The built environment as determinant of childhood obesity: a systematic literature review

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Abbreviations:

PRISMA	Preferred Reporting Items for systematic Reviews and Meta-Analysis
PROSPERO	International Prospective Register of Systematic Reviews
BMI	Body mass index
BMI z-score	BMI standardised for age and sex
PIAMA	Prevention and incidence of asthma and mite allergy birth cohort
IOTF	International Obesity Task Force
NO_x	Nitrogen oxides
STAMINA	Standard Model Instrumentation for Noise Assessments
$PM_{2.5}$	Particulate matter with diameter less than 2.5 µm
NO_2	Nitrogen dioxide
PM_{10}	Particulate matter with diameter less than 10 µm
SO_2	Sulphur dioxide
ESCAPE	European Study of Cohorts for Air Pollution Effects
CDC	Centers for Disease Control and Prevention
WHO	World Health Organization
UK	United Kingdom
NDVI	Normalized Difference Vegetation Index

Abstract

We evaluated the epidemiological evidence on the built environment and its link to childhood obesity, focusing on environmental factors such as traffic noise and air pollution, as well as physical factors potentially driving obesity-related behaviours, such as neighbourhood walkability and availability and accessibility of parks and playgrounds. Eligible studies were i) conducted on human children below the age of 18 years, ii) focused on body size measurements in childhood, iii) examined at least one built environment characteristic, iv) reported effect sizes and associated confidence intervals, and v) were published in English language. A *z*-Test, as alternative to the meta-analysis, was used to quantify associations due to heterogeneity in exposure and outcome definition. We found strong evidence for an association of traffic-related air pollution (nitrogen dioxide and nitrogen oxides exposure; $p<0.001$) and built environment characteristics supportive of walking (street intersection density; $p<0.01$ and access to parks; $p<0.001$) with childhood obesity. We identified a lack of studies which account for interactions between different built environment exposures or verify the role and mechanism of important effect modifiers such as age.

Introduction

The prevalence of childhood obesity has more than tripled over the last four decades. Latest figures suggest that up to 30 percent of children in Europe are with overweight or obesity.[1] The growing rate of children with overweight and obesity is the most important preventable public health crisis of the 21st century, with serious health, social and economic implications. Obesity in childhood often persists into adulthood with severe consequences for health. An expanding set of chronic diseases has been linked to childhood obesity including increased risk of developing cardiovascular disease, type 2 diabetes and certain cancers, as well as diminished mental health.[2-5]

Obesity is preventable and reversible. Restricting energy intake and increasing energy expenditure have previously been the focus of prevention and treatment strategies. Most efforts and initiatives have, however, so far been unsuccessful at a population level and a broadened approach is warranted.[6] The causes of obesity are multifactorial ranging from individual, household, to policy settings. In this context, place-based obesogenic factors are increasingly being recognised as important determinants of obesity, including the social context, the environment individuals live in, and behaviours linked to modern, urban living.[7] In order to target place-based mitigation approaches, interventions and policy implementations, a clear understanding of the spatial context in which obesity determinants act is needed.[8]

The place we live in has increasingly been recognised as a strong determinant of health, including obesity.[9] In this context, the term ‘built environment’ has been coined to describe the physical and built infrastructure in which people live, learn, work, play, socialise and travel.[10] Within urban settings, the natural infrastructure is an integral part of the wider concept of the built environment. The built environment has strong influences on residents’ behaviours, with physical activity and sedentary lifestyles being the most widely studied.[11] Additionally, environmental pollution linked to the built environment such as air pollution and traffic also have strong impacts on urban health.[12]

This systematic review synthesises the empirical evidence on the built environment as determinant of childhood obesity. We focused on environmental factors including traffic noise and air pollution, as well as physical factors potentially driving obesity-related behaviours, including neighbourhood walkability and availability and accessibility of parks and playgrounds. Supported by a rigorous quality assessment and a focus on objectively measured built environment characteristics, we provide a quantitative synthesis of the updated evidence base with an emphasis on conceptual and methodological aspects, and public health implications.

Methods

Search strategy

We followed the Preferred Reporting Items for Systematic reviews and Meta-Analysis (PRISMA) guidelines and registered the protocol with the International Prospective Register of Systematic Reviews (PROSPERO) database (registration number CRD42020170337). We used a comprehensive and reproducible search strategy to identify peer-reviewed journal articles in the English language, published from inception until February 2020, focusing on three databases: EMBASE, MEDLINE and Web of Science. A preliminary search identified relevant keywords and MeSH terms at the intersection of two concept clusters: “childhood obesity” and “built environment” (Table S1).

Eligibility criteria

Studies were eligible for inclusion if they met the following criteria: (1) Population: Children and/or adolescents under the age of 18 years; (2) Exposure: Objectively measured environmental and physical features of the built environment potentially linked to the onset of obesity; (3) Outcomes: Objectively measured and self-reported body mass index (BMI), or BMI standardised for age and sex (BMI *z*-score); (4) Study design: Observational studies (cross-sectional and longitudinal) quantitatively assessing associations of outcome and exposure. We excluded studies which assessed the built environment as confounder only, those which used self-reported perceived features of the built environment, and studies using controlled experiments in manipulated settings (Table S2). We also excluded studies with an explicit focus on the food environment as this was outside the scope of the review. After the removal of duplicates, articles were screened independently by two reviewers (D.M. and D.F.) against the eligibility criteria, using the online tool Covidence.[13]

Data extraction

Data extraction was performed independently by two reviewers (D.M., E.H.), discrepancies were mediated by D.F. Information was extracted on study characteristics (first author, year, study design, study area, sample size), participant characteristics (age, sex), exposure (built environment characteristic, data collection method), outcome measures (outcome, data collection methods, measure of association), individual- and area-level confounders and main findings (direction and magnitude of association, statistical significance).

