Parks, pedestrians, and pediatric adiposity: A spatiotemporal analysis in an urban longitudinal cohort

Lucas Shen[1,*]; Mya Thway Tint[1,2]; Yung Seng Lee[2]; Fabian Yap[3,4]; Evelyn Loo[1]; Yap Seng Chong[1,2]; Keith M. Godfrey[5]; Johan G. Eriksson[1,2]; Jonathan Y. Huang[4,6,*]

Affiliations: [1] Institute for Human Development and Potential, Agency for Science, Technology and Research, Singapore; [2] Yong Loo Lin School of Medicine, National University of Singapore, Singapore; [3] KK Women's and Children's Hospital, Singapore; [4] Duke–NUS Medical School, Singapore; [5] MRC Lifecourse Epidemiology Centre and NIHR Southampton Biomedical Research Centre, University of Southampton and University Hospital Southampton NHS Foundation Trust; [6] Thompson School of Social Work and Public Health, University of Hawai'i at Mānoa, Honolulu, United States

Correspondence*: lucas_shen@a-star.edu.sg; huangjy@hawaii.edu.

Funding/Support: The GUSTO study is supported by the National Research Foundation (NRF) under the Open Fund–Large Collaborative Grant (OF-LCG; MOH-000504) administered by the Singapore Ministry of Health's National Medical Research Council (NMRC) and the Agency for Science, Technology and Research (A*STAR). In RIE2025, the study is supported by funding from the NRF's Human Health and Potential (HHP) Domain, under the Human Potential Programme. KMG is supported by the UK Medical Research Council (MC_UU_12011/4) and the National Institute for Health and Care Research (NIHR Senior Investigator (NF-SI-0515-10042) and NIHR Southampton Biomedical Research Centre (NIHR203319)).

Ethics: Ethical approval for the GUSTO cohort (0–9 years) was granted by the Domain Specific Review Board (DSRB) [D/2009/00021] and the Centralised Institutional Review Board (CIRB) [2018/2767/D]. Ethical approval for the GUSTO follow-up (10 years onwards) was provided by the CIRB and DSRB [2019/2655/E]. Informed parental consent was obtained for all participants.

Conflict of Interest: The authors declare no competing interests.

Data & Code Availability: All code for analyses will be made publicly available. Requests for access to GUSTO data can be submitted to the GUSTO Data Access Committee and will be reviewed in accordance with institutional and ethical guidelines.

Abbreviations: BMI, body mass index; GUSTO, Growing Up in Singapore Towards healthy Outcomes; GPS, global positioning system; SD, standard deviation; CI, confidence interval; WHO, World Health Organization

Keywords: Child adiposity; BMI; Pediatric health; Park access; Urban pedestrian density; Longitudinal cohort; Spatiotemporal exposure

Date: November 23, 2025

Word count (main text): 3691 (auto-computed via `texcount`)

Abstract word count: 275 (auto-computed via `texcount`)

Abstract

Background Cities shape child health. Effects are unlikely to be driven by a single feature, but are likely influenced jointly by built and social environments.

Methods We investigated the interaction between increasing park accessibility and pedestrian density on child and adolescent adiposity in 1032 children across 15 years from the Growing Up in Singapore Towards healthy Outcomes (GUSTO, 11,027 child-year observations, 2010–2024). Annual changes in park accessibility within a 15-minute commute were computed using up-to-date governmental inventories. Pedestrian density was derived from anonymized mobile phone GPS data, spatially smoothed to 0.1 km^2 hexagons. We modeled annual changes in BMI (kg/m^2) from birth to early adolescence as a function of changes in park access interacted with density using linear fixed-effect models, adjusting for child, calendar year, and origin/destination neighborhood effects and two-way clustered standard errors (child, geographical region).

Results Mean annual BMI gain was 0.3 kg/m^2 (SD 1.3), consistent with expected child growth. Overall, increasing park accessibility was associated with a -0.006 (95% CI: $[-0.016, -0.004]$) annual BMI gain. However, the parks–density interaction was negative (-0.021, 95% CI: $[-0.033, -0.009]$), implying different effects of parks access at different pedestrian densities: One-SD increase in park access (~3 parks) was associated with -0.005, -0.02, and -0.03 kg/m^2 at low (25th percentile), median, and high (75th percentile) density. Age-specific analyses indicate stronger park-density interactions at ages 5–7 and 10–11 years. Results were robust across sensitivity models and consistent across weight-related anthropometric measures (BMI and weight z-scores).

Conclusion Environmental effects are highly context-dependent. In this urban cohort, increases in park access were associated with lower BMI only in higher-density, higher-footfall areas. Increasing parks in low-density areas may not realize the same benefit.

1 Background

Childhood obesity has quadrupled globally since 1990,[1] with childhood overweight and obesity prevalence likely to reach 30% by 2030.[2] Recent data suggests these trends accelerated during the 2020 pandemic.[2–4] Excess adiposity increases long-term risks, including metabolic disorders, cardiovascular diseases, and psychosocial difficulties that persist into adulthood.[4–10] On current trends, the projected global economic cost will exceed US$4.3 trillion, or 3% of the world economy (equivalent to the 2020 pandemic shock).[3]

Given that 55% of children (~1.5 billion) now live in cities,[11] an important public health conversation has turned to how cities can create anti-obesogenic environments[10,12–14] that buffer adverse adiposity effects from urbanization.[2,11] A large body of work has established that neighborhood parks in urban spaces can promote outdoor play in children.[14–19] Since play and physical activity directly buffer excess adiposity,[20–22] neighborhood parks and green spaces have emerged as a natural lever for intervention.

While some studies have found protective associations of urban green spaces and parks for adiposity and related cardiometabolic markers,[23–29] many others report null or adverse associations.[10,12–14,17,30,31] These inconsistencies suggest that park effects are context-dependent. One such salient context is urban density: the level of pedestrian activity and foot traffic in daily lived experiences.[32,33] Higher-density areas typically imply more amenity stops, greater walkability, and therefore a greater propensity for unstructured outdoor activities that spur spontaneous visits to parks.[16]

Singapore, where childhood obesity mirrors global numbers in quadrupling (Fig. 1), is a well-suited testbed to examine how parks and urban pedestrian activity shape child adiposity. As a compact city-state with residential densities comparable to Tokyo and New York, Singapore maintains relatively egalitarian amenity distribution through public housing and ethnic integration.[34] Low crime rates minimize concerns about neighborhood safety that might complicate interpretations.[35] Active urban planning translates into temporal variation in parks as neighborhoods are (re)developed over time. Finally, the Growing Up in Singapore Towards healthy Outcomes (GUSTO) cohort offers an opportunity to follow children from birth to age 14 (at the time of study), with repeated and objective anthropometric measurements and geographically diverse residential histories that we can link to land use and density for within-child comparison over time.

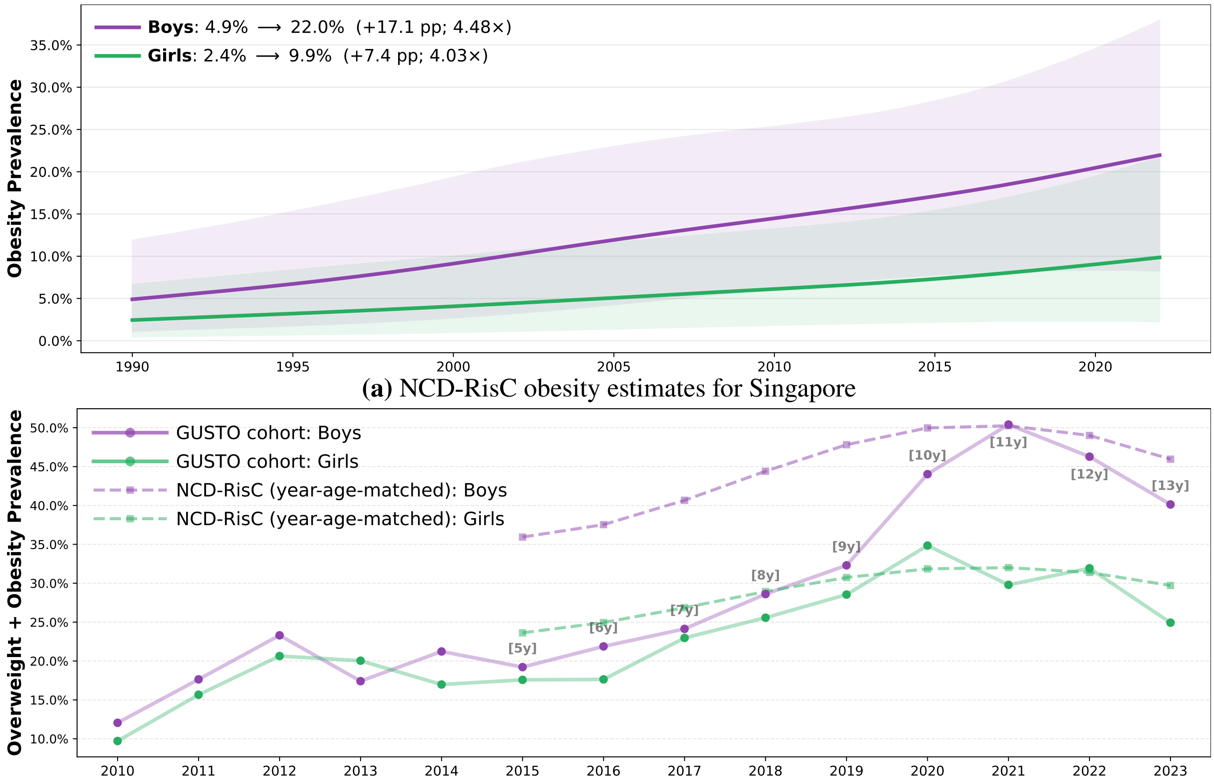
This study examined the protective association of neighborhood parks and whether urban density changes that association. Specifically, we tested whether living in high-footfall areas strengthens the inverse association between park access and adiposity measures, combining repeated park and anthropometric measures with network-based travel-time data and anonymized mobile phone trace data in a longitudinal model.

2 Methods

2.1 Study population

The study population is drawn from the Growing Up in Singapore Towards healthy Outcomes (GUSTO) cohort, a prospective mother-offspring birth cohort established in 2009. Pregnant women in their first trimester were recruited over the course of 2009–2010 from two major public maternity hospitals (KK Women's and Children's Hospital and National University Hospital). The study recruited 1247 pregnant women aged 20–50 years, mostly of Chinese, Indian, and Malay ethnicity (approximately 97% of ethnic composition).[1] [36] Eligibility criteria included being aged 18 or older, a Singapore citizen or permanent resident, and intending to reside locally for at least five years. Women were excluded if they had significant medical conditions (e.g., type 1 diabetes mellitus, psychosis). There were 1,177 deliveries, with an average annual attrition of approximately 3%, resulting in a population closer to 800 by 2020. Although not geographically representative by design, participant residences closely matched those of women aged 20–50 in the 2010 Census, with a correlation of 0.93 across neighborhoods.[19,37] For this analysis, we used 15 years of follow-up data (2010–2024) from 1,032 children, contributing 11,027 child-year observations (Tab. 1).

