigation of potentially causal associations revealed that ADHD prevalence increases obesity prevalence, through the mediating role of physical inactivity. Our analysis highlights the role of physical activity as a feasible target for intervention, being sensitive to variations in college education, food insecurity, and access to mental health providers. Besides substantiating city-level findings, the study of individual-level data in children helped identify a direct causative role of ADHD on obesity during development.

Sublinear scaling of obesity prevalence and physical inactivity is in agreement with the literature [32]. A potential explanation could be the increased social contacts and the associated increments in fitness pressure experienced by dwellers of larger cities [34]. Through the lens of urban scaling, physical well-being may be viewed akin to criminality and viral infections, which spread more in larger cities where dwellers create on average denser networks of contacts [43]. The superlinear scaling of the prevalence of mental health providers was somehow in line with the increased gross domestic product and personal income in larger cities [43], which could promote active seeking of psychologists and psychiatrists by city dwellers. Increased wealth in larger cities may also represent a key driver for the observed superlinear scaling of college education (*e.g.,* more jobs being available to individuals with a higher level of education) and sublinear scaling of food insecurity. Another factor favouring the increased access to mental health providers is the documented increase in psychiatric disorders in urban versus rural settings [48]. In this vein, city dwellers will call for a larger share of the market of mental health providers, compared to rural residents.

Yet, in partial contrast with the last statement, we observed that ADHD prevalence scale sublinearly, not superlinearly, with population size. One simple, albeit speculative, explanation may entail evolutionary adaptive considerations related to founders' effect, wherein smaller populations are characterised by smaller genetic diversity [49]. Under this premise, the elevated heritability of ADHD [50] may favour its exhibition in smaller cities. Another explanation lies directly in the increased availability of mental health providers. Within this framework, a higher prevalence of psychologists and psychiatrists is expected to moderate the prevalence of children suffering from ADHD. Although the benefits of social skill training on ADHD are yet to be confirmed [51], one might argue that the increased number of social interactions afforded by larger cities may indirectly benefit children suffering from ADHD. We acknowledge that the observed scaling is not strongly sublinear, yet the presence of sublinearity still carries significance, as it indicates diminishing per capita prevalence with increasing city size, which must be taken into consideration when performing causal discovery (see Sect D of S1 Appendix).

The similarity in the scaling of ADHD and obesity prevalence supports the growing body of evidence about a link between impulse control and obesity [11–13]. Urban scaling alone is, however, prone to spurious correlations; a more principled approach to explore associations is to apply causal discovery tools to the resulting SAMIs. Such an analysis allowed us to identify an indirect influence of ADHD in childhood on obesity in adulthood, moderated by the level of physical inactivity. Albeit not in a causal sense, an association between ADHD and physical activity has been documented by Cook et al. [52]. Therein, the authors report that "[youth with ADHD] were significantly less likely to meet recommended levels of physical activity" and attributed this observation to "poor motor skills and executive function deficits." These factors may reduce the interest of individuals suffering from ADHD to engage in physical activity and challenge their ability to adhere to the strict regimens of physical exercise. Our analysis suggests that mental health care may contrast these tendencies, promoting awareness about health values of physical activity in city dwellers. The increment in generalized wealth observed in larger cities seems to be causally linked to somatic health. Thus, higher prevalence of college education and reduced prevalence of food-insecure households reduce proneness to obesity via the modulation of physical inactivity. Additionally, although we were not able to identify directional cause-and-effect links, we observed that college education was positively associated with access to mental health providers, and negatively associated with food insecurity. The extended benefits of higher education align with the evidence reported in countless studies [53].

