The Upstream Environment for the Obesity Epidemic

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Abstract. While individual behaviour and genetics are major risk factors for obesity, for health prevention it is important also to consider upstream influences on obesity, stemming from the urban physical and social environment. Environmental features such as urban sprawl, food deserts and varying access to exercise and green space, affect both activity and diet, and hence body weight. Thus many studies show environmental impacts on obesity, and on related outcomes such as diabetes, with these impacts being socially and spatially differentiated: with worse food and exercise access in lower income areas. Analytical frameworks for the obesity epidemic should reflect wide geographic differences in incidence and prevalence between regions and small areas, including spatial clustering. A case study of changing obesity in US counties shows widening inequalities and persistently high obesity clustering in some regions.


1. Introduction: The Obesity Epidemic

The obesity epidemic continues across the world, and is now recognised as a major public health issue, though this recognition is only relatively recent (James, 2009). In the US, the prevalence of obesity in 2015-16 was 40%, affecting about 93 million adults, though the upward trend is irregular (Flegal et al, 2012). The trend extends to the third world, with an estimating doubling in global obesity levels since 1980 (Bhurosy and Jeewon, 2014; Fox et al, 2019). Obesity is a major risk factor for cardiovascular disease, diabetes, and hypertension, and hence a major factor for increased health care costs (Biener et al, 2017).

While genetic factors, possibly linked to ethnicity, and adverse health behaviours, influence susceptibility to obesity (Stryjecki, et al 2018; Riveros-McKay et al, 2019), they cannot in themselves account for the rapid increases in obesity. Instead, behavioural changes linked to changes in the food and exercise environments are also relevant to increased obesity (Faith, and Kral, 2006; Caballero, 2007; Cummins and MacIntyre, 2006; Egger and Swinburn, 1997). Physical activity is declining, and sedentary behaviour increasing, both in developed societies, and lower and middle-income countries (Gaskin and Orellana, 2018). Dietary changes, such as growing fast food consumption and the “nutrition transition” (Popkin, 2015) are also relevant.

2. Comorbidities of Obesity

Obesity is a risk factor for increased morbidity and mortality, particularly for cardiovascular disease, diabetes and hypertension, but also for cancer and other chronic diseases, including liver and kidney disease and osteoarthritis. The impact of obesity and overweight on health and mortality is recognised in disease burden studies (Must et al, 1999).
Regarding overall mortality, there is some debate about impacts of overweight (BMI 25-<30) and grade 1 obesity (BMI 30-<35), with Flegal et al (2013) reporting excess mortality only at obesity grades 2 and above. However, the study by the Global BMI Mortality Collaboration (2016) reports no mortality benefit from any grade of overweight or obesity, and finds a log-linear increase in mortality as BMI increases compared to a reference BMI of 20-25.

Among the diseases linked to obesity, the recent spread of diabetes has been most evident, and like obesity has been characterised as an epidemic (e.g. Zimmet, 2017; Unnikrishnan et al, 2017). The diabetes epidemic affects low and middle income countries, such as India and China, as well as higher income developed societies. In the US, around 9% of adults (or 30 million) have diabetes according to a report by CDC (2017), and similar levels are reported in China (Yang et al, 2010) and India (Health Issues India, 2019). In LMIC countries diabetes is generally higher in urban settings, and hence is associated with urbanisation (Gassasse et al, 2017; Ramachandran et al, 1999).

The impact of obesity on the risk of diabetes, particularly type 2 diabetes (T2D), is well-established, though diabetes is multifactorial, and many other risk factors than obesity or BMI per se are relevant. For example, among normal weight subjects, diabetes incident risk is especially related to waist size (Carnethon et al, 2012). Population ethnic composition also relevant to variations diabetes risk. Thus raised diabetes risk among south Asian groups has been attributed to increased insulin resistance, even adjusting for adiposity (McKeigue et al, 1991). There is also increased research on diabetes risk through epigenetic changes that can be transmitted from one generation to another thus reinforcing increases in diabetes.