[1] https://web.archive.org/web/20121021001924/https://www.singstat.gov.sg/pubn/popn/population2012b.pdf.



(a) NCD-RisC obesity estimates for Singapore

(b) Prevalence of overweight and obesity in GUSTO cohort

Figure 1|Prevalence of overweight and obesity in Singapore based on NCD-RisC estimates and the GUSTO cohort. **Panel (a)** NCD-RisC's (NCD Risk Factor Collaboration) posterior mean prevalence for Singapore, 1990–2022, for ages 5–19.[1] The legend shows the percentage increase in prevalence from 1990 to 2022. **Panel (b)** GUSTO cohort prevalence from birth. Dashed lines show age- and calendar-year matched series from the NCD-RisC estimates for Singapore (same sex, same age, same year), where available. 2023 value in the NCD-RisC line is from 2022 data. No NCD-RisC estimates below 5.

2.2 Anthropometry

Weight was measured using calibrated electronic scales and length/height using stadiometers (recumbent length < 24 months, standing height thereafter). Each record was linked to the child's exact age in days, which is used to map to residence. To construct a regular child-year panel, all anthropometric measures were combined into a long dataset indexed by date. For years without a measure, values were linearly interpolated (no extrapolation) between the nearest observations. Age- and sex-standardized z-scores were derived using World Health Organization standards (Fig. 1).[19]

2.3 Residential history

Residential histories were constructed from the time-stamped postcode records collected during follow-up. Residential moving was defined by observed changes in postal code across follow-

Table 1|Overview of data

A. Sample		
No. children (n)	1,032	—
No. years (n, range)	15	(2010–2024)
No. child-year observations (n)	11,027	—
Year obs. per child (mean, SD)	10.7	(3.5)
Avg. days between measures (mean, SD)	353.9	(36.6)
Non-movers (n, %)	400	(38.8)
Movers (n, %)	632	(61.2)
B. Main measures	Mean	(SD)
ΔNumber of parks within 15-minute trip	0.36	(3.51)
Urban density (0.1km^2), 2020 GPS traces	1147.5	(1538.4)
ΔBMI (kg/m^2) per year	0.30	(1.30)
ΔWeight (kg) per year	3.33	(2.70)
ΔHeight (cm) per year	7.61	(4.32)
BMI at age 5 (2015)	15.6	(2.1)
BMI at age 10 (2020)	18.6	(4.1)
BMI $\geq$ age 12 (2023/24)	20.0	(4.2)
C. Geography		
No. Subzones (neighborhoods)	177	—
No. Planning areas (geographical cluster var.)	32	—
Avg. child per planning area per year (mean, SD)	26.4	23.4

up. We classified children as non-movers (400, 39%) and movers (632, 61%). Among movers, we identified serial movers (207, 20%) as those who moved twice or more within the 15 years. Multiple relocations within a relatively short timeframe may reflect a salient preference for location choice, raising stronger concerns about selection.[38] Cognizant of this, fully adjusted models excluded serial movers to account for location choice.[38] We assigned residence based on the closest residential record before the child's birthday. Each residence was then geocoded to the spatial units, including planning areas, subzones (neighborhoods), and hexagonal cells used for exposure construction.

2.4 Access to parks

Annual measures of park access were derived by combining a government annual land use inventory of park parcels with high-resolution, network-based travel times. We first represented the city as a grid of 0.1km^2 hexagonal cells (200 m edge length), restricting to approximately 2.4k cells covering populated, on-land areas (excluding sea, water catchments, nature reserves, and sparsely inhabited locations). We then enumerated 2.87 million centroid-to-centroid pairs and queried HERE Technologies' routing engine for door-to-door travel durations, incorporating networks, walking infrastructure, and public-transport schedules. For each child-year residence (mapped to its postal-code hex cell), we identified all cells reachable within 15 minutes, consistent with '15-minute city' frameworks emphasizing proximity to daily needs,[39] and overlaid them with park parcels (public parks, gardens, and pedestrian green linkages). Park access was defined as the number of parks within this 15-minute catchment, recomputed annually to reflect contemporaneous land zoning and residence.[37]

2.5 Urban pedestrian density

We measure neighbourhood urban density using anonymised global positioning system (GPS) ping traces from CITYDATA.ai, aggregated over January–March 2020 (excluding Chinese New Year), with device IDs hashed and daily presence observed at the neighbourhood level.[34,40] To derive a spatially refined density measure, we areally interpolated GPS traces from the neighborhood polygons to a regular grid of ~0.1 km^2 hexagons (0.1 km^2 ≈ 25 acres; width 350 m ≈ 1,150 ft; ~city block size).[41] Before interpolation, hexagons were clipped to official neighborhood boundaries to avoid overlap with water bodies and other uninhabitable areas, and further masked using satellite-derived Copernicus Land Monitoring data to exclude non-urban land.[42] For each hexagon, we then computed weekly median traces per hexagon and winsorized the top 1%. This urban density is time-invariant, under the assumption that neighborhood activity ranks remain stable over the sample period, but spatially varying at high resolution, capturing relative baselines of human presence and pedestrian activity across the city.

2.6 Individual-level child and maternal covariates

All models adjust for baseline maternal and child characteristics collected at recruitment. Maternal covariates included age (at delivery), ethnicity (Chinese, Indian, Malay, Other), education (college vs. non-college), monthly household income ($<$ 2000 vs. $\geq$ 2000 SGD), country of birth, housing type (public vs. private), and occupation. All models adjust for the child's sex and age in days (from clinic visit dates).

2.7 Statistical Analyses

We modeled the short-run, contemporaneous annual change in BMI as a function of the annual change in park access and urban density:

$$\Delta\text{BMI}_{ijt} = \beta_1\Delta\text{Parks}_{ijt} + \beta_2\text{Density}_{ij} + \beta_3(\Delta\text{Parks}\times\text{Density})_{ijt} + \gamma\mathbf{X}_i + \text{neighborhood}_{ij} + \text{child}_i + \text{year}_t + \varepsilon_{ijt}, \quad (1)$$

where i indexes children, j residential neighborhoods, and t years. $\mathbf{X}_{it}$ includes the child and maternal baselines (Section 2.6). β_1 captures the association between a one-unit increase in park access and ΔBMI (evaluated at the reference mean density). β_3 captures how the association between changes in park access and ΔBMI varies with urban density.

To adjust for selection into neighborhoods for movers, we include fixed effects for their origin and destination neighborhoods, allowing families that come from or relocate to the same neighborhoods to have shared effects. We likewise adjust for the residing neighborhood for non-movers. child_i control for time-invariant child and family-level factors. τ_t captures the broad developmental trends, with exact age adjusted separately ($\mathbf{X}_{it}$). This structure compares within-child changes, holding constant neighborhood-specific unobservables and trends. Fully-adjusted models two-way cluster standard errors by child and planning area, recognizing that residuals are likely serially correlated within child (e.g., growth spurts, family history, routines) and correlated across children within the same areas (e.g., local amenities, school catchment). The key assumption is that any within-child changes in BMI resulting from unobserved factors related to changes in parks have been absorbed by shared developmental trends across time and age, neighborhood characteristics (including location choices), and child-specific predispositions.

2.7.1 Predicting changes in BMI by parks and density

To visualize how BMI responds to park access at different levels of urban density, we used the fully adjusted model to generate average adjusted predictions. For each chosen density level (e.g., quartiles), we fixed density, varied park access, and predicted BMI for every observation while holding other factors constant. We then averaged these predictions across individuals.

2.7.2 Age-specific associations

To assess how the park-density interaction varies across childhood, we used sliding age windows. For a window centered at age a days, we define $w_{i,a(t)} = 1$ if a child's exact age in days lies within ±500 days of a; and 0 otherwise. For each a, advanced in 30-day increments, we estimated the fully adjusted model on the full sample with an additional (triple) interaction that allows the parks–density term to differ inside the window w:

$$\Delta\text{BMI}_{ijt} = \beta_1 \Delta\text{Parks}_{ijt} + \beta_2 \text{Density}_{ij} + \beta_3 (\Delta\text{Parks} \times \text{Density})_{ijt} + \gamma \mathbf{X}_i + \gamma_a \left(\Delta\text{Parks}_{it} \times \text{Density}_{ij} \times w_{i,a(t)}\right) + \text{neighborhood}_{ic} + \text{child}_i + \text{year}_t + \varepsilon_{ijt}. \quad (2)$$

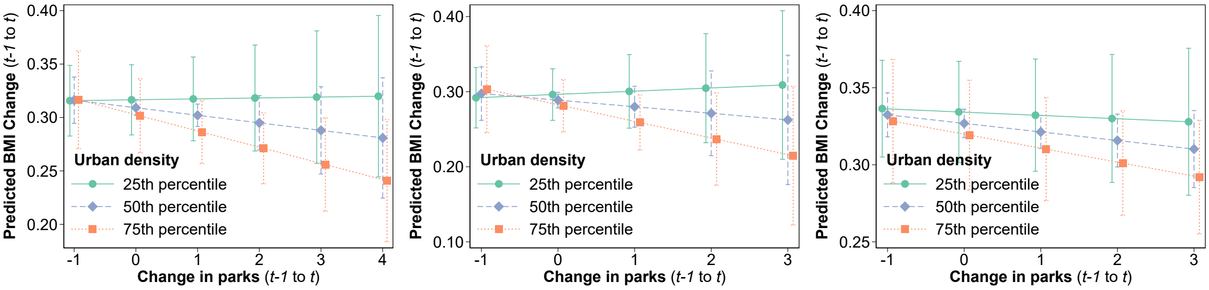
To show age-specific associations, we then plot the age-specific interaction effect $(\hat{\beta}_3 + \hat{\gamma}_a)$ across the values of a.

2.7.3 Sensitivity analyses

We assessed sensitivity in four ways. First, we examined related anthropometric outcomes—z-BMI, weight (kg), and z-weight—to evaluate consistency across adiposity measures. Second, height is largely driven by genetics and long-run nutrition and should not respond to short-run annual changes in parks. We therefore re-estimated the model with height and height-for-age z-score as negative control outcomes, where non-null estimates would imply residual confounding. Third, recognizing that large annual changes in parks are uncommon, we collapsed the continuous measure to a binary indicator for any increase (from $t-1$ to t) as an alternative specification. Fourth, we replicated the age-specific analyses with different window widths of ±365 and ±730 days to assess sensitivity to the window size. We adjusted for tests of multiple overlapping age windows with the Benjamini–Hochberg procedure.