Quality assessment

The quality of the eligible studies was assessed independently by two reviewers (D.M., E.H.), discrepancies were mediated by D.F. We used a modified Newcastle-Ottawa scale for quality assessment,[14] which we adapted for the assessment of observational studies. The elements used for the assessment include (1) representativeness of the exposed population, (2) selection of the non-exposed population, (3) objective ascertainment of the exposure, (4) sample size, (5) appropriateness of considered confounding factors, (6) assessment of the outcome, and (7) statistical test used for analysis (Table S3). Stars were assigned for each criterion with a maximum of twelve stars. A score of 0-4 was defined as poor quality, 5-8 as fair quality and 9-12 as good quality. Publication bias was assessed using a funnel plot.

Data synthesis

Due to the heterogeneity in exposure metrics and methodologies used across eligible studies, a meta-analysis was not possible. Instead, we used an alternative methodology to assess and synthesise the strength of associations, the weighted *z*-Test.[15] This approach has previously been used for systematic reviews on the built environment and health[16, 17] and is based on the number of studies with findings in the expected direction and their level of significance. For each study, we assigned a *z*-value based on the level of statistical significance (α) and direction of association (expected direction of association based on research hypothesis vs. unexpected direction of association). If associations were in the expected direction, then $z = 1.96$ for $\alpha = 0.05$, and $z = 1.64$ for $\alpha = 0.10$; if associations were in the unexpected direction, then $z = -1.96$ for $\alpha = 0.05$, and $z = -1.64$ for $\alpha = 0.10$; $z = 0.00$ was assigned to null (statistically not significant) associations with $p > 0.10$. We summed the *z*-value for each reported finding and weighted these by the quality assessment score for each study, divided by the square root of the sum of squared quality assessment scores. To determine the strength of association for each built-environment-outcome combination, a two-tailed *p*-value was computed for each weighted *z*-value with interpretation of weak evidence if $p<0.05$, strong evidence if $p<0.01$ and very strong evidence if $p<0.001$.[16] To avoid overrepresentation of individual studies reporting built environment-outcome associations by different subgroups (e.g., boys/girls, geographic area, age group), we applied fractional weights to each finding so that the sum of the weights across all reported associations was 1.[17] For example, if a study reported a positive association of fine particulate matter with childhood obesity, but that association was significative ($\alpha = 0.05$) only in boys ($z = 1.96$) and not in girls ($z = 0.00$), the *z*-value assigned to the study was $1.96 * 0.5 + 0 * 0.5 = 0.98$. Following the standard set for meta-analysis, associations for each built environment feature-outcome combination were only synthesised if five or more studies reported such associations.

Results

Results are presented separately for each built environment characteristics: (1) traffic noise, (2) air pollution, (3) neighbourhood walkability, and (4) accessibility and availability of parks and playgrounds. PRISMA flow diagrams are shown in Figures S1 to S4, respectively.

Our search initially identified 1192 studies with some studies included in more than one built environment domain. After the removal of duplicates and applying screening criteria, we included four studies on traffic noise and childhood obesity, 14 studies on air pollution, 19 studies on neighbourhood walkability and 28 studies on accessibility and availability of parks and playgrounds. Data extracted for all studies meeting eligibility criteria are presented in Tables S4a to S4d. We did not find evidence for publication bias (Figure S5).

Childhood obesity and traffic noise

Study characteristics

The four studies investigating effects of traffic noise on childhood obesity were recent (2016-2019) longitudinal studies from Northern Europe (Table 1).[18-21] Two studies used national birth cohorts,[19,20] the others longitudinal studies with national coverage. Sample sizes ranged from 3,963 to 40,974 participants. All studies assessed exposure to noise through standard modelling methods, linked to the home addresses of the subjects. Three studies used an implementation of the Nordic prediction method for road traffic noise, one study a national noise standard[18]. Methodologies between studies were generally comparable. The Swedish study[20] obtained height and weight from school and health records and, in part measurements, while the three other studies used height and weight from questionnaires. The Norwegian study[21] accounted for age and sex in the model via interaction terms to explore the effect of noise on BMI trajectory, while all others studies either used a age/sex standardisation of BMI (BMI *z*-score) and/or categorised BMI based on sex and age-specific cut-offs for overweight and obese from the International Obesity Task Force (IOTF). All studies accounted for age, sex and maternal education in analysis, in addition to other study-specific confounders including maternal BMI prior pregnancy,[19-21] parental smoking,[18-20] neighbourhood socioeconomic status[18] and physical activity.[20] One study controlled further for urbanisation and nitrogen oxides (NO_x).[19] Studies used either linear mixed models,[18,21] multiple regression[19] or quantile regression[20] with increasing levels of adjustment. All studies were of high quality with scores of 9 to 10 out of the maximum 12 stars (see Table S5).

Summary of findings

Due to the small number of studies, meta-analysis was not applied, and findings descriptive. Impacts of traffic noise on childhood obesity were observed in three studies, but overall results were mixed and varied by life stage (see Table S4a). Positive associations of road-traffic noise exposure during pregnancy and the risk of being with overweight in school-age children (7/8 years) were observed in Denmark and Norway,[19,21] but not Sweden.[20] For the same age group, no impact of childhood noise exposure on weight was found.[18-21] Wallas et al. (2019), however, studied the effect of traffic noise exposure during adolescence and found a strong association with adolescence BMI between the ages of 8 and 16 years, which was slightly stronger for girls.[20]