Despite such qualifications, the majority of T2D subjects are obese, and higher obesity grades elevate the risk of diabetes, as does longer established obesity. Abdullah et al (2010) report an overall relative risk (RR) of 7.2 for diabetes among obese persons compared to those of normal weight, and for overweight a RR of 3.0. Among different obesity indices, waist-to-height ratio may offer better predictions of diabetes risk than waist circumference or BMI (Ashwell, and Browning, 2011). Potential biophysical mechanisms include: abdominal obesity causing fat cells to release pro-inflammatory reducing sensitivity to insulin; obesity triggering metabolic changes that cause adipose tissue to release increased amounts of fatty acids, glycerol, hormones, and pro-inflammatory cytokines involved in developing insulin resistance.

3. Environment and Obesity

A major research effort has focused on the impacts on obesity of the urban built environment and its obesogenic aspects. Thus Cummins and McIntyre (2006) refer to the “over-emphasis on the role of individual health behaviours, which has tended to ignore the influence of the complex social and physical contexts in which individual behavioural decisions are made. Such critiques have led to a new focus on ‘environmental’ exposures that encourage excessive food intake and discourage physical activity”. Environmental exposures are partly invoked to explain wide contrasts in obesity prevalence between income groups. Prominent themes in
this work are the impacts of urban sprawl, of food deserts and access to healthy food outlets, and of inequity in access to exercise opportunities.

3.1 Sprawl

Impacts of the built environment on both physical activity and diet have been framed especially in terms of neighbourhoods with high walkability and connectivity, as opposed to car dependent environments associated with urban sprawl. Sprawl relates to land use patterns associated with recent suburban car-dependent development, though the concept has many facets and varying definitions. Thus a literature and conceptual review of sprawl by Galster et al (2001) found no common definition and relatively few attempts at operationalisation. These authors posit eight distinct dimensions: density, continuity, concentration, clustering, centrality, nuclearity, mixed uses, and proximity.

Thus sprawl is generally characterised as low density suburban development, with low connectivity, car dependence, low walkability, and discouraged physical activity. This land use pattern is distinct from that in more central city areas, or in compact mixed-use development, with relatively high connectivity and walkability, and high levels of active commuting (Kashef, 2011). As examples of land use effects on obesity (and diabetes), Creatore et al (2016) find adverse trends in these outcomes in less walkable Ontario neighbourhoods over the period 2001-12, but no such trends in the most walkable neighbourhoods; while Flint and Cummins (2016) find obesity linked to car-only commuting.

3.2 Food Access

The food environment and access to healthy food outlets is another feature of the urban environment that has been linked to growing obesity, and also to sprawl (Mead, 2008; Hamidi, 2019). Food type is relevant to the concept of food deserts, typified as areas with low access to supermarkets, and hence to fresh fruit and vegetables, and more likely to be deprived or ethnic majority areas (Fleischhacker et al, 2011). For example, Larsen and Gililand (2008) find low-income residents of inner-city neighborhoods in London (Ontario) had poorer access to supermarkets than high-income residents, and that inequalities in access to supermarkets had increased.

A concomitant concept is that of “food swamps” (Cooksey-Stowers et al, 2017) whereby low-income areas have food access biased to fast food outlets and convenience stores offering especially foods high in fat, salt, or sugar (HFSS) (Food Foundation, 2019). For child obesity, school location in relation to fast food outlets is therefore potentially important (Alviola et al, 2014).

Consumption of healthy food in deprived areas is also affected by relative costs of healthy food as against HFSS foods (Drewnowski and Specter, 2004). Thus Jones et al (2014) report that since 2002, healthier foods have been consistently more expensive than less healthy ones, with a growing gap between them.
3.3 Exercise Access and Physical Activity

Access to exercise opportunities and natural space is associated with greater physical activity and improved health; for example, Angraal et al (2019) show an adverse impact on cardiovascular mortality of diminished access to exercise opportunities. However, access to such opportunities is socially and spatially unequal. Thus the location of urban green space and parks is typically biased to areas containing higher income and white ethnic groups. In particular, in the US, non-white and low-income groups, generally live in the urban core or in inner suburbs where green space is limited and/or poorly maintained, while upper income groups live on the suburban periphery with more abundant green space (Wolch et al, 2014).