3 Results

The sample includes 11,027 child-year observations from 1032 children across 15 years from birth (Tab. 1). The oldest child-year observation in the sample period was 13.6 years old.



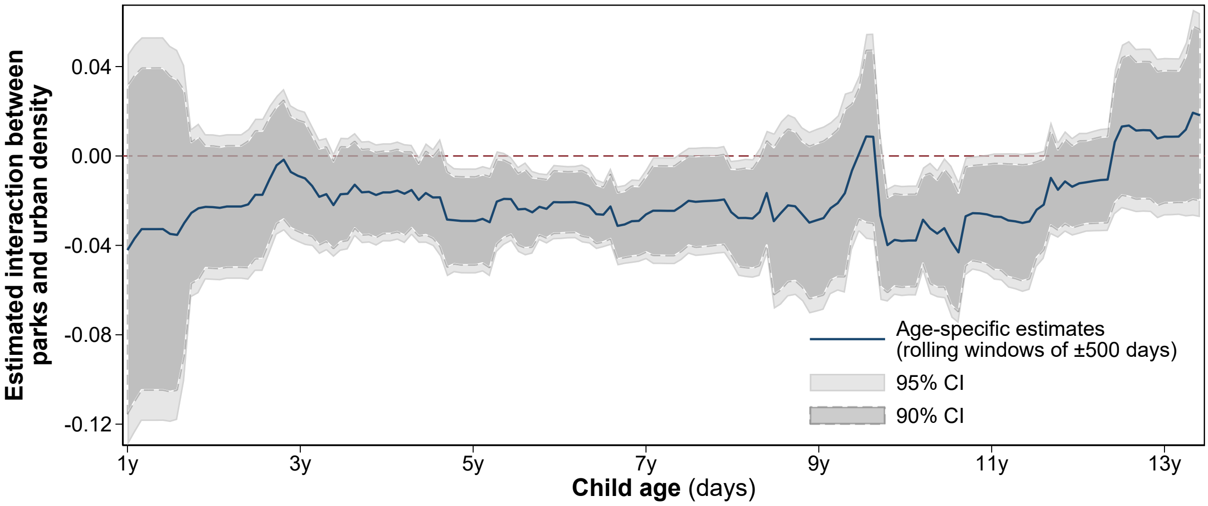
(a) Full sample **(b)** Non-movers **(c)** Movers

Figure 2|Predicted changes in child BMI associated with changes in parks at different levels of urban density (top panels) and age-specific estimates of the parks × density interaction (bottom panel). **Panels (a)–(c)** show predicted BMI changes from a regression model that allows the interaction effect to differ by non-movers versus movers, across quartiles of local urban density. Capped vertical lines are 95% confidence intervals based on clustered standard errors.

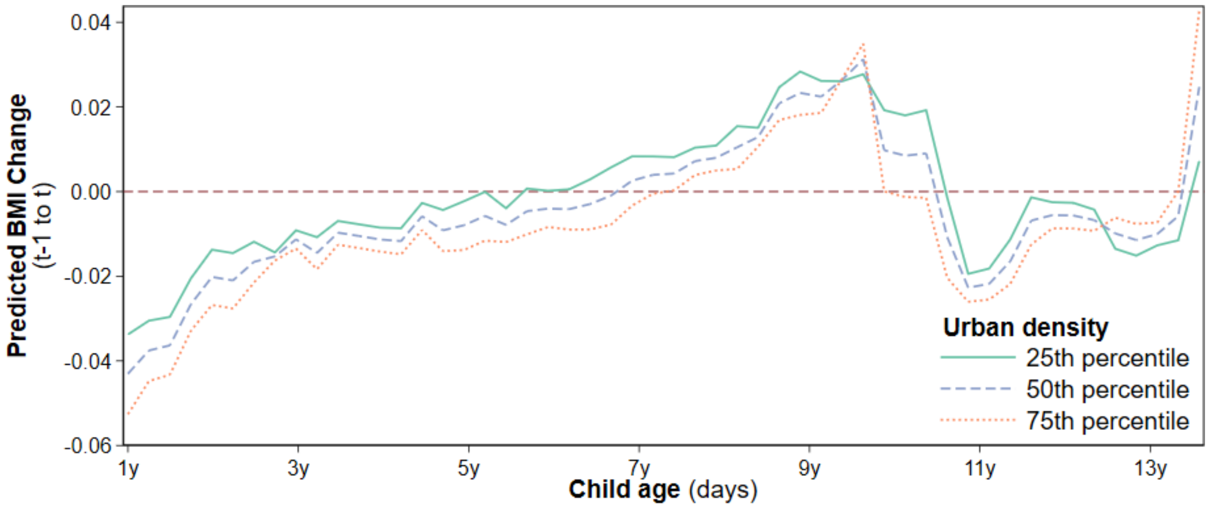
Average yearly change in BMI was 0.3 kg/m^2 (SD 1.3). Similar to the global rise in prevalence,[1], the GUSTO prevalence of overweight and obesity has more than tripled over our observation period (Fig. 1). Average yearly change in park access was 0.4 (SD 3.5) parks within a 15-minute trip from the child's residence. More than half the sample moved at least once across the 15 years, with 400 non-movers (38.7%), 425 (41.3%) who moved once, and 207 (20%) serial movers. We did not observe a huge difference in the number of per-year change in park between movers and non-movers (0.06 parks, 95% CI: $[-0.06, 0.17]$, $p = .32$), but movers were 5.2 percentage points (95% CI: $[3.8, 6.6]$, $p < .001$) more likely to experience an increase in parks.

In the fully adjusted model that accounts for location choice, fixed child effects, age, and broad developmental trends, the parks-density estimated interaction was negative ($\hat{\beta}_3 = -0.021$, 95% CI: $[-0.033, -.009]$, $p = .001$; Tab. S1), indicating that increases in park access were associated with lower BMI trajectories in higher-density areas, with minimal (or slightly positive associations) in lower-density areas. At the mean urban density, one additional park was associated with a –0.006 kg/m^2 lower BMI (95% CI: $[-0.016, 0.004]$, $p = .24$). The interaction shows that park–BMI effect strengthens with density: a one-SD increase in park access (~3 parks) was associated with -0.005 lower BMI at the 25th percentile (offsetting 1.6% of annual BMI increase), -0.02 at the median (offsetting 6.6%), and -0.03 at the 75th percentile (offsetting 11.3%). The predicted margins illustrate diverging slopes across density quartiles (Fig. 2a).

As noted earlier, movers were more likely to experience park increases. In models with baseline covariates only and without the density interaction ($\beta_2 = \beta_3 = 0$), increases in park access were associated with lower BMI ($\hat{\beta}_1 = -0.007$, 95% CI: $[-0.013, -0.000]$, $p = .04$; Tab. S1)



(a) Age-specific parks–density interaction



(b) Age-specific parks–density interaction

Figure 3|Age-specific associations between park access, urban density, and BMI. Both panels report the same series of rolling-window regressions (±500 days), in which the park–density interaction is re-estimated /repeatedly for successive child-age intervals. Each point on the x-axis corresponds to a separate full-sample regression that allows observations falling within an overlapping window around that age to have a different moderation effect. **Panel (a)** Age-specific estimates of the park access × urban density interaction (Eq. (2)). Shaded regions show 90% and 95% confidence intervals clustered by child and planning area. **Panel (b)** Age-specific predicted change in BMI from changes in parks at different levels of urban density.

After adjusting for location choice, this main effect became null. This attenuation suggests that, without accounting for location choice, unadjusted averages may reflect neighborhood selection rather than the underlying association between park access and BMI.

Nonetheless, the predicted margins stratified by non-movers versus movers showed similar patterns in lower predicted BMI change with increases in parks at higher density, but clearer gradients among non-movers (Figs. 2b to 2c).

We also examined age-specific heterogeneity in the park-density interaction, by estimating a series of 154 fully-adjusted regressions using rolling age windows of ±500 days (each window covering on average 1,759 (SD 354) child-year observations). The interaction was strongest around ages 5–7 and 10–11 years, with 95% CIs excluding 0 in those bands and wider intervals elsewhere (Fig. 3a). We did not observe abnormal jumps in residential relocation in those years (Fig. S2). Predicted margins likewise showed greater BMI decrease in denser neighborhoods, except around age 9 and after age 12 when the interaction effect attenuated or reversed (Fig. 3b).

The pattern implied by the park-density interaction was evident geographically. Aggregating child-year predictions to neighborhoods after stratifying child-year observations by whether park access increased or decreased, we observed steeper BMI reductions at higher levels of urban density when parks increased, with the reverse pattern when parks decreased (Fig. 4). To provide ground context for these associations, we mapped the terciles of urban density and annual change in park access across all public residences, illustrating the combinations at a higher spatial resolution (Fig. 5). Finally, we computed, for each residential postal point, the predicted change in BMI from a one SD increase in park access, holding that point's urban density fixed. The postcode-level predictions were then averaged to the 0.1 km^2 hexbins, indicating that larger predicted BMI reductions clustered around pockets in the north-east, west, and central regions where footfall is higher (Fig. 5, Fig. 6).

We explored other potential moderating effects of the park–density interaction with child sex and geographical characteristics (five official planning regions, mature versus middle-aged versus young areas), post-relocation year, and socioeconomic status. We found no heterogeneity across those strata, except that post-relocation years attenuated the park-density measure towards zero (Fig. S3).

Finally, we tested for sensitivity. First, we found similar patterns across related anthropometric outcomes for z-BMI, weight, and z-weight, where higher urban density strengthened the association between increases in parks and decreases in BMI (Tab. S2). Second, and as placebo outcomes, height and z-height showed no such pattern (Tab. S2). Third, we found similar patterns when we used a binary indicator for an increase in parks (Tab. S3). Fourth, the age-specific patterns at years 5–7 and 10–11 persisted after correcting for multiple testing (Fig. S1), and when we used different age windows (Fig. S4).