Access to individual-level data allowed us to conduct a targeted analysis of these interactions and preliminarily test the hypotheses generated by urban scaling data. In agreement with our city-level analysis, individual-level data indicated that ADHD severity may indirectly increase BMI via physical activity. Additionally, in line with our hypotheses, we observed that, at an individual level, increased severity of ADHD directly increases BMI. In line with the literature [11], we posit that impaired impulse control may promote unhealthy food choices (*e.g.*, emotional and binge eating) which ultimately predispose to obesity. Neuroimaging studies [54] have identified specific brain circuits associated with impulsivity in food consumption, highlighting the neural basis of dysregulated eating behaviors. Notably, impairments in the brain pathways involved in impulse control (*e.g.*, anterior cingulate cortex [55]), have been linked to aberrant food choices that contribute to obesity [56]. The presence of a biological link between obesity and aberrant impulse control is also substantiated by genetic studies. Mota and colleagues conducted a genome-wide association study to investigate potential links between obesity and ADHD and identified a set of genes encoding for dopaminergic neurotransmission as candidate mediators of the observed association [16]. Importantly, in the same study, genes involved in dopaminergic transmission were also associated with variations in putamen volume, a brain region implicated in obesity and ADHD [16]. With respect to college education, the use of individual-level data allows us to detail the associations of the edges that could not be oriented from city-level data. Specifically, our analysis confirms the pivotal role of college education on individual well-being, whereby children reared in a household with highly educated caretakers are more frequently referred to mental health providers and have access to a better diet (and experience the metabolic benefits thereof). To some degree of surprise, increased referral to mental health providers reduces the time spent in physical activity, likely due to the limited time budget of a family.

Although our results were obtained using data across the USA, studies conducted outside the USA also support the association between ADHD and obesity. For example, research on Dutch children with ADHD revealed an increased risk of being overweight compared to their peers [57], while Korean children with ADHD have a higher risk of obesity [58]. This suggests that unhealthy overeating behaviors driven by impulsivity are consistent across different cultural contexts. However, variations in policy interventions, food environments, or mental health supports may shape the relationship we observe [59–61]. In this vein, regulated school meal programs, available in most European countries, help to structure children's diets, whereas the low-level taxes on fast food implemented in the USA have limited effect [60].

The work is not free of limitations, especially in the context of the causal analysis. First, the analysis implemented in the PC algorithm comes with a number of assumptions, including the absence of hidden variables, the causal Markov condition, and acyclicity. Since the Markov assumption is inherent to the PC algorithm, this algorithm removes links when nodes are found to be (conditionally) independent; circumventing this assumption would require adopting alternative causal discovery methods. Traditional regression models, such as multiple linear regression, as well as structural equation modeling [62], could serve as alternatives by explicitly modeling causal pathways and allowing for statistical adjustments of confounders. While the inclusion of several urban and individual variables in the causal discovery provides some confidence regarding the observed relationship between obesity and impulsivity, excluding hidden variables and verifying acyclicity would require additional covariates and temporal, highly resolved data, which are not available at present. Given the large network and the sensitivity analysis conducted, the possibility of a hidden confounder is unlikely, but it remains a possibility that could strain our results. Second, city- and individual-level data are not fully comparable, due to the age difference between the groups, the type of data (continuous versus categorical), and the scoring of the variable. Third, individual-level data lacks information about the specific city where the individual resides. Such a limitation, combined with the observed inequalities within cities of the same State, makes it unfeasible to combine city- and individual-level data in a causal analysis. Lastly, we cannot exclude the possibility that spatial dependency may play a role in the scaling behaviors [63]; exploring such an effect would require data at finer resolutions, beyond those we currently have.