Regarding exercise opportunities (and hence levels of physical activity vs sedentary behaviour), Timperio et al (2010) show that BMI among older children is reduced by a higher density of public open spaces designated for sport or recreation.

3.4 Neighbourhood Social Environment

Neighbourhood impacts on obesity and diabetes extend to the social environment, including area deprivation, social capital, crime perceptions and neighbourhood safety (Lee et al, 2019a). Thus impacts of area deprivation may occur even after controlling for adverse impacts of lower individual socio-economic status (Diez-Roux et al, 1997), sometimes described as “deprivation-amplification” (Cummins and Macintyre, 2006). The widespread adoption of multilevel perspectives is based on a premise that characteristics of communities are potentially related to cardiovascular outcomes, and risk factors, independently of individual-level variables. Hence one should seek to elucidate their independent and combined effects.

Regarding crime, there are plausible pathways through which neighbourhood crime levels influence obesity, with impacts on activity and sedentary behaviour acting as mediating or intermediate variables in the association (Richardson et al, 2017; An et al, 2017).

Regarding social capital, Wu et al (2018) find that adults with higher network diversity and high generalized trust have lower obesity risk, while social capital also impacts on obesity-related behaviours, including smoking, diet, and physical activity. Social capital is generally lower in deprived areas, and social capital may mediate the typically negative relationship between neighbourhood deprivation and self-rated health (Verhaeghe and Tampubolon, 2012).

3.5 Environment and Diabetes

Given the downstream obesity response to environmental factors, similar environmental impacts on diabetes have been reported, though with some specificity also.

Thus Bravo et al (2019) consider quality of the built environment as a potential influence on T2D, beyond the usual research focus on food and physical activity access. Environmental quality is likely to mediate the impact of area deprivation on obesity and diabetes prevalence.

Den Braver et al (2018) also mention urban residence per se (as opposed to rural residence) as a risk factor for diabetes. This may be related to adverse impacts of the urban physical
environment, especially pollution, on diabetes risk (Rajagopalan, Brook, 2012; O'Donovan, Cadena-Gaitán, 2018). Migration to city areas may also enhance diabetes risk (Ruiz-Alejos et al, 2018).

Other studies report similar risk factors in environment-diabetes ecological studies as in environment-obesity studies. Thus Dendup et al (2018) refer to walkability, physical activity resources, and access to green space as protective factors reducing diabetes risk.

4 Spatio-Temporal Aspects of Obesity

Trends to higher levels of obesity are highly disparate at sub-national levels. For example, at state level in the US, adult obesity rates in 2018 range nearly two-fold from 39.5% in Mississippi to 23% in Colorado. Variations at small area scales, such as county or zip code level, are much wider.

In addition many studies show spatial clustering in obesity (and related outcomes) at small area levels (e.g. Huang et al, 2015; Schuurman et al, 2009), and such spatial concentrations tend to be persistent (Joost et al, 2016). Such spatial clustering reflects clustering in risk factors, namely sociodemographic aspects of different areas (compositional impacts in the terminology of multilevel studies), and contextual effects, such as food and exercise access, and sprawl patterns (Schneider et al, 2017).

From a spatio-temporal perspective, one may be interested in the persistence of high obesity risk in certain areas ((Joost et al, 2016), in future spatial predictions (Guo et al, 2018), and in epidemic models and the role of collective vs individual behaviour in the obesity epidemic (e.g. Gallos et al, 2012).

To exemplify spatial clustering and persistent clustering, we consider obesity prevalence data for 3141 US counties from the Behavioral Risk Factor Surveillance System (BRFSS) for four periods, 2005-07, 2008-10, 2011-13, and 2014-16. Figure 1 maps out obesity prevalence in the final period (for the US continental counties), with break points at quintiles.