Childhood obesity and air pollution

Study characteristics

The majority (n = 11) of the 14 reviewed studies were longitudinal studies, the others cross-sectional. Half of the eligible studies were conducted in the U.S. (n = 7), followed by European (n = 5) and Asian studies (n = 2). The largest sample size was 30,056 children in a cross-sectional study.[22] Longitudinal studies were smaller, also due to a loss to follow-up.[23] Most studies (n = 8) were conducted in urban settings, resulting in ~80% of participants residing in urban areas. Most studies (n = 9) focused on childhood, only three studies on adolescents.[18,24,25] Studies analysed a wide range of air pollutants in relation to childhood obesity. The most studied pollutant was particulate matter with diameter less than 2.5 µm ($PM_{2.5}$) (n = 8), followed by NO_x (n = 7), nitrogen dioxide (NO_2) (n = 6), PM_{10} (n = 5), sulphur dioxide (SO_2) (n = 2), ozone (n = 1) and black carbon (n = 1). All studies assessed air pollution exposure at the home address, one study also at school.[26] Five studies modelled air pollution exposure using dispersion models,[25,27-30] five others used Land Use Regression.[18,26,31-33] Two studies interpolated measurement data from multiple monitoring stations using inverse distance weighting[24,34] and two studies linked measurements from the nearest monitoring station.[22, 35] BMI was used as main outcome in six studies[24,25,27,30,31,34], two longitudinal studies used BMI trajectories[30,32] and seven studies used weight status classification. Different growth charts and guidelines were used to standardize BMI to adjust for age and sex (BMI z-score). The most common was the Centers for Disease Control and Prevention (CDC) growth chart, used in five US studies[24,25,30,31,35] and one study from China[22]. Three studies used the World Health Organization (WHO) growth reference data and one the IOTF indications. Two studies utilized national standards from the UK[29] and Sweden.[28] The majority of studies adjusted for age and sex, one study used Tanner stage,[24] and one only studied four-year old children.[28] Three studies did not adjust for age but used age and sex standardised BMI measures.[26,29,35] Covariates varied widely across studies and included parental socioeconomic status, maternal BMI, birth weight, parental smoking and passive smoking exposure. All studies had a quality rating of good, ranging from 9 to 11 stars (see Table S5).

Summary of findings

To synthesise findings using the z-Test, we combined NO_2 and NO_x results, $PM_{2.5}$ and PM_{10} were considered separately (Table 2). No z-statistics was derived for SO_2, ozone and black carbon due to the small number of studies. Of the eleven studies which looked at NO_2/NO_x, five reported significative associations with BMI-derived outcomes,[18,22,24,25,27] four studies did not find significative results.[26,28,29,33] Two of the studies had mixed results, one found an effect only in boys,[34] in one study the effect dependent on the exposure period.[30] Overall, the association of NO_2/NO_x exposure on childhood obesity was strong with a two-tailed *p*-value from the weighted *z*-value being $p = 0.003$. Overall, there was no statistically significant effect of $PM_{2.5}$ on childhood obesity with $p = 0.10$. Five out of the eight studies investigating $PM_{2.5}$ did not find any significant effect, two showed a positive association,[24,35] and one found an effect only in boys.[31] Only one of the five studies looking at PM_{10} reported an effect,[22] reflected by the *p*-value of 0.15. SO_2 and ozone were associated with increased prevalence of obesity in one of the studies[34] but in another study a higher SO_2 in utero and in childhood was associated with lower BMI at ~13 and ~15 years.[22] Four studies did not find any significant evidence of a link between air pollution and childhood obesity,[26,28,32,33] two of which were conducted in areas of modest level air pollution.[28,32]

Childhood obesity and neighbourhood walkability

Study characteristics

Most of the 19 included studies used a cross-sectional study design ($n = 14$), four were longitudinal and one study included both a longitudinal and a cross-sectional approach.[37] Most studies were conducted in the USA ($n = 12$), three were conducted in Canada[38-40] and the others in Germany,[41] UK,[23] Spain[42] and Israel.[43] Four studies were based on large population samples ($n > 35,000$) reflecting the cross-sectional study designs, one was a longitudinal study conducted in the USA with a small loss during follow-up.[44] Six studies relied on medium sample sizes ($9,440 < n < 14,084$) and the other nine on small sample sizes ($n < 1,000$). Four studies were focused on children (< 7 years old), six included only adolescents and the nine studies included both categories. Among the included studies several methodologies were used to quantify neighbourhood walkability. The most common method ($n = 10$) was the walkability index based on the approach developed by Frank et al. (2006).[45]

The original method by Frank et al. (2006) incorporated land use mix, street connectivity, net residential density, and retail floor area ratios, giving street connectivity twice the weight of the other three variables. Often studies used modified versions of the walkability index, i.e. giving street connectivity the same weight as the other variables, using destinations as proxy for land use, not accounting for the retail floor area ratio or including additional elements such as access to facilities and parks. The three main components of the walkability index (land use mix, street connectivity, net residential density) were often individually analysed. Two studies used the Walk Score,[38,46] a web-based tool (www.walkscore.com) which relies mainly on the distance to various amenities, but also includes population density and road metrics such as block length and intersection density. One study adopted a different approach by deriving a walkability index composed of land use mix, sidewalks, sidewalk buffers, sidewalk/street lighting, other sidewalk elements, traffic lights, pedestrian signal at traffic lights, marked crosswalks, pedestrian crossing and other signage and public transport.[47] Except one study which analysed percentage of body fat as outcome,[48] all studies used BMI-derived outcomes (BMI *z*-score, BMI trajectories, overweight and obesity prevalence), two of which analysed waist circumference[40] and skinfold thickness[49] as additional measures. Sex was always considered as covariate and age was missing only in one study.[36] Other covariates relating to individual, household and neighbourhood confounders were included in the models. The most used were race/ethnicity, parental education, and neighbourhood socioeconomic status. In general, studies were of good quality, with scores ranging from 8 to 11. The main factors which penalized some of the studies were small sample size, low representativeness of the general population and the use of self-reported data.[44,47,50]

Summary of findings

There was limited evidence that the walkability index is linked to childhood obesity ($p = 0.28$), with only one out of ten studies finding significant associations[41] (Table 2). Two further studies showed mixed results based on sex (effect on bodyweight status in girls, but not boys)[39] and geographic area (healthy BMI associated with higher levels of walkability in one of three studied cities).[43] The Walk score was associated with decreased BMI *z*-score in rural but not urban youths in one study,[46] but did not show any significant association in another study.[38] The walkability index based on street element characteristics, however, did identify a significant association with childhood obesity. With regards to individual walkability indicators, street intersection density was the most widely used indicator ($n = 7$). Three studies found significant associations with childhood obesity,[37,44,51] one study found a weak positive association,[52] mixed results were found in two studies, with effects observed in girls but not boys[39] and one out of three studied cities.[43] The *z*-Test revealed strong evidence to support a link between street intersection density and obesity measures ($p = 0.005$). Out of six studies analysing associations with population density, only one study found an effect of lower residential density being linked to higher BMI *z*-score[37] and one study found an effect only in girls. Overall, the evidence did not suggest a link between population density and childhood obesity ($p = 0.23$). Land use mix was only analysed in four studies, with one study finding a significant association.