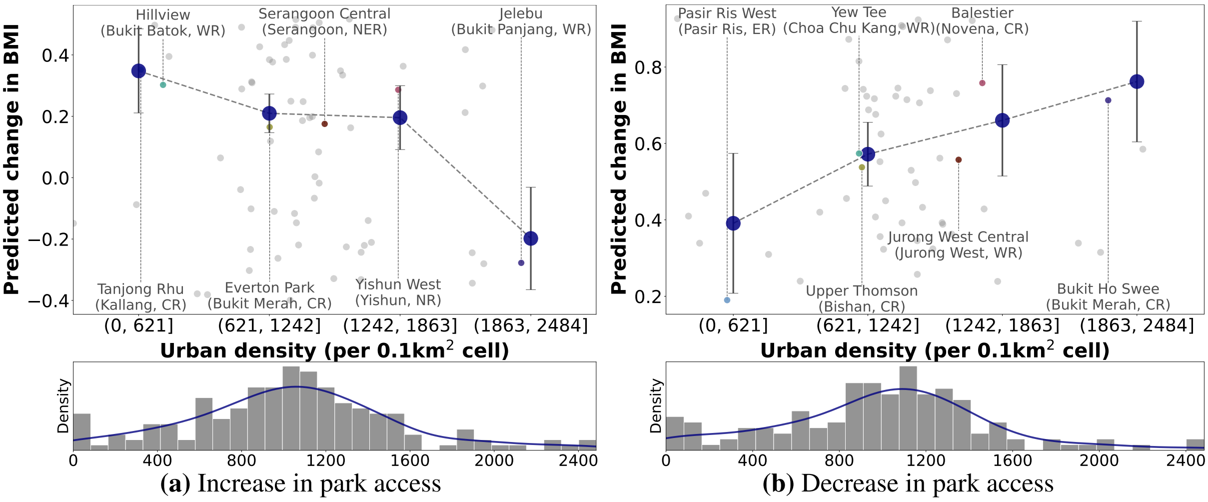


Figure 4|Model-predicted change in BMI against urban density, stratified by increases in park access (**Panel (a)**) versus decreases in park access (**Panel (b)**). Each gray point represents a neighborhood's average predicted BMI change across child-year observations. Large blue points show the average across neighborhoods within each density range; capped vertical lines indicate the standard errors of means. Bottom panels show the underlying distribution of urban density. Selected neighborhoods are labeled with their planning area and region (CR = Central Region, ER = East Region, NR = North Region, NER = North-East Region, WR = West Region).

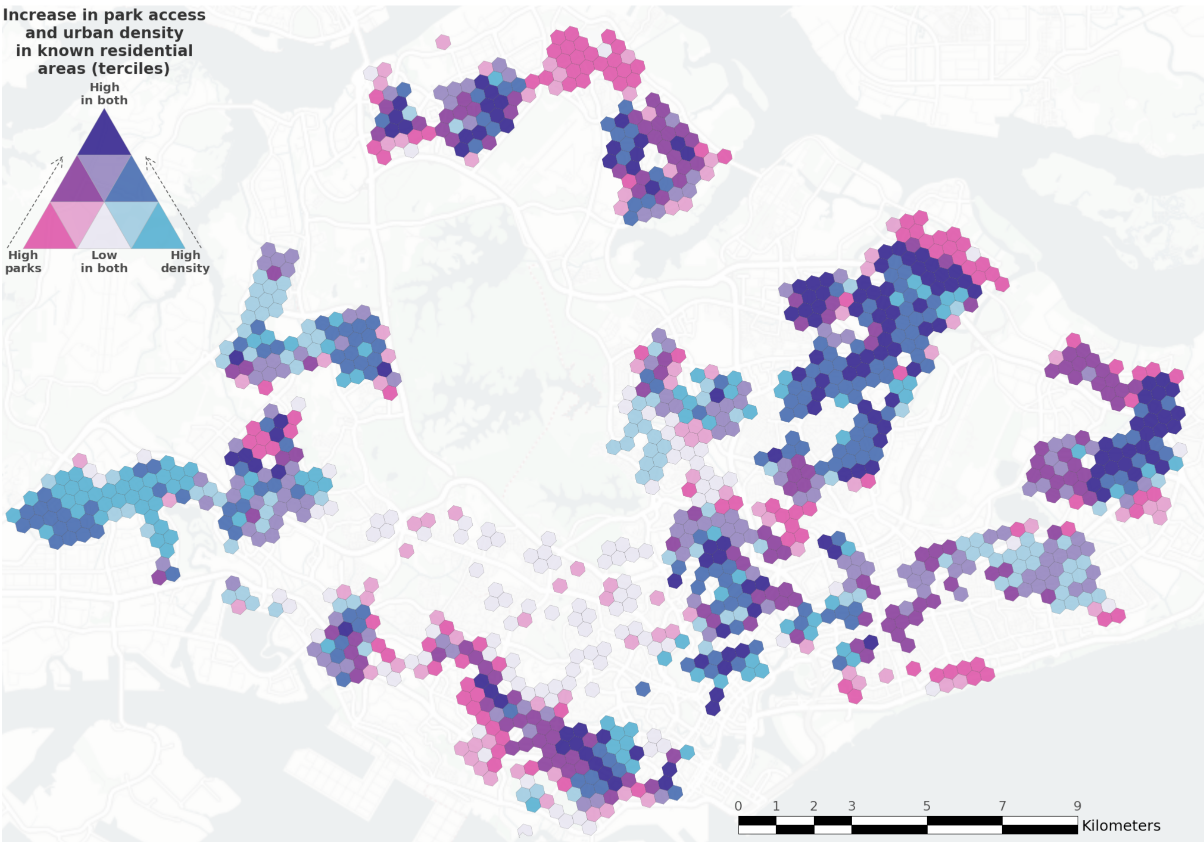


Figure 5|Spatial Distribution of Changes in Park Access and Footfall Density. Colors indicate the 3×3 tercile combination of the two variables (low/medium/high for each), based on ~13,000 residential points.

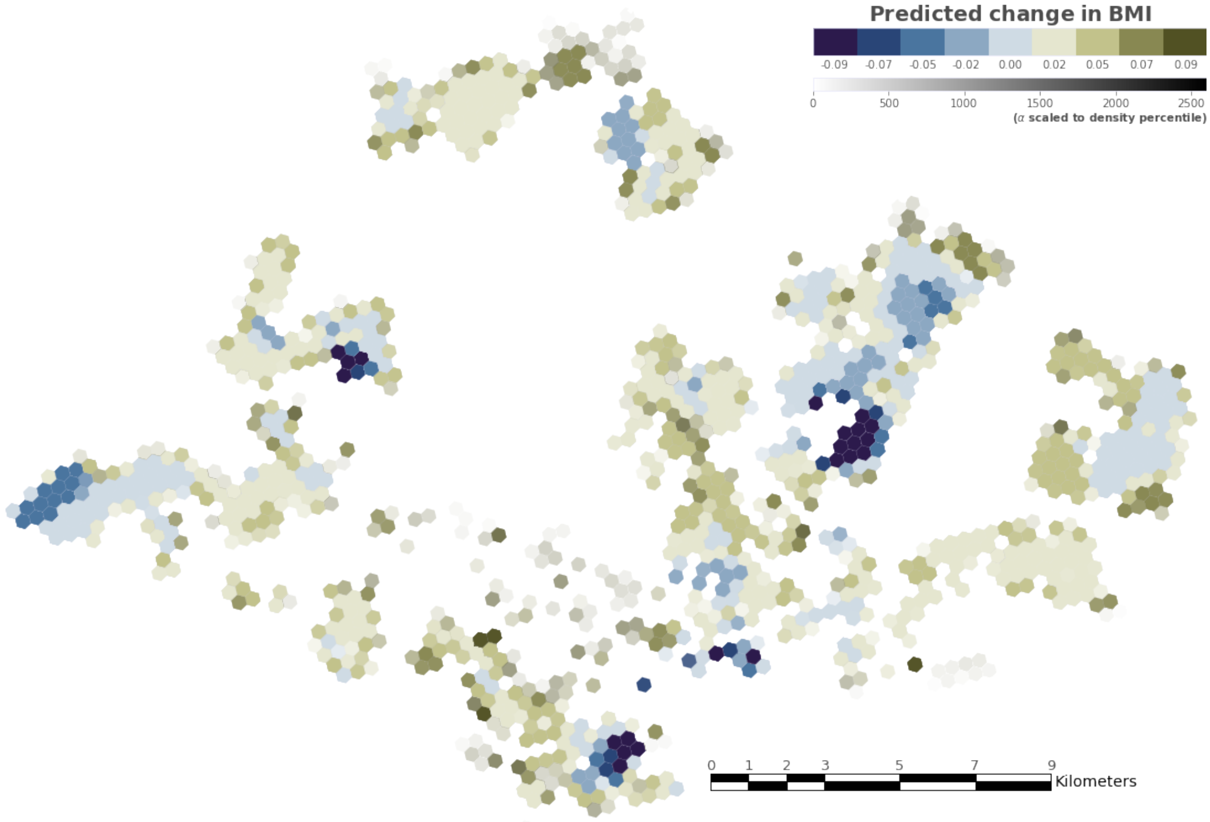


Figure 6|Spatial Distribution of Predicted BMI Changes from Park Access Improvements. Each hexbin is ~0.1 km^2, color-coded by the predicted BMI change from a one standard deviation increase in park access, holding urban density and all other factors constant. Graph transparency (α value) reflects urban density, with less populated areas shown as more transparent (and vice versa).

4 Discussion

4.1 Principal findings

We constructed and analyzed a 15-year panel of annual child anthropometric measures from a Singapore birth cohort, following 1,032 children from birth to adolescence for 11,027 child-year observations. We linked the cohort residential histories to time-varying measures of park access, computed from annual park zoning inventories and network-based travel time, and to urban density, derived from GPS density traces. Using within-child comparisons, controls for residential origins and destinations, and cluster-robust standard errors, we found that increases in park access were associated with lower adiposity increases, but only in dense urban areas. In models without the density interaction, park increases showed a negative association with BMI, but attenuated to null after accounting for location choice. Age-specific analyses revealed that the park-density interaction is stronger around ages 5–7 and 10–11 years. These patterns persisted across multiple anthropometric measures, but not for height as a placebo outcome.

4.2 Prior work

The literature on urban green spaces and child adiposity has found inconsistent evidence, including null or even adverse associations.[10,12–14] These differences are founded in how green space is measured (e.g., vegetation, proximity, park counts, trees),[10–13] the prevalence of cross-sectional settings,[10,12,13,17,23,25,27–29] contextual modifiers,[10,12–14,19,23] and bias from location choice.[12,26,38,43] Our findings speak to this heterogeneity by using network-based measures of access to park parcels from annual land-use inventories, examining urban density as a contextual modifier, and comparing within children in longitudinal models that follow them from birth into early adolescence.