The mechanism of obesity development represents an intricate and multifaceted health condition, typically emerging from a combination of genetic, environmental, and behavioral factors. Our findings corroborate the claim that environmental factors and lifestyle choices, including access to mental health services and physical activity, influence the development of obesity. The current obesogenic environment, characterised by an abundance of nutritionally, calorie-rich food offerings [64], predisposes people towards unhealthy eating behaviors, including overeating and unhealthy food choices. The origins of obesity are often traced back to childhood, marked by inadequate nutritional practices fueled by impulsive behavior. Children exposed to unhealthy food options, calorie-dense snacks, and sugary beverages are prone to cultivating sub-optimal eating habits that may endure into adulthood. The metabolic *sequelae* occurring thereof impinge on those brain structures involved in executive functions and impulse control. This cycle of subpar nutrition, lack of physical activity, aberrant metabolism, and impaired cognition tend to perpetuate itself, resulting in mental and somatic comorbidities in adulthood. Our findings suggest that rural environments are more conducive to fuel this pandemic, calling for local municipalities and state officials to take an active role in promoting equitable mental health care for residents, devising ways to prompt healthy food choices and physical activity, and sharing data to help researchers devise effective urban interventions. Given the link between ADHD, physical inactivity, and obesity, policymakers should prioritize integrating structured physical activity into ADHD treatment strategies. Schools and workplaces should incorporate movement-friendly environments, while healthcare providers must emphasize exercise as part of ADHD management. Public health initiatives should raise awareness and provide resources to foster active lifestyles, ensuring that interventions address both mental and physical well-being.

Methods

City-level data

To evaluate how obesity, physical activity, access to mental health care, food insecurity, education, and ADHD in children vary as a function of the population size in the USA and whether/how they are associated, we used two different, freely available, county level, data sources. Specifically, we utilized the 2015 County Health Rankings National Findings Report [65] for prevalence of obesity (*OB*), access to mental health providers (*MHP*), prevalence of physical inactivity (*PI*), prevalence of college education (*CE*), and prevalence of food insecurity(*FI*), and the 2016–2018 multivariate regression model by Zgodic et al. [66] for prevalence of ADHD among children (*ADHD*), see Table 1.

Prevalence of obesity is defined as the number of adults who reported BMI ≥ 30 kg/m^2. Access to mental health providers in a county is the number of mental health providers located within the county. Prevalence of physical inactivity is the number of adults aged 20 and over reporting no leisure-time physical activity. Prevalence of college education is the number of adults aged 25–44 with some post-secondary education. Prevalence of individuals with a lack of access to enough food (*i.e.*, struggling to access nutritionally adequate diets). We grouped the counties in 915 metropolitan and micropolitan statistical areas (MSAs) using CBSA to FIPS County Crosswalk files [67].

Individual-level children data

To address whether the relationships identified at the city-level were consistent at an individual level, we screened available sources to identify a dataset reporting paired data for each single individual. We obtained this individual-level dataset from the 2021 National Survey of Children's Health [68], a nationwide survey that screens households with children under 18 years of age. In each household, one child was randomly selected as the subject of the questionnaire, providing information about the following categorical variables: body mass index (*BMI*), physical activity (*PA*), ADHD severity (*SEV*), food insufficiency (*FINS*), use of mental health care services (*UMH*), and adult education (*EDU*), see Table 2.

In the available dataset, *BMI* is scored as underweight, healthy weight, overweight, and obese. *PA* quantifies the number of days per week the child does physical activities for at least 60 minutes: 0, 1–3, 4–6, and 7 days. *SEV* quantifies the severity of ADHD condition, classifying into three categories: does not currently have ADHD, current ADHD rated mild, and current ADHD rated moderate or severe. *UMH* offers an indicator of whether the child received any treatment or counselling from a mental health professional during the past 12 months with two categories: received mental health care or not. *FINS* is used as an indicator of the household's ability to afford the food they need with four categories: could always afford to eat nutritious food, could always afford enough to eat but not always the kinds of food should eat, sometimes could not afford enough to eat, and often could not afford enough to eat. *EDU* indicates the highest education level attained by the adults in the child's household, categorized by less than high school, high school or general educational development, some college or technical school, and college degree. For this dataset, we debiased the columns that contained missing values for any of these five variables, and we limited our study to children aged between 10 and 17 years as this was the only age range that contained values for all of the five variables. After debiasing, the dataset contained data from 19,333 children.