Childhood obesity and accessibility and availability of parks and playgrounds

Study characteristics

The dominant study design of the 28 included studies was cross-sectional ($n = 20$), the others longitudinal ($n = 8$).[18,29,54-58] One of the longitudinal studies conducted a quasi-experiment which considered a pre-park and post-park time frame and dividing the children into those who live near the park (the exposure group) and those who live further from the park (the control group) to examine how exposure to a newly built park translates to changes in BMI *z*-score over time.[59] Almost half of the studies were conducted in the USA ($n = 13$), ten studies were conducted in Europe, four of which in the UK[29,57,60,61], two studies from Germany[62,63] and Spain[64,65] and one from Netherlands[18] and Lithuania[66]. The sample sizes ranged from 93 to 41,283. Seven studies used small cohorts with less than 1,000 subjects,[48,49,53,54,65,67,68] most studies used medium size cohorts (*n* = 15) not exceeding 7,000 participants, four studies included larger samples over 10,000 participants[69-72] and two studies included very large samples of around 40,000 subjects.[52,73] Five studies considered a wide age range up to 18 years. Seven studies included children under the age of 9 years,[49,60,61,63,66,70,74] and four studies included exclusively adolescents of at least 10 years.[62,65,73,75] Twelve studies included both children and adolescents with ages ranging from 4 to 18 years.

Most studies analysed park accessibility and availability based on children's place of residence, two studies focus on the school environment.[63,72] The definition of the sphere of influence was in 14 studies based on circular or network buffers ranging from 100 to 3,000 meters in radius from the pace of residence, one study which considered a ten miles (16,000 meters) radius.[54] Eight studies based their analysis on official administrative or statistical boundaries and three studies analysed distance from the nearest park, without defining a sphere of influence.[52,59,65]. The remaining studies used neighbourhood area without further specifications on the delimitations. The most used exposure metric was the relative amount of park surface in the spere of influence (*n* = 11). Other studies quantified exposure through the dichotomous variable presence/absence of parks, the number or density of parks, and the distance from the nearest park. Four studies used the satellite-derived Normalized Difference Vegetation Index (NDVI) to quantify the greenness of the surrounding environment. The definition of park/greenspace was inconsistent across studies. Most studies identified areas intended as urban free-usable greenspace. Some studies identified specific features (e.g. children playgrounds), others used a broad approach (e.g. NDVI), considering the total amount of vegetation without distinct function.

The outcomes analysed were BMI *z*-score, BMI trajectories, BMI percentiles and weight status. Anthropometric measures were rarely used: waist circumference (*n* = 3),[56,76,77] waist-to-height ratio (*n* = 1),[56] sum of skinfold (*n* = 1)[49] and percentage body fat (*n* = 2).[48,77]

The quality of the studies was either fair (*n* = 6) or good (*n* = 22). The main reasons for fair quality were small sample sizes, self-reported outcomes (height and weight) or study population scarcely representative of the population (see Table S5).

Summary of findings

Due to the great variability in exposure metrics, we synthesised findings across the following exposure categories: distance to the nearest park (*n* = 9), park area (*n* = 10), number of parks (*n* = 8) and presence/absence of parks (*n* = 5). Only three studies analysed NDVI which was insufficient for meta-analysis according to our criteria (Table 2). The *z*-Test and related *p*-value suggest that there was insufficient evidence to support an association of distance to park and childhood obesity (p = 0.170). Out of the nine studies only one found a significant association.[67] Two studies concluded with mixed findings: one study found a significant association in boys of all ages and girls of high school age, but not in younger girls,[52] one study found an significant association in children living in urban areas but not those in rural areas.[18] The *p*-value suggested weak evidence of an association with percentage of park area ($p = 0.014$). Three studies found significant associations, six studies found no statistically significant effects and two studies had mixed results, with effects only found in boys and older children. The *p*-value showed little evidence of an effect of number or density of parks on childhood obesity ($p = 0.148$). One study found a significant association, five studies did not find significant associations, and one studies reported mixed results with effects only in girls.[69] The intervention study did, however, find an effect in the intervention group which could not be replicated in the control group.[54] We identified strong evidence on the presence of a park within the sphere of influence and childhood obesity ($p < 0.001$). Out of the five studies, four studies found statistically significant effects. Results from the three studies which explored the effect of greenness via the NDVI suggest a potential association in the more proximal environment of less than 250 metres.[18,64,66] Three studies specifically focussed on playgrounds and none of them found statistically significative associations.

Discussion

Impact of built environment characteristics on childhood obesity

We systematically reviewed the epidemiological evidence on the influence of four built environment characteristics on obesity outcomes in children: traffic noise, air pollution, neighbourhood walkability and accessibility and availability of parks and playgrounds. To our knowledge, this is the first systematic review on this topic that applied a systematic synthesise of findings to evaluate the strength of the available evidence.

Studies were generally of high quality, using objectively measured outcome and exposure measures and adjusting for relevant confounders. Some studies, however, had small sample sizes which were not necessarily representative of the overall population. Overall, 42% of studies used longitudinal data, however, the small number of longitudinal studies investigating effects of neighbourhood walkability and parks accessibility should be emphasized.

We found very strong evidence of association of BMI-derived obesity outcomes with NO_2/NOx (p<0.001) and presence/absence of parks in the neighbourhood (p<0.001), strong evidence with intersection density (p<0.01) and some evidence with the amount of park area in the neighbourhood (p<0.05). There was little evidence of an effect on childhood obesity in relation to $PM_{2.5}$, PM_{10}, walkability index, residential density, distance to the nearest park, number of parks and access to playgrounds.