Prior cross-sectional[23,25,27–29] and longitudinal studies[24,26] found a protective association with adiposity or cardiometabolic markers. Other cross-sectional[17,31] and longitudinal[30] studies found null associations. Without the density moderation and adjusting for an extensive set of child and maternal baseline characteristics, we found the hypothesized protective association. However, the first-order endogeneity concern is that families select into neighborhoods that support their preferred healthy lifestyle.[12,19,26,38,43] When we adjusted for such location choices,[38] the protective association attenuated to null.[19]

4.3 Pedestrian density as effect modifier

We found statistical evidence of the protective association in higher-density city blocks (approximately 315 m x 315 m if in square grids), which persisted after adjustment for location choice. Density has been examined as a focal exposure or as an adjustment of the environmental context,[16,24,26,31] but rarely as contextual moderators.[23] A prior study found that vegetation cover is associated with lower overweight risk only in towns categorized as high residential density.[23] Our study advances this evidence in five ways using: (1) a smooth measure of urban density capturing pedestrian activity at a spatial resolution consistent with city blocks (rather than binary strata across coarse administrative boundaries);[32,33] (2) a longitudinal panel with repeated measures of both park access and anthropometric outcomes; (3) walking- and transitbased access to park parcels; (4) an explicit modeling of location choice;[38] and (5) formally testing the park–density interaction to directly quantify effect moderation.

A 14-city study found that urban density predicts higher accelerometer-measured physical activity.[16] Hence, high-footfall city blocks may capture urban activity and pedestrian-friendly spaces, corresponding to higher walking and outdoor propensities that convert the dormant park access into unstructured, ad-hoc visits to parks,[37] in ways beneficial to metabolic health.[20–22]

We note that the exceptionally low crime rates in Singapore minimize concerns about a lack of safety and natural surveillance in density cold spots, so lower effects there do not necessarily capture higher neighborhood violence or crime.[35] However, our findings do not preclude the possibility that the park–density interaction captures a supply-side built environmental feature: that parks around low-activity city blocks are more dilapidated.[23,44] Another possible interpretation is that parks around denser areas relate to livelier social spaces, where co-occupancy creates informal activity hubs that encourage child outdoor play.

4.4 Age-specific effects

Prior reviews flagged age as a contextual factor,[10–12] but studies examining age-related associations are rare. We leveraged the temporal resolution of the GUSTO cohort to examine age-specific effects, finding that the park–density interaction was strongest around 5–7 and 10–11 years. These periods coincide with developmental transitions in Singapore, when children gain increasing independence in daily activities and mobility as they enter (age 7) and exit (age 12) primary school. The latter period also coincides with a sharp rise in overweight prevalence among boys (Fig. 1). The attenuation during adolescence could reflect pubertal changes independent of the environment, changes in structured activities (e.g., tuition, enrichment courses in sports, arts, or music), or lack of late adolescent observations. Age 9–10 for GUSTO children also coincided with the COVID-19 pandemic, a period where nearby parks might have particular strong protective effects when other structured activities ceased.[19] The literature lacks a clear prior for ages where associations are strongest,[10–12] so we interpret these patterns as speculative. Nonetheless, they suggest that environmental associations are not static but vary across developmental stages, a pattern that warrants further study.

4.5 Implications

Holding urban density constant, the largest benefits from increasing park access accrued in pockets within the central, northeastern, and western neighborhoods (Fig. 6) with moderate-to-high urban density (Fig. 5). This pattern suggests that park investments are most effective in denser areas with greater pedestrian activity, and that benefits are not uniform even within the neighborhood boundaries typically used as units of policy planning. However, if the park–density interaction captures poorer park quality in low-density areas, and vice versa,[23,44] then directing funds toward high footfall city blocks could entrench inequalities.

Moreover, Singapore is dense, with a residential density of 8,300/km^2, and the urban density measure implies a mean of 11,500/km^2, comparable to cities such as New York (11,300/km^2), Tokyo (15,700/km^2), and Barcelona (16,600/km^2), but far above many North American cities, such as Los Angeles (3,200/km^2), Seattle (3,600/km^2), and Toronto (4,400/km^2). Hence, our findings likely do not generalize to lower-density urban sprawls that are more car-dependent.

However, the principle of context-dependent environmental benefits should generalize broadly. Prior inconsistent evidence might reflect such unexamined heterogeneities.[10,12–14,23] Urban planners should therefore consider how parks interact with other environmental features rather than as isolated interventions.

4.6 Limitations and Strengths

The park exposure measures align with time-budgeted access rather than straight-line Euclidean proximity-based measures that ignore urban morphology and travel frictions. However, the land-use inventories lack indicators of park programming and quality.[12,13] The anonymized GPS traces approximate lived experiences at a finer spatial scale, capturing pedestrian co-presence and bustle near home,[32,33] but they may underrepresent the very young and very old.[34]

Anthropometric outcomes were measured longitudinally, but physical activity and actual park use were not observed at comparable scales, so we lack direct behavioral evidence.[10–12,14,19] The temporal resolution of the cohort measurements enabled us to examine age-specific effects. But this is ultimately constrained by the data that ends when the children were about 13–14 years old (at the time of study), and therefore lacks comparisons through later adolescent years.

Finally, repeated cohort measures of outcomes and parks enabled a longitudinal within-child design with adjustment for observed neighborhood selection. These help account for time-invariant family factors, health-seeking behavior, genetic predispositions, and location choice.[38] Nonetheless, our study remains observational,[38] not experimental.[43]

4.7 Conclusion

Following an urban cohort from birth into early adolescence, we found that parks protect against rising BMI in urban pockets with higher pedestrian activity. This context dependency within neighborhoods implies that urban interventions cannot follow a one-size-fits-all approach. Parks woven into active urban settings can be levers for health, but those in quieter areas may remain untapped green spaces. Urban planning should therefore orchestrate built and social environments together, rather than treating them as isolated levers.

References

1 Phelps NH, Singleton RK, Zhou B, Heap RA, Mishra A, Bennett JE, et al. Worldwide trends in underweight and obesity from 1990 to 2022: a pooled analysis of 3663 population-representative studies with 222 million children, adolescents, and adults. The Lancet. 2024 Mar;403(10431):1027-50. Available from: https://doi.org/10.1016/S0140-6736(23)02750-2.

2 Gao L, Peng W, Xue H, Wu Y, Zhou H, Jia P, et al. Spatial–temporal trends in global childhood overweight and obesity from 1975 to 2030: a weight mean center and projection analysis of 191 countries. Globalization and Health. 2023 August 4;19(1):53. Available from: https://doi.org/10.1186/s12992-023-00954-5.

3 Lobstein T, Jackson-Leach R, Powis J, Brinsden H, Gray M. World Obesity Atlas 2023; 2023. World Obesity Federation. Available from: https://data.worldobesity.org/publications/?cat=19.

4 Ochoa-Moreno I, Taheem R, Woods-Townsend K, Chase D, Godfrey KM, Modi N, et al. Projected health and economic effects of the increase in childhood obesity during the COVID-19 pandemic in England: The potential cost of inaction. PLOS ONE. 2024 01;19(1):1-19. Available from: https://doi.org/10.1371/journal.pone.0296013.

5 Simmonds M, Llewellyn A, Owen CG, Woolacott N. Predicting adult obesity from childhood obesity: a systematic review and meta-analysis. Obesity Reviews. 2016;17(2):95-107. Available from: https://onlinelibrary.wiley.com/doi/abs/10.1111/obr.12334.

6 Rooth DO. Obesity, Attractiveness, and Differential Treatment in Hiring. Journal of Human Resources. 2009;44(3):710-35. Available from: https://jhr.uwpress.org/content/44/3/710.

7 Reichert AR. Obesity, Weight Loss, and Employment Prospects: Evidence from a Randomized Trial. Journal of Human Resources. 2015;50(3):759-810. Available from: https://jhr.uwpress.org/content/50/3/759.

8 Ruffle BJ, Shtudiner Z. Are Good-Looking People More Employable? Management Science. 2015;61(8):1760-76. Available from: https://doi.org/10.1287/mnsc.2014.1927.

9 Goulão C, Lacomba JA, Lagos F, Rooth DO. Weight, attractiveness, and gender when hiring: A field

experiment in Spain. Journal of Economic Behavior & Organization. 2024;218:132-45. Available from: https://www.sciencedirect.com/science/article/pii/S0167268123004341.

10 Jia P, Cao X, Yang H, Dai S, He P, Huang G, et al. Green space access in the neighbourhood and childhood obesity. Obesity Reviews. 2021;22(S1):e13100. E13100 OBR-06-20-4551. Available from: https://onlinelibrary.wiley.com/doi/abs/10.1111/obr.13100.

11 Sugar S. The Necessity of Urban Green Space for Children's Optimal Development; 2021. UNICEF Discussion Paper. Available from: https://www.unicef.org/media/102391/file/Necessity%20of%20Urban%20Green%20Space%20for%20Children%E2%80%99s%20Optimal%20Development.pdf.

12 Lachowycz K, Jones AP. Greenspace and obesity: a systematic review of the evidence. Obesity Reviews. 2011;12(5):e183-9. Available from: https://onlinelibrary.wiley.com/doi/abs/10.1111/j.1467-789X.2010.00827.x.

13 Luo YN, Huang WZ, Liu XX, Markevych I, Bloom MS, Zhao T, et al. Greenspace with overweight and obesity: A systematic review and meta-analysis of epidemiological studies up to 2020. Obesity Reviews. 2020;21(11):e13078. E13078 OBR-03-20-4317.R2. Available from: https://onlinelibrary.wiley.com/doi/abs/10.1111/obr.13078.

14 Fyfe-Johnson AL, Hazlehurst MF, Perrins SP, Bratman GN, Thomas R, Garrett KA, et al. Nature and Children's Health: A Systematic Review. Pediatrics. 2021 10;148(4):e2020049155. Available from: https://doi.org/10.1542/peds.2020-049155.

15 Boone-Heinonen J, Casanova K, Richardson AS, Gordon-Larsen P. Where can they play? Outdoor spaces and physical activity among adolescents in U.S. urbanized areas. Preventive Medicine. 2010;51(3):295-8. Available from: https://www.sciencedirect.com/science/article/pii/S0091743510002926.

16 Sallis JF, Cerin E, Conway TL, Adams MA, Frank LD, Pratt M, et al. Physical activity in relation to urban environments in 14 cities worldwide: a cross-sectional study. The Lancet. 2016 May;387(10034):2207-17. Available from: https://doi.org/10.1016/S0140-6736(15)01284-2.

17 Benjamin-Neelon SE, Platt A, Bacardi-Gascon M, Armstrong S, Neelon B, Jimenez-Cruz A. Greenspace, physical activity, and BMI in children from two cities in northern Mexico. Preventive Medicine Reports. 2019;14:100870. Available from: https://www.sciencedirect.com/science/article/pii/S2211335518301797.

18 Nguyen PY, Astell-Burt T, Rahimi-Ardabili H, Feng X. Effect of nature prescriptions on cardiometabolic and mental health, and physical activity: a systematic review. The Lancet Planetary Health. 2023 Apr;7(4):e313-28. Available from: https://doi.org/10.1016/S2542-5196(23)00025-6.