We first consider spatial clustering in 2014-16 using the local Moran statistic as an local index of spatial association or LISA (Anselin et al, 2006). In particular, this assesses which areas can be classed as the centre of “high-high” clusters, whereby both the area itself and the areas surrounding it have significantly raised obesity. Figure 2 shows the location of these clusters as well as other possible cluster types under the LISA cluster scheme. Figure 2 shows that whereas county clusters with high obesity concentrate in south east US, clusters with low obesity are in the west and mountain states.

To exemplify spatio-temporal modelling of the obesity epidemic in the USA, we use a Bayesian disease mapping approach (Lawson & Lee, 2017) in BUGS (Lunn et al, 2009). Denote age standardised obesity rates in county i (=1,...,3141) and period t (=1,...,4) as $r_{it}$. These are modelled as

$$r_{it} = \beta_0 + s_i + d_i t + u_{it},$$
where $\beta_0$ is the intercept, and $s_i$ are spatially correlated effects measuring the obesity level in period 1, as in the scheme of Besag et al (1991). The $d_i$ are spatially correlated linear growth rates, while the $u_{it}$ are iid normal errors. This is an analogue of the varying intercepts, varying slopes model used in analysing longitudinal and multilevel data, but allows for spatial clustering (as evident in Figures 1 and 2).

Figure 3 maps out the estimated growth rates $d_i$, and shows that the highest growth in obesity to be in the same counties which have the highest obesity according to Figures 1 and 2. This suggests both persistent spatial inequalities in obesity, and a widening of such inequalities.

Conclusion: Unresolved Issues and Future Perspectives

There is by no means a consensus in the literature about the impacts of the urban environment on obesity and related outcomes, and debate continues. For example, on the fundamental question of the relative importance of diet and physical activity, some studies emphasize physical activity (Fisher et al, 2013: Blair et al 2013), while some stress dietary changes (Malhotra et al, 2015; Luke et al, 2013). Regarding debate on specific environment impacts, and in particular the impact of sprawl, Eid et al (2008) consider panel survey data on obesity status and particularly the role of propensity to obesity. They characterise the sprawl effect in terms of levels of mixed use, and find a negative correlation between mixed-use and obesity, after controlling for observable individual characteristics. However, they report that “once we take advantage of the panel dimension of our data to control for unobserved propensity to be obese, the correlation between obesity and mixed-use also vanishes”. In related work considering self-selection of residential location, Plantinga and Bernell (2007) find that individuals who move to denser locations lose weight, but that BMI affects the choice of a dense or sprawling location, such that dense locations are unlikely to be selected by high BMI individuals.

Regarding food access and location of healthy as against unhealthy food outlets [a supply side influence], sceptical studies have instead focused on the reliance on observational data to establish the role of the food environment (Hall, 2018), and on demand differences between income groups. Thus Allcott et al (2017) considered supermarket location, and concluded that “the causal impact of unhealthful food supply is small, relative to either the overall obesity rate or the nutrition-income relationship.” Other studies have reported equivocal findings on the impact of access to fast food outlets, with Fraser et al (2010) reporting "conflicting results between obesity/overweight and fast food outlet availability". Snowdon (2018) found a lack of evidence to support official policies (in the UK) to restrict location of fast food outlets near schools. In a review of 74 studies of the relationship between the density and proximity of fast food outlets and the prevalence of obesity, Snowdon reports that only fifteen (20%) found a positive association between the proximity and/or density of fast food outlets and obesity/body mass, while 44 (60%) found no positive association.

Therefore future research has a role in clarifying such unresolved aspects of the environment-obesity nexus. In terms of quantitative analysis of obesity, especially its spatio-temporal
aspects, there is more scope to investigate geographic heterogeneity, for example in terms of spatially varying impacts of ecological factors on obesity prevalence and obesity related mortality (Lee et al, 2019b; Wen et al, 2010). Spatio-temporal modelling might also be used to analyse the varying pace of the obesity epidemic, and the considerable national variation in obesity levels (Hruby & Hu, 2015).

References


Figure 1 Obesity Prevalence, US Continental Counties (2014-16)
Figure 2 US Continental Counties, Obesity 2014-16, Cluster Patterns
Figure 3 Obesity Prevalence Growth Rates (%), US Continental Counties, 2005-07 to 2014-16