Air pollution has been shown to decrease birth weight[78] and might independently affect weight in childhood through epigenetic and behavioural adaptation. Some hypotheses on the mechanism involved in the exposure both during pregnancy and childhood were highlighted in previous publications: prenatal growth restrictions can lead to growth spurts in early childhood with implications on increased weight into later childhood and adolescence;[79] heavy traffic roads, an important sources of air pollution, might deter active transport and reduce physical activity.[80] Our findings point towards this direction with traffic-related air pollutants NO_2 and NO_x having a strong impact on increased weight in childhood, but not particulate matter ($PM_{2.5}$ and PM_{10}) which is driven to a lesser degree by local traffic.[18] Another explanation could be the biochemical mechanism which emphasizes the role of NO_2 as active oxidant involved in many physiological pathways in the human body which might impact consequently the onset of obesity.[81]

Despite evidence suggesting a link between walkability and physical activity[82], we found little evidence of neighbourhood walkability decreasing BMI-derived outcomes. Intersection density is the only indicator of walkability which showed strong evidence of a negative association with childhood obesity. The central role of this measure in the walkability index has already been highlighted in the original equation by Frank et al. (2006), which gave street connectivity twice the weight of the other variables. Given the same source, road traffic, future studies should explore the effect of collinearity between the walkability components and other traffic-related factors such as traffic noise and air pollution. Studies on walkability were mainly conducted in the United States with only small number of studies from Europe North American cities have a different urban structure compared to European cities and results might not be directly comparable and transferable. This should be explored further in future studies.

We also found strong evidence for the presence (or accessibility) of parks with decreased prevalence of childhood obesity, while studies focusing on playgrounds did not find significative associations. This is supported by findings from Bird et al. (2016) who concluded that parks that emphasize unstructured activities (i.e. with few team sport installations) were associated with lower percentage of truncal fat among children at risk of being with obesity.

Methodological considerations

Some of the included studies investigated more than one built environment characteristics. Several studies explored walkability and parks.[29,48,49,52,70,84] Among the studies which considered walkability and greenspaces, walkability was not statistically significant, except intersection density in boys in one of the studies[48], and greenspace was at least partially associated with weight outcomes in all studies. No multi-exposure interactions were evaluated in these studies, except for a Pearson correlation coefficient between intersection density and park space, which did not show collinearity[48]. Overall, we found a lack of studies which explore the interaction between multiple exposures on childhood obesity. Bloemsma (2019)[18] investigated the combined effect of noise, air pollution and park accessibility. They found that the association of NO_2 with overweight remained after adjustment for noise and greenspace, but the associations between greenspace and overweight weakened substantially after adjustment for NO_2, indicating that NO_2 is driving the relationship. To better understand the complex relationship of multiple built environment characteristics on childhood obesity more evidence is required.

Our review highlighted a strong presence of effect modifiers. Sex was the most studied effect modifier but there was no consistency across studies. Two studies reported an increased effect in boys for the association between air pollution exposure and BMI[31, 34], but one of the studies found also an opposite effect considering waist-to-hip ratio as anthropometric measure, which was statistically significant only in girls[31]. Walkability and intersection density were found to be associated with body weight status in girls but not in boys in one of the studies[39], but in another study, a high level of street connectivity was related to lower percentage of body fat only in boys[48]. The association between park accessibility and obesity was gender-dependent in five studies, of which three showed more significant effects on boys[52, 55, 56] and two on girls[48, 69]. Overall, sex affected the results in nine studies, concluding with an increased effect in boys in five studies, in girls in three studies and with opposite effects depending on the considered anthropometric measure in one of the studies. Age was another common effect modifier, showing differential results in five studies. In one study the exposure to road traffic noise was associated with increased BMI from school age to adolescence, but not at earlier ages, the relation increased in the older age groups.[20] Age also modified the association between greenspace exposure and BMI in four studies (two of them were based on the same sample), always with increased effects in older children[29, 52, 55, 56]. Another effect modifier was urbanisation, with one study finding a negative association between walk score and BMI *z*-score for youths in rural settings and a positive association among urban youths[46], whereas in another study children living in a urban area had a negative association of the distance to the nearest park with weight status and no association for those living in rural areas[18]. No studies analysed effect modification by socioeconomic status, an important omission which could potentially highlight important pathways to health inequalities.

This systematic review assessed the strength of the evidence and identified the role of different elements of the built environment on childhood obesity, consolidated associations and indicating areas in need of further evidence. Our review has some limitations. Due to the observational nature of included studies, no direct causal relationships can be inferred from the results. The absence of sample size restriction in the selection of studies allowed the inclusion of very small cohorts with results potentially not being transferable beyond the specific setting. The fact that some of the studies used self-reported outcomes (weight and height) could also influence the quality of the results due to the introduction of error and bias in the outcome measures. Finally, it was not possible to conduct a meta-analysis due to the large heterogeneity in study results which could have influenced the validity of our findings. Previous reviews on the effect of the physical and built environment on childhood obesity, however, expressed the results only through descriptive synthesis or narrative review. The use of the *z*-Test is a strength which allows us to assess and quantify the strength of the associations.

Conclusion

In summary, we found strong evidence for an association of traffic-related air pollution (nitrogen dioxide and nitrogen oxides exposure; $p<0.001$) and built environment characteristics supportive of walking (street intersection density; $p<0.01$ and access to parks; $p<0.001$) with childhood obesity. Studies on traffic noise had mixed results and were too few to be included in the *z*-Test analysis. Future studies should consider the interactions between different environmental exposures and verify the role of age and sex as an effect modifier.

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References

1 Nittari G, Scuri S, Petrelli F, Pirillo I, di Luca NM, Grappasonni I. Fighting obesity in children from European World Health Organization member states. Epidemiological data, medical-social aspects, and prevention programs. *Clin Ter*. 2019; 170: e223-e30.

2 Umer A, Kelley GA, Cottrell LE, Giacobbi P, Innes KE, Lilly CL. Childhood obesity and adult cardiovascular disease risk factors: a systematic review with meta-analysis. *BMC Public Health*. 2017; 17: 683-83.