19 Shen L, Sum KK, Kee MZ, Tint MT, Law EC, Yap F, et al. Transient Buffering Effects of Parks Accessibility Against Movement Control Policies on Child Weight Status: A Quasi-Experimental Analysis in Singapore; 2025. Manuscript.

20 Ness AR, Leary SD, Mattocks C, Blair SN, Reilly JJ, Wells J, et al. Objectively Measured Physical Activity and Fat Mass in a Large Cohort of Children. PLOS Medicine. 2007 03;4(3):1-9. Available from: https://doi.org/10.1371/journal.pmed.0040097.

21 Riddoch CJ, Leary SD, Ness AR, Blair SN, Deere K, Mattocks C, et al. Prospective associations between objective measures of physical activity and fat mass in 12-14 year old children: the Avon Longitudinal Study of Parents and Children (ALSPAC). BMJ. 2009;339. Available from: https://www.bmj.com/content/339/bmj.b4544.

22 Kelley GA, Kelley KS, Pate RR. Exercise and adiposity in overweight and obese children and adolescents: a systematic review with network meta-analysis of randomised trials. BMJ Open. 2019;9(11). Available from: https://bmjopen.bmj.com/content/9/11/e031220.

23 Liu GC, Wilson JS, Qi R, Ying J. Green Neighborhoods, Food Retail and Childhood Overweight: Differences by Population Density. American Journal of Health Promotion. 2007;21(4_suppl):317-25.

PMID: 17465177. Available from: https://doi.org/10.4278/0890-1171-21.4s.317.

24 Bell JF, Wilson JS, Liu GC. Neighborhood Greenness and 2-Year Changes in Body Mass Index of Children and Youth. American Journal of Preventive Medicine. 2008;35(6):547-53. Available from: https://www.sciencedirect.com/science/article/pii/S0749379708007344.

25 Richardson EA, Mitchell R. Gender differences in relationships between urban green space and health in the United Kingdom. Social Science & Medicine. 2010;71(3):568-75. Available from: https://www.sciencedirect.com/science/article/pii/S027795361000345X.

26 Wolch J, Jerrett M, Reynolds K, McConnell R, Chang R, Dahmann N, et al. Childhood obesity and proximity to urban parks and recreational resources: A longitudinal cohort study. Health & Place. 2011;17(1):207-14. Health Geographies of Voluntarism. Available from: https://www.sciencedirect.com/science/article/pii/S1353829210001528.

27 Lovasi GS, Schwartz-Soicher O, Quinn JW, Berger DK, Neckerman KM, Jaslow R, et al. Neighborhood safety and green space as predictors of obesity among preschool children from low-income families in New York City. Preventive Medicine. 2013;57(3):189-93. Available from: https://www.sciencedirect.com/science/article/pii/S0091743513001758.

28 Markevych I, Thiering E, Fuertes E, Sugiri D, Berdel D, Koletzko S, et al. A cross-sectional analysis of the effects of residential greenness on blood pressure in 10-year-old children: results from the GINIplus and LISAplus studies. BMC Public Health. 2014 May;14(1):477. Available from: https://doi.org/10.1186/1471-2458-14-477.

29 Schalkwijk AAH, van der Zwaard BC, Nijpels G, Elders PJM, Platt L. The impact of greenspace and condition of the neighbourhood on child overweight. European Journal of Public Health. 2017 03;28(1):88-94. Available from: https://doi.org/10.1093/eurpub/ckx037.

30 Bloemsma LD, Gehring U, Klompmaker JO, Hoek G, Janssen NAH, Lebret E, et al. Green space, air pollution, traffic noise and cardiometabolic health in adolescents: The PIAMA birth cohort. Environment International. 2019;131:104991. Available from: https://www.sciencedirect.com/science/article/pii/S0160412019310335.

31 Warembourg C, Nieuwenhuijsen M, Ballester F, de Castro M, Chatzi L, Esplugues A, et al. Urban environment during early-life and blood pressure in young children. Environment International. 2021;146:106174. Available from: https://www.sciencedirect.com/science/article/pii/S0160412020321292.

32 Matthews SA, Yang TC. Spatial Polygamy and Contextual Exposures (SPACEs): Promoting Activity Space Approaches in Research on Place And Health. American Behavioral Scientist. 2013;57(8):1057-81. PMID: 24707055. Available from: https://doi.org/10.1177/0002764213487345.

33 Perchoux C, Chaix B, Cummins S, Kestens Y. Conceptualization and measurement of environmental exposure in epidemiology: Accounting for activity space related to daily mobility. Health & Place. 2013;21:86-93. Available from: https://www.sciencedirect.com/science/article/pii/S1353829213000117.

34 Lim JZ, Shen L. Neighborhood Mismatch and Visits. Research Paper #18-2022, Asia Competitiveness Institute Research Paper Series. 2022. Available from: https://lkyspp.nus.edu.sg/docs/default-source/aci/acirp202218.pdf.

35 World Bank. Intentional homicides (per 100,000 people) — Singapore; 2025. . Available from: https://data.worldbank.org/indicator/VC.IHR.PSRC.P5?contextual=aggregate&locations=SG.

36 Soh SE, Tint MT, Gluckman PD, Godfrey KM, Rifkin-Graboi A, Chan YH, et al. Cohort profile: Growing Up in Singapore Towards healthy Outcomes (GUSTO) birth cohort study. Int J Epidemiol. 2014 Oct;43(5):1401-9. Available from: http://dx.doi.org/10.1093/ije/dyt125.

37 Shen L, Kee MZL, Huang J, Sum KK, McCrickerd K, Chung G, et al. Parent-specific effects of parks accessibility on child resilience: A longitudinal cohort study; 2024. .

38 Finkelstein A, Gentzkow M, Williams H. Sources of geographic variation in health care: Evidence

from patient migration. Q J Econ. 2016 Nov;131(4):1681-726. Available from: https://doi.org/10.1093/qje/qjw023.

39 Allam Z, Bibri SE, Chabaud D, Moreno C. The 15-Minute City concept can shape a net-zero urban future. Humanities and Social Sciences Communications. 2022 Apr;9(1):126. Available from: https://doi.org/10.1057/s41599-022-01145-0.

40 Lee SE, Lim JZ, Shen L. Segregation Across Neighborhoods in a Small City. Research Paper #07-2021, Asia Competitiveness Institute Research Paper Series. 2021. Available from: https://lkyspp.nus.edu.sg/docs/default-source/aci/acirp202107.pdf?sfvrsn=a862240a_2.

41 Rey S, Anselin L. PySAL: A Python library of spatial analytical methods. Review of Regional Studies. 2007;37(1):5-27. Available from: https://rrs.scholasticahq.com/article/8285.pdf.

42 Buchhorn M, Smets B, Bertels L, Roo BD, Lesiv M, Tsendbazar NE, et al. Copernicus Global Land Service: Land Cover 100m: version 3 Globe 2015-2019: Product User Manual; 2021. Dataset v3.0, doc issue 3.4. Available from: https://doi.org/10.5281/zenodo.4723921.

43 Ludwig J, Sanbonmatsu L, Gennetian L, Adam E, Duncan GJ, Katz LF, et al. Neighborhoods, Obesity, and Diabetes — A Randomized Social Experiment. New England Journal of Medicine. 2011;365(16):1509-19. Available from: https://www.nejm.org/doi/full/10.1056/NEJMsa1103216.

44 Cohen DA, Han B, Derose KP, Williamson S, Marsh T, McKenzie TL. Physical Activity in Parks: A Randomized Controlled Trial Using Community Engagement. American Journal of Preventive Medicine. 2013 Nov;45(5):590-7. Available from: https://doi.org/10.1016/j.amepre.2013.06.015.

Appendix

Table S1|Association between changes in park access and urban density on changes in BMI.

	Dependent variable is yearly change in BMI			
	(1)	(2)	(3)	(4)
Change in parks	-0.007^{**}	-0.007	-0.006	-0.006
	(0.003)	(0.007)	(0.005)	(0.005)
	[−0.013, − 0.000]	[−0.022,0.007]	[−0.017,0.004]	[−0.016,0.004]
	$< p = .040 >$	$< p = .313 >$	$< p = .244 >$	$< p = .237 >$
Urban density				-0.030
				(0.072)
				[−0.176,0.117]
				$< p = .682 >$
(Change in parks) × (Urban density)				-0.021^{***}
				(0.006)
				[−0.033, − 0.009]
				$< p = .001 >$
Mean BMI change	0.309	0.309	0.306	0.306
Std. dev. of change in parks	3.524	2.849	2.843	2.843
Std. dev. of density	—	—	—	0.444
Maternal/child baselines	✓	✓	✓	✓
Origin/destination nbh FE		✓	✓	✓
Child FE			✓	✓
Year FE			✓	✓
Years	14	14	14	14
Neighborhoods	177	163	164	164
Planning areas (Cluster var. 1)	32	30	30	30
No. child observations (Cluster var. 2)	1032	815	835	835
No. child-year observations	11,027	8,475	8,658	8,658

Note: Change in parks is the yearly change in parks within 15 minutes of residence. Urban density is the rasterized GPS traces (in thousands) per 0.1 km^2 hexagonal bin, mean-centered. Child baselines include age in days and sex. Maternal baselines include age (at delivery), ethnicity, college education, low income, and place of birth. Columns (2)–(4) cluster standard errors at the planning area level. Parentheses report standard errors clustered by child and planning area. Square brackets report 95% CI. Angular brackets report p-values. Significance levels: * 0.1 ** 0.05 *** 0.01.

Table S2|Robustness of the association between park access and urban density across anthropometric measures.