Fitting of urban scaling laws

To determine the values of β and all of the SAMIs (ξ_i)'s for each feature, we applied ordinary least squares (OLS) regression to the logarithmically transformed data. Heteroskedasticity was considered by using a heteroskedasticity-consistent covariance estimator (so-called HC1) [69] in OLS regression when calculating standard errors for R^2 and the 95% confidence interval for β. The linear regression analyses were conducted using the Python library *Statsmodels* [70].

Measurement of resource inequality

The Gini coefficient is commonly used to study equality of urban resources [71]. We computed the Gini index of the SAMIs for all of our urban features for each state. The original Gini index is developed for non-negative values only, as it was used for income. Since the SAMIs can be negative too, we relied on the modified Gini index developed by Raffinetti et al. [72],

$$G = \frac{\sum_{i=i}^{N}\sum_{j=i}^{N}|Y_i - Y_j|}{2(N-1)(T^{+} + T^{-})}, \tag{2}$$

where $T^{+} = \sum_{i=i}^{N}\max(0, Y_i)$ and $T^{-} = -\sum_{i=i}^{N}\min(0, Y_i)$. N is the number of cities within each state and the Ys represent the SAMIs of the corresponding cities. Similar to the original Gini index, $G = 1$ corresponds to maximum inequality and $G = 0$ corresponds to maximum equality.

According to the United Nation's definition [73], a Gini coefficient below 0.2 represents absolute income equality, while 0.2–0.3 indicates relative equality. Ranges of 0.3–0.4 and 0.4–0.5 signify adequate equality and relative inequality, respectively. Values above 0.5 indicate severe inequality.

Causal discovery

We relied on the PC algorithm [74]. The PC algorithm is a heuristic approach to explore causal structures in multivariate datasets, in the form of directed acyclic graphs (DAGs), through an interactive process of statistical conditional independence tests. The algorithm has two main phases: skeleton discovery and orientation phase. During the skeleton discovery, the algorithm starts with a complete undirected graph, and it iteratively removes links when the conditional independence test fails to reject the null hypothesis of conditional independence between variables. The second phase is the application of deterministic rules based on the skeleton structure and the results of the statistical tests to orient the links of the graph.

For the study of city-level data, we implemented the PC algorithm on the SAMIs of the six main variables with a significance level of 0.050 and the Fisher's z score [75] as the statistical test. The order of the variables used was: *OB, FI, ADHD, MHP, CE*, and *PI*. For the individual-level children data, we also implemented the PC algorithm with a significance level of 0.050, but we used Spearman-ranked correlation as the measure of independence to reflect the categorical nature of the dataset. The order of the variables used was: *EDU, BMI, FINS, UMH, SEV*, and *PA*. For both cases, we relied on the PC implementation in Python from the package gCastle [76].

Supporting information

S1 Appendix. This appendix consists of four sections that provide additional details supporting the claims made in the main manuscript. Sect A: Scale-adjusted metropolitan indicators. Sect B: Implementation of the causal discovery tool. Sect C: Sensitivity analysis. Sect D: Spurious dependencies from per-capita data.
(PDF)

Acknowledgments

We would like to thank the NYU graduate students Ajayrangan Kasturirangan, Yu Ze Toh, and Sameer Upadhye for their preliminary efforts as part of their Complex Urban Systems group project and Christopher Buglino for his constructive input.

Author contributions

Conceptualization: Simone Macrì, Maurizio Porfiri.

Data curation: Tian Gan.

Formal analysis: Tian Gan.

Funding acquisition: Simone Macrì, Maurizio Porfiri.

Resources: Maurizio Porfiri.

Software: Tian Gan, Rayan Succar.

Supervision: Simone Macri, Maurizio Porfiri.

Visualization: Tian Gan.

Writing – original draft: Tian Gan, Rayan Succar, Simone Macrì, Maurizio Porfiri.

Writing – review & editing: Tian Gan, Rayan Succar, Simone Macrì, Maurizio Porfiri.

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