3 Juonala M, Magnussen CG, Berenson GS, *et al.* Childhood adiposity, adult adiposity, and cardiovascular risk factors. *New Engl J Med*. 2011; 365: 1876-85.

4 Lauby-Secretan B, Scoccianti C, Loomis D, Grosse Y, Bianchini F, Straif K. Body fatness and cancer - viewpoint of the IARC working group. *New Engl J Med*. 2016; 375: 794-98.

5 Zametkin AJ, Zoon CK, Klein HW, Munson S. Psychiatric aspects of child and adolescent obesity: a review of the past 10 years. *J Am Acad Child Adolesc Psychiatry*. 2004; 43: 134-50.

6 Han JC, Lawlor DA, Kimm SYS. Childhood obesity. *Lancet*. 2010; 375: 1737-48.

7 Galvez MP, Pearl M, Yen IH. Childhood obesity and the built environment. *Curr Opin Pediatr*. 2010; 22: 202-07.

8 Oshan TM, Smith JP, Fotheringham AS. Targeting the spatial context of obesity determinants via multiscale geographically weighted regression. *Int J Health Geogr*. 2020; 19: 11.

9 Letarte L, Pomerleau S, Tchernof A, Biertho L, Waygood EOD, Lebel A. Neighbourhood effects on obesity: scoping review of time-varying outcomes and exposures in longitudinal designs. *BMJ Open*. 2020; 10: e034690.

10 Barton H, Grant M. A health map for the local human habitat. *J R Soc Promo Health*. 2006; 126: 252-53.

11 Masoumi HE. Associations of built environment and children's physical activity: a narrative review. *Rev Eenviron Health*. 2017; 32: 315-31.

12 Pyko A, Eriksson C, Oftedal B, *et al.* Exposure to traffic noise and markers of obesity. *Occup Environ Med*. 2015; 72: 594.

13 Covidence systematic review software VHI, Melbourne, Australia. Available at www.covidence.org.

14 Wells GA, Shea B, O'Connell S, Peterson J, V Welch, M Losos, P Tugwell. The Newcastle-Ottawa Scale (NOS) for assessing the quality of nonrandomised studies in meta-analyses. 2012. Available from: *http://www.ohri.ca/programs/clinical_epidemiology/oxford.asp*.

15 Whitlock MC. Combining probability from independent tests: the weighted Z-method is superior to Fisher's approach. *J Evol Biol*. 2005; 18: 1368-73.

16 Chandrabose M, Rachele JN, Gunn L, *et al.* Built environment and cardio-metabolic health: systematic review and meta-analysis of longitudinal studies. *Obes Rev*. 2019; 20: 41-54.

17 Cerin E, Nathan A, van Cauwenberg J, *et al.* The neighbourhood physical environment and active travel in older adults: a systematic review and meta-analysis. *Int J Behav Nutr Phy*. 2017; 14: 15.

18 Bloemsma LD, Wijga AH, Klompmaker JO, *et al.* The associations of air pollution, traffic noise and green space with overweight throughout childhood: The PIAMA birth cohort study. *Environ Res*. 2019; 169: 348-56.

19 Christensen JS, Hjortebjerg D, Raaschou-Nielsen O, Ketzel M, Sorensen TIA, Sorensen M. Pregnancy and childhood exposure to residential traffic noise and overweight at 7 years of age. *Environ Int*. 2016; 94: 170-76.

20 Wallas A, Ekstrom S, Bergstrom A, *et al.* Traffic noise exposure in relation to adverse birth outcomes and body mass between birth and adolescence. *Environ Res*. 2019; 169: 362-67.

21 Weyde KV, Krog NH, Oftedal B, *et al.* A longitudinal study of road traffic noise and body mass index trajectories from birth to 8 Years. *Epidemiology*. 2018; 29: 729-38.

22 Dong GH, Qian Z, Liu MM, *et al.* Ambient air pollution and the prevalence of obesity in chinese children: The seven northeastern cities study. *Obesity*. 2014; 22: 795-800.

23 Wilding S, Ziauddeen N, Smith D, Chase D, Roderick P, Alwan NA. Associations of area characteristics at birth with overweight and obesity among school-aged children in the south of England: an analysis of a population-based cohort. *Lancet*. 2019; 394: S98.

24 Alderete TL, Habre R, Toledo-Corral CM, *et al.* Longitudinal associations between ambient air pollution with insulin sensitivity, beta;-cell function, and adiposity in Los Angeles Latino Children. *Diabetes*. 2017; 66: 1789-96.

25 McConnell R, Shen E, Gilliland FD, *et al.* A longitudinal cohort study of body mass index and childhood exposure to secondhand tobacco smoke and air pollution: The Southern California Children's Health Study. *Environ Health Persp*. 2015; 123: 360-66.

26 de Bont J, Casas M, Barrera-Gomez J, *et al.* Ambient air pollution and overweight and obesity in school-aged children in Barcelona, Spain. *Environ Int*. 2019; 125: 58-64.

27 Jerrett M, McConnell R, Wolch J, *et al.* Traffic-related air pollution and obesity formation in children: A longitudinal, multilevel analysis. *Environ Health*. 2014; 13: 49.

28 Frondelius K, Oudin A, Malmqvist E. Traffic-related air pollution and child BMI-A study of prenatal exposure to nitrogen oxides and body mass index in children at the age of four years in Malmo, Sweden. *Int J Env Res Pub He*. 2018; 15: 2294.

29 Wilding S, Ziauddeen N, Smith D, Roderick P, Chase D, Alwan NA. Are environmental area characteristics at birth associated with overweight and obesity in school-aged children? Findings from the SLOPE (Studying Lifecourse Obesity PrEdictors) population-based cohort in the south of England. *BMC Med*. 2020; 18: 43.

30 Kim JS, Alderete TL, Chen Z, *et al.* Longitudinal associations of in utero and early life near-roadway air pollution with trajectories of childhood body mass index. *Environ Health*. 2018; 17: 64.

31 Chiu YH, Hsu HH, Wilson A, *et al.* Prenatal particulate air pollution exposure and body composition in urban preschool children: Examining sensitive windows and sex-specific associations. *Environ Res*. 2017; 158: 798-805.