	Dependent variable is yearly change in:					
	BMI	zBMI	Weight	zWeight	Height	zHeight
	(1)	(2)	(3)	(4)	(5)	(6)
Change in parks	−0.006	−0.000	−0.008	−0.002	0.007	−0.001
	(0.005)	(0.003)	(0.008)	(0.003)	(0.009)	(0.002)
	[−0.016,0.004]	[−0.007,0.006]	[−0.024,0.007]	[−0.007,0.004]	[−0.011,0.026]	[−0.004,0.002]
	$< p = .237 >$	$< p = .921 >$	$< p = .282 >$	$< p = .524 >$	$< p = .434 >$	$< p = .634 >$
Urban density	−0.030	−0.048	0.076	0.005	0.039	0.032
	(0.072)	(0.037)	(0.088)	(0.044)	(0.222)	(0.054)
	[−0.176,0.117]	[−0.124,0.028]	[−0.104,0.256]	[−0.084,0.095]	[−0.416,0.493]	[−0.079,0.143]
	$< p = .682 >$	$< p = .204 >$	$< p = .396 >$	$< p = .904 >$	$< p = .862 >$	$< p = .561 >$
(Change in parks) × (Urban density)	-0.021^{***}	-0.008^{**}	-0.037^{***}	-0.010^{**}	−0.025	−0.004
	(0.006)	(0.004)	(0.013)	(0.005)	(0.023)	(0.003)
	[−0.033, − 0.009]	[−0.016, − 0.001]	[−0.062, − 0.011]	[−0.020, − 0.001]	[−0.072,0.021]	[−0.010,0.002]
	$< p = .001 >$	$< p = .025 >$	$< p = .007 >$	$< p = .031 >$	$< p = .271 >$	$< p = .183 >$
Height (cm)			0.143^{***}			
			(0.013)			
			[0.117,0.169]			
			$< p = .000 >$			
zHeight				0.107^{***}		
				(0.017)		
				[0.072,0.142]		
				$< p = .000 >$		
Mean BMI change	0.306	0.0441	3.343	0.0573	7.654	0.0316
Std. dev. of change in parks	2.843	2.843	2.843	3.028	2.843	2.843
Std. dev. of density	0.444	0.444	0.444	0.446	0.444	0.444
Maternal/child baselines	✓	✓	✓	✓	✓	✓
Origin/destination nbh FE	✓	✓	✓	✓	✓	✓
Child FE	✓	✓	✓	✓	✓	✓
Year FE	✓	✓	✓	✓	✓	✓
Years	14	14	14	11	14	14
Neighborhoods	164	164	164	161	164	164
Planning areas (Cluster var. 1)	30	30	30	29	30	30
No. child observations (Cluster var. 2)	835	835	835	835	835	835
No. child-year observations	8,658	8,658	8,658	6,561	8,658	8,658

Note: Change in parks is the yearly change in parks within 15 minutes of residence. For comparison, Column (1) reproduces the same estimate for BMI from Column (4) from Tab. S1. The model for Weight (zWeight) additionally adjust for Height (zHeight). All specifications are otherwise the same as Column (4) from Tab. S1. Columns (2)–(4) cluster standard errors at the planning area level. Parentheses report standard errors clustered by child and planning area. Square brackets report 95% CI. Angular brackets report p-values. Significance levels: * 0.1 ** 0.05 *** 0.01.

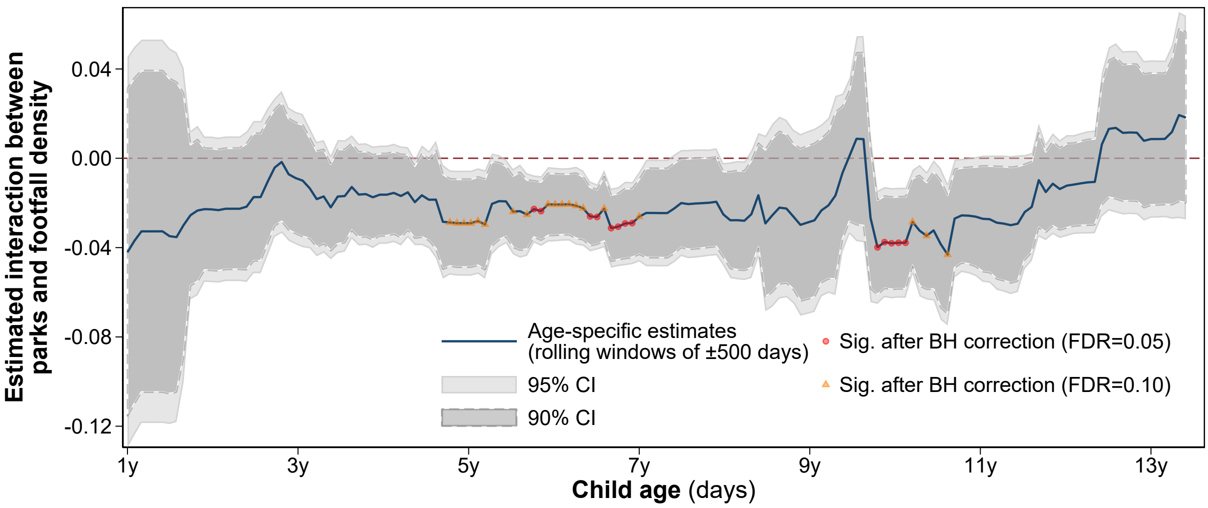


Figure S1|Age-specific parks-density interaction (with multiple hypotheses correction for Fig. 3). Markers indicate estimates that remain statistically significant after applying Benjamini-Hochberg correction (BH correction) for multiple testing: red circles denote significance at a 5% false discovery rate (FDR), while orange triangles indicate significance at a 10% FDR.

Table S3|Association of a binary indicator for increases in park access and urban density on changes in BMI.

	Dependent variable is yearly change in BMI			
	(1)	(2)	(3)	(4)
Increase in parks = 1	−0.066**	−0.038	−0.073*	−0.065*
	(0.031)	(0.038)	(0.037)	(0.033)
	[−0.127, − 0.004]	[−0.115,0.039]	[−0.150,0.003]	[−0.133,0.004]
	$< p = .037 >$	$< p = .321 >$	$< p = .059 >$	$< p = .062 >$
Urban density				0.031
				(0.070)
				[−0.112,0.174]
				$< p = .664 >$
(Increase in parks = 1) × (Urban density)				−0.274***
				(0.075)
				[−0.427, − 0.122]
				$< p = .001 >$
Mean BMI change	0.309	0.309	0.306	0.306
Share with park increase (= 1)	0.183	0.178	0.178	0.178
Maternal/child baselines	✓	✓	✓	✓
Origin/destination nbh FE		✓	✓	✓
Child FE			✓	✓
Year FE			✓	✓
Years	14	14	14	14
Neighborhoods	177	163	164	164
Planning areas (Cluster var. 1)	32	30	30	30
No. child observations (Cluster var. 2)	1032	815	835	835
No. child-year observations	11,027	8,475	8,658	8,658

Note: Increase in parks is a binary indicator for an having an increase in the number of parks within 15 minutes compared to the previous year. All specification is otherwise the same as in Tab. S1. Parentheses report standard errors clustered by child and planning area. Square brackets report 95% CI. Angular brackets report p-values. Significance levels: * 0.1 ** 0.05 *** 0.01.

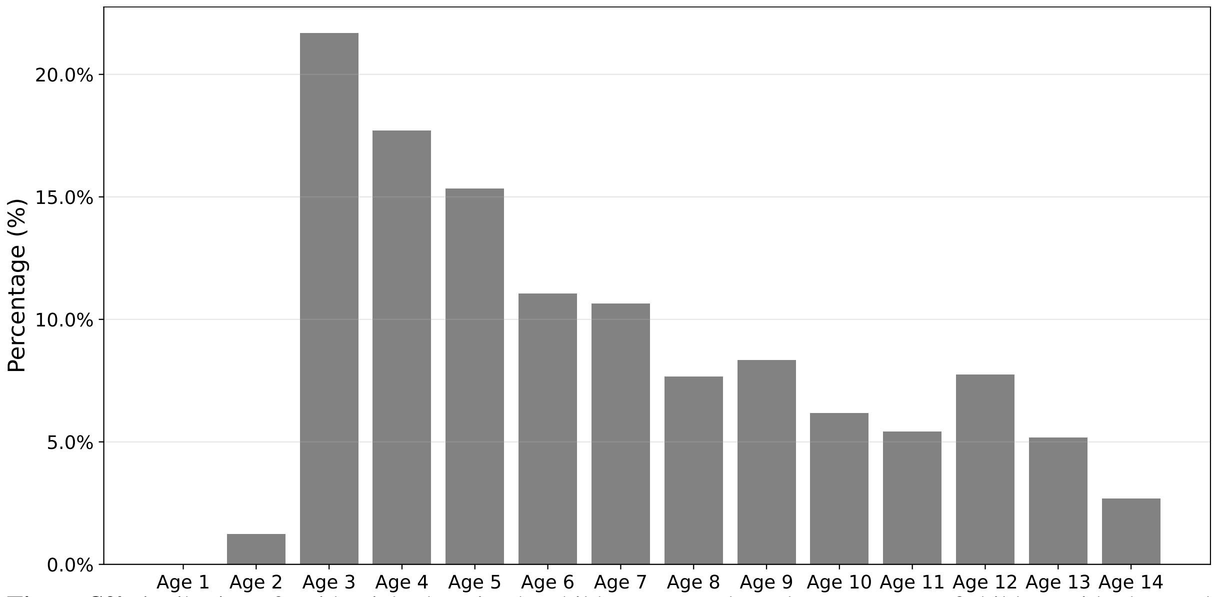


Figure S2|Distribution of residential relocation by child age. Bars show the percentage of children with observed changes in 6-digit postal code between consecutive years.

Table S4|Crosswalk between Planning Areas and Towns/Estates' age classifications.

Planning Area	**HDB Town/Estate**	**Age Category**	**Type**
Punggol	Punggol	Young	Town
Sembawang	Sembawang	Young	Town
Sengkang	Sengkang	Young	Town
Bishan	Bishan	Middle-aged	Town
Bukit Batok	Bukit Batok	Middle-aged	Town
Bukit Panjang	Bukit Panjang	Middle-aged	Town
Bukit Timah	Bukit Timah	Middle-aged	Estate
Choa Chu Kang	Choa Chu Kang	Middle-aged	Town
Hougang	Hougang	Middle-aged	Town
Jurong East	Jurong East	Middle-aged	Town
Jurong West	Jurong West	Middle-aged	Town
Pasir Ris	Pasir Ris	Middle-aged	Town
Serangoon	Serangoon	Middle-aged	Town
Tampines	Tampines	Middle-aged	Town
Woodlands	Woodlands	Middle-aged	Town
Yishun	Yishun	Middle-aged	Town
Ang Mo Kio	Ang Mo Kio	Mature	Town
Bedok	Bedok	Mature	Town
Bukit Merah	Bukit Merah	Mature	Town
Clementi	Clementi	Mature	Town
Downtown Core	Central Area	Mature	Estate
Geylang	Geylang	Mature	Town
Kallang	Kallang/Whampoa	Mature	Town
Marine Parade	Marine Parade	Mature	Estate
Novena	Kallang/Whampoa	Mature	Town
Outram	Central Area	Mature	Estate
Queenstown	Queenstown	Mature	Town
River Valley	Central Area	Mature	Estate
Rochor	Central Area	Mature	Estate
Singapore River	Central Area	Mature	Estate
Tanglin	Central Area	Mature	Estate
Toa Payoh	Toa Payoh	Mature	Town