32 Fleisch AF, Aris IM, Rifas-Shiman SL, *et al.* Prenatal exposure to traffic pollution and childhood body mass index trajectory. *Front Endocrinol*. 2019; 10.

33 Fioravanti S, Cesaroni G, Badaloni C, Michelozzi P, Forastiere F, Porta D. Traffic-related air pollution and childhood obesity in an Italian birth cohort. *Environ Res*. 2018; 160: 479-86.

34 Huang JV, Leung GM, Schooling CM. The association of air pollution with body mass index: evidence from Hong Kong's "Children of 1997" birth cohort. *Int J Obes*. 2019; 43: 62-72.

35 Mao G, Nachman RM, Sun Q, *et al.* Individual and joint effects of early-life ambient PM2.5 exposure and maternal prepregnancy obesity on childhood overweight or obesity. *Environ Health Persp*. 2017; 125: 067005.

36 Kligerman M, Sallis JF, Ryan S, Frank LD, Nader PR. Association of neighborhood design and recreation environment variables with physical activity and body mass index in adolescents. *Am J Health Promot*. 2007; 21: 274-77.

37 Duncan DT, Sharifi M, Melly SJ, *et al.* Characteristics of walkable built environments and BMI z-scores in children: evidence from a large electronic health record database. *Environ Health Persp*. 2014; 122: 1359-65.

38 Shahid R, Bertazzon S. Local spatial analysis and dynamic simulation of childhood obesity and neighbourhood walkability in a major Canadian city. *AIMS Public Health*. 2015; 2: 616-37.

39 Spence JC, Cutumisu N, Edwards J, Evans J. Influence of neighbourhood design and access to facilities on overweight among preschool children. *Int J Pediatr Obes*. 2008; 3: 109-16.

40 Colley RC, Christidis T, Michaud I, Tjepkema M, Ross NA. An examination of the associations between walkable neighbourhoods and obesity and self-rated health in Canadians. *Health Rep*. 2019; 30: 14-24.

41 Gose M, Plachta-Danielzik S, Willie B, Johannsen M, Landsberg B, Muller MJ. Longitudinal influences of neighbourhood built and social environment on children's weight status. *Int J Env Res Pub He*. 2013; 10: 5083-96.

42 Molina-Garcia J, Queralt A, Adams MA, Conway TL, Sallis JF. Neighborhood built environment and socio-economic status in relation to multiple health outcomes in adolescents. *Prev Med*. 2017; 105: 88-94.

43 Hagani N, Moran MR, Caspi O, Plaut P, Endevelt R, Baron-Epel O. The relationships between adolescents' obesity and the built environment: Are they city dependent? *Int J Env Res Pub He*. 2019; 16: 1579.

44 Xue H, Cheng X, Jia P, Wang Y. Road network intersection density and childhood obesity risk in the US: a national longitudinal study. *Public Health*. 2020; 178: 31-37.

45 Frank LD, Sallis JF, Conway TL, Chapman JE, Saelens BE, Bachman W. Many pathways from land use to health: associations between neighborhood walkability and active transportation, body mass index, and air quality. *J Am Plann Assoc*. 2006; 72: 75-87.

46 Stowe EW, Hughey SM, Hallum SH, Kaczynski AT. Associations between walkability and youth obesity: differences by urbanicity. *Child Obes*. 2019; 15: 555-59.

47 Slater SJ, Nicholson L, Chriqui J, Barker DC, Chaloupka FJ, Johnston LD. Walkable communities and adolescent weight. *Am J Prev Med*. 2013; 44: 164-68.

48 Hsieh S, Klassen AC, Curriero FC, *et al.* Built environment associations with adiposity parameters among overweight and obese Hispanic youth. *Preventive Medicine Reports*. 2015; 2: 406-12.

49 Lovasi GS, Jacobson JS, Quinn JW, Neckerman KM, Ashby-Thompson MN, Rundle A. Is the environment near home and school associated with physical activity and adiposity of urban preschool children? *J Urban Health*. 2011; 88: 1143-57.

50 Sallis JF, Conway TL, Cain KL, *et al.* Neighborhood built environment and socioeconomic status in relation to physical activity, sedentary behavior, and weight status of adolescents. *Prev Med*. 2018; 110: 47-54.

51 Jia P, Xue H, Cheng X, Wang Y. Association of neighborhood built environments with childhood obesity: Evidence from a 9-year longitudinal, nationally representative survey in the US. *Environ Int*. 2019; 128: 158-64.

52 Yang Y, Jiang Y, Xu Y, Mzayek F, Levy M. A cross-sectional study of the influence of neighborhood environment on childhood overweight and obesity: Variation by age, gender, and environment characteristics. *Prev Med*. 2018; 108: 23-28.

53 Potwarka LR, Kaczynski AT, Flack AL. Places to play: Association of park space and facilities with healthy weight status among children. *J Commun Health*. 2008; 33: 344-50.

54 Armstrong B, Lim CS, Janicke DM. Park Density Impacts Weight Change in a Behavioral Intervention for Overweight Rural Youth. *Behav Med*. 2015; 41: 123-30.

55 Sanders T, Feng X, Fahey PP, Lonsdale C, Astell-Burt T. Greener neighbourhoods, slimmer children Evidence from 4423 participants aged 6 to 13 years in the Longitudinal Study of Australian children. *Int J Obes*. 2015; 39: 1224-29.

56 Sanders T, Feng X, Fahey PP, Lonsdale C, Astell-Burt T. Green space and child weight status: does outcome measurement matter? Evidence from an Australian longitudinal study. *J Obes*. 2015; 2015: 194838.

57 van der Zwaard BC, Schalkwijk AAH, Elders PJM, Platt L, Nijpels G. Does environment influence childhood BMI? A longitudinal analysis of children aged 3-11. *J Epidemiol Commun H*. 2018; 72: 1110-16.

58 Wolch J, Jerrett M, Reynolds K, *et al.* Childhood obesity and proximity to urban parks and recreational resources: A longitudinal cohort study. *Health Place*. 2011; 17: 207-14.

59 Goldsby TU, George BJ, Yeager VA, *et al.* Urban park development and pediatric obesity rates: A quasi-experiment using electronic health record data. *Int J Env Res Pub He*. 2016; 13: 411.

60 Poole R, Moon G. What is the association between healthy weight in 4-5-year-old children and spatial access to purposefully constructed play areas? *Health Place*. 2017; 46: 101-06.

61 Schalkwijk AAH, van der Zwaard BC, Nijpels G, Elders PJM, Platt L. The impact of greenspace and condition of the neighbourhood on child overweight. *Eur J Public Health*. 2018; 28: 88-94.

62 Lange D, Wahrendorf M, Siegrist J, Plachta-Danielzik S, Landsberg B, Muller MJ. Associations between neighbourhood characteristics, body mass index and health-related behaviours of adolescents in the Kiel Obesity Prevention Study: A multilevel analysis. *Eur J Clin Nutr*. 2011; 65: 711-19.

63 Schule SA, Fromme H, Bolte G. Built and socioeconomic neighbourhood environments and overweight in preschool aged children. A multilevel study to disentangle individual and contextual relationships. *Environ Res*. 2016; 150: 328-36.

64 Dadvand P, Villanueva CM, Font-Ribera L, *et al.* Risks and benefits of green spaces for children: a cross-sectional study of associations with sedentary behavior, obesity, asthma, and allergy. *Environ Health Persp*. 2014; 122: 1329-35.

65 Gutierrez-Zornoza M, Sanchez-Lopez M, Garcia-Hermoso A, Gonzalez-Garcia A, Chillon P, Martinez-Vizcaino V. Active commuting to school, weight status, and cardiometabolic risk in children from rural areas: the Cuenca study. *Health Educ Behav*. 2015; 42: 231-39.

66 Petraviciene I, Grazuleviciene R, Andrusaityte S, Dedele A, Nieuwenhuijsen MJ. Impact of the social and natural environment on preschool-age children weight. *Int J Env Res Pub He*. 2018; 15: 449.

67 Manandhar S, Suksaroj TT, Rattanapan C. The association between green space and the prevalence of overweight/ obesity among primary school children. *Int J Occup Env Med*. 2019; 10: 1-10.

68 Ohri-Vachaspati P, Lloyd K, DeLia D, Tulloch D, Yedidia MJ. A closer examination of the relationship between children's weight status and the food and physical activity environment. *Prev Med*. 2013; 57: 162-67.

69 Morgan Hughey S, Kaczynski AT, Child S, Moore JB, Porter D, Hibbert J. Green and lean: Is neighborhood park and playground availability associated with youth obesity? Variations by gender, socioeconomic status, and race/ethnicity. *Prev Med*. 2017; 95: S101-S08.

70 Lovasi GS, Schwartz-Soicher O, Quinn JW, *et al.* Neighborhood safety and green space as predictors of obesity among preschool children from low-income families in New York City. *Prev Med*. 2013; 57: 189-93.

71 McCarthy SM, Hughey SM, Kaczynski AT. Examining Sociodemographic Differences in Playground Availability and Quality and Associations with Childhood Obesity. *Child Obes*. 2017; 13: 324-31.

72 Wasserman JA, Suminski R, Xi J, Mayfield C, Glaros A, Magie R. A multi-level analysis showing associations between school neighborhood and child body mass index. *Int J Obes*. 2014; 38: 912-18.

73 Nesbit KC, Kolobe TA, Arnold SH, Sisson SB, Anderson MP. Proximal and distal environmental correlates of adolescent obesity. *J Phys Act Health*. 2014; 11: 1179-86.

74 Potestio ML, Patel AB, Powell CD, McNeil DA, Jacobson RD, McLaren L. Is there an association between spatial access to parks/green space and childhood overweight/obesity in Calgary, Canada? *Int J Behav Nutr Phy*. 2009; 6: 77.

75 Wall MM, Larson NI, Forsyth A, *et al.* Patterns of obesogenic neighborhood features and adolescent weight: A comparison of statistical approaches. *Am J Prev Med*. 2012; 42: e65-e75.

76 Rossi CE, Correa EN, das Neves J, *et al.* Body mass index and association with use of and distance from places for physical activity and active leisure among schoolchildren in Brazil. Cross-sectional study. *Sao Paulo Med J*. 2018; 136: 228-36.

77 Juonala M, Harcourt BE, Saner C, *et al.* Neighbourhood socioeconomic circumstances, adiposity and cardiometabolic risk measures in children with severe obesity. *Obes Res Clin Pract*. 2019; 13: 345-51.

78 Smith RB, Fecht D, Gulliver J, *et al.* Impact of London's road traffic air and noise pollution on birth weight: retrospective population based cohort study. *Brit Med J*. 2017; 359: j5299.

79 Kim JH, Lee C, Sohn W. Urban natural environments, obesity, and health-related quality of life among hispanic children living in Inner-City neighborhoods. *Int J En Res Pub He*. 2016; 13: 121.

80 Su JG, Jerrett M, McConnell R, *et al.* Factors influencing whether children walk to school. *Health Place*. 2013.

81 Kirsch M, Korth HG, Sustmann R, Groot Hd. The pathobiochemistry of nitrogen dioxide. *Biol Chem*. 2005.

82 Terrón-Pérez M, Molina-García J, Martínez-Bello VE, Queralt A. Relationship between the physical environment and physical activity levels in preschool children: a systematic review. *Curr Environ Health Rep*. 2021; 8: 177-95.

83 Bird M, Datta GD, van Hulst A, Cloutier MS, Henderson M, Barnett TA. A park typology in the QUALITY cohort: Implications for physical activity and truncal fat among youth at risk of obesity. *Prev Med*. 2016; 90: 133-38.

84 Saelens BE, Sallis JF, Frank LD, *et al.* Obesogenic neighborhood environments, child and parent obesity: the Neighborhood Impact on Kids study. *Am J Prev Med*. 2012; 42: e57-64.

680