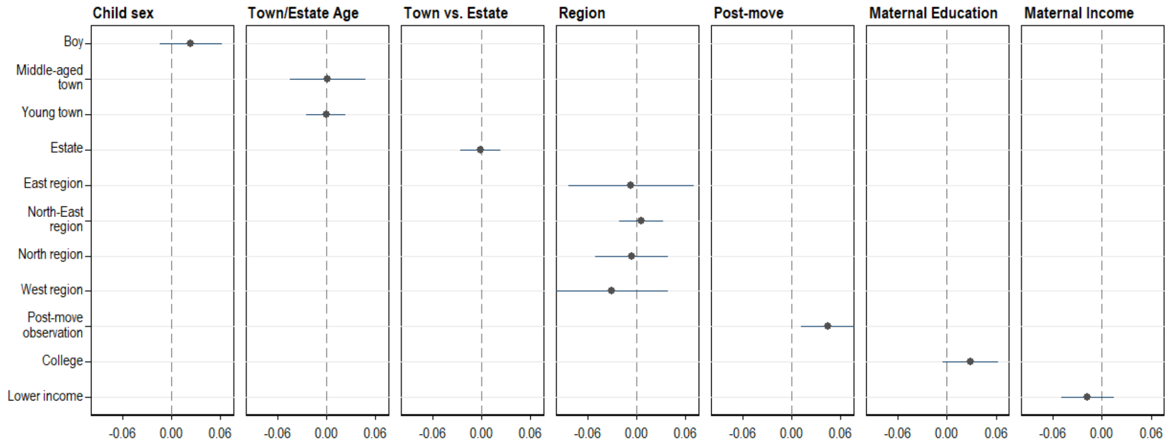
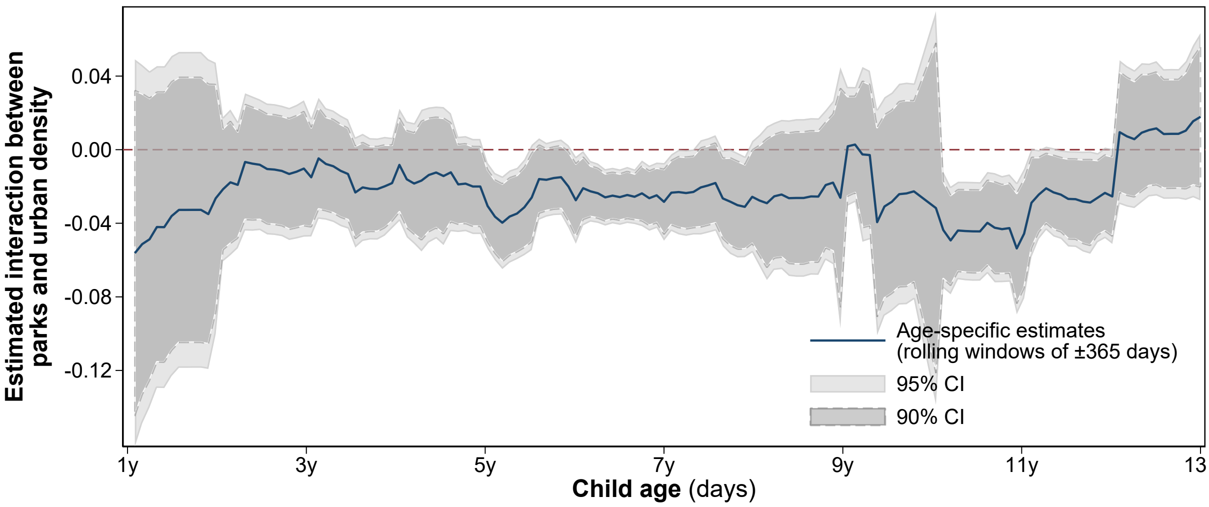
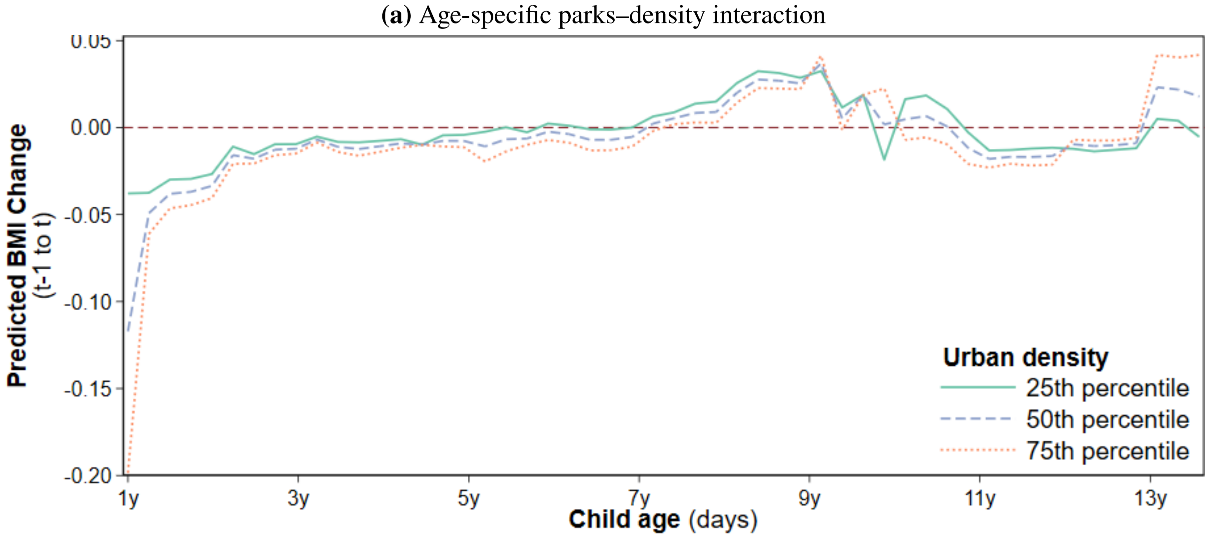


Figure S3|Moderations of the park–density interaction. Each panel plots the triple interaction coefficient(s) from Eq. (1). Coefficients are the differential effect of park access × urban density across levels of each moderator. Reference categories: girl, mature town (Tab. S4), town, West region, non-mover/pre-move observation, no college degree, higher income (> SGD2000/month). Points show coefficient estimates with 95% confidence intervals. Standard errors clustered by individual and planning area.

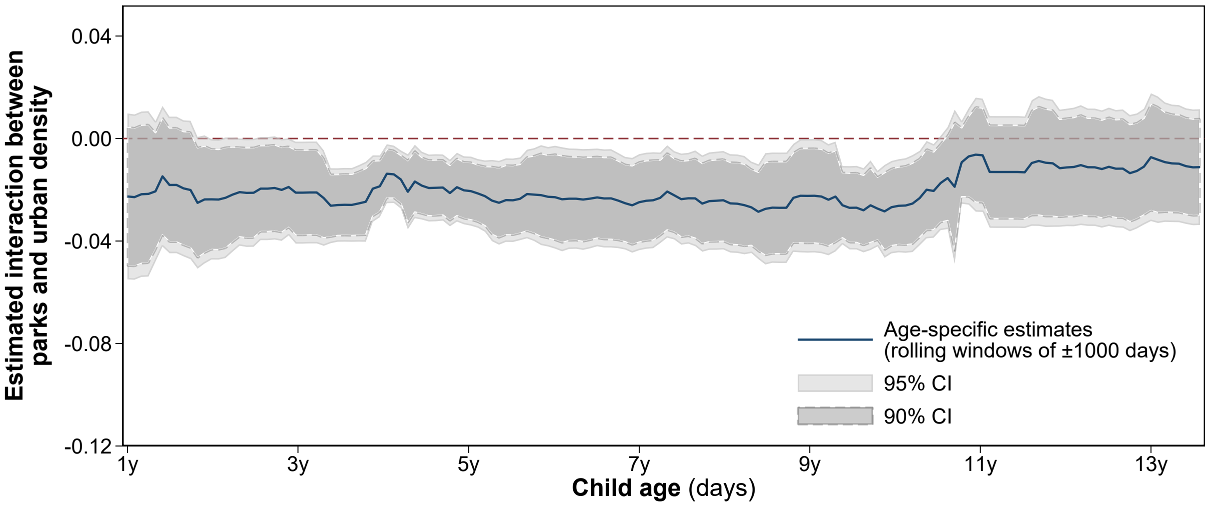


(a) Age-specific parks–density interaction

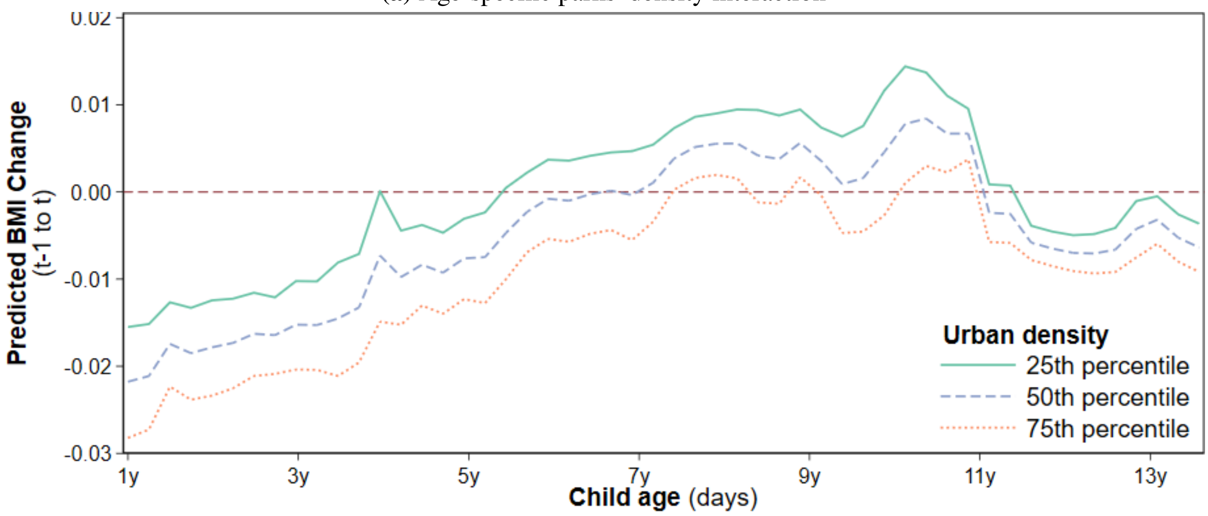


(b) Age-specific parks–density interaction

Figure S4|Age-specific associations between park access, urban density, and BMI. Similar to Fig. 3 except with rolling-window regressions of ±365 days.



(a) Age-specific parks–density interaction



(b) Age-specific parks–density interaction

Figure S5|Age-specific associations between park access, urban density, and BMI. Similar to Fig. 3 except with rolling-window regressions of ±1000 days.