Relocating obesity with multiauthor ethnography

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Abstract
Reflecting on the World Health Organization’s (WHO) account of obesity and recent developments in ethnography, I advocate for a collaborative, multiauthor approach to studying obesity and, more broadly, chronic disease. To illustrate this, I show how recent ethnographies of obesity and metabolism have convincingly challenged and reframed the WHO’s account of obesity. I further suggest that future ethnographic studies of obesity (and chronic disease) could expand their analytical scope – without sacrificing a critical and people-centred approach – through coordination and collaboration. A multiauthor approach to obesity research would increase the capacity of ethnography to demonstrate the many conditions that must be fulfilled for a person to become ‘obese’, productively foregrounding how ‘obesity’ emerges out of a web of social, economic, political, chemical, and historical connections. This would enable a more comprehensive understanding of the uneven emergence of obesity (and other chronic diseases) worldwide.

Keywords
obesity, political ecology, ethnographic theory, medical anthropology, methodology
Introduction
Reflecting on the World Health Organization’s (WHO) account of obesity and recent developments in ethnography and biomedicine, in this think piece I advocate for a collaborative, multiauthor approach to studying obesity and, more broadly, chronic disease. Recently, there appears to be a growing recognition within global health and biomedical circles of the causal significance of public policies in the production of ‘obesity’ and chronic diseases. However, in presenting public policies as ‘disease-causing agents’ biomedically and global health organisations (such as the WHO) often uncritically reproduce stigmatising assumptions about large bodies and privilege biomedical perspectives as ‘fundamental’, while problematically presenting simplistic understandings of ‘society’ as fact. The most effective way to challenge, complicate, and provide more rigorous alternative understandings of these problematic accounts of ‘obesity’, I argue, is through multisited, multiauthor ethnographies.

To situate this discussion, I first review the WHO’s definition of obesity in terms of BMI (body mass index), and then explore Harris Solomon’s (2016) ethnography of metabolism in Mumbai to illustrate how race and risk were used to justify changing the boundaries for ‘normal’ and ‘obese’ in India. Second, I review the WHO’s description of the ‘fundamental cause’ of obesity and its model of development as naturally ‘obesogenic’, pointing out some of the limitations of this model. I then discuss Emily Yates-Doerr’s (2015) ethnography of the production of obesity in Guatemala to illustrate how economic policy and biomedical imperialism can be understood as ‘disease-causing agents’. These ethnographies convincingly explore and challenge universalistic accounts of the causation of ‘obesity’ and other metabolic illnesses. However, these accounts are necessarily restricted to their particular locations. In view of these limitations, I conclude by suggesting that ethnographic studies of obesity and chronic disease should continue to expand their analytical scope temporally (such as through archival research) and geographically (such as through multisited, multinational projects) – without sacrificing a critical and people-centred approach – through coordination and collaboration in multiauthor projects (Biehl 2016). The benefits of a multiplicity of authorship, I suggest, come from an increased capacity to document the many conditions that must be fulfilled for a given person to become ‘obese’.

\[1\] See the editorial by The Lancet Diabetes & Endocrinology (2017) for an example.

\[2\] I mean ‘archive’ in a broad and inclusive sense, and I understand that potential archives could be collections of letters, memoirs, etc. An archive does not need to be a formal institution entitled ‘archive’ (see Garcia 2017).
Understanding BMI as a globally manufactured and locally adapted metric

Before exploring how the WHO frames obesity and overweight, I will first examine body mass index (BMI) and its use in the diagnosis of obesity. The WHO’s (2018) ‘Obesity and Overweight’ fact sheet illustrates a common and influential biomedical framing of obesity. In this document, obesity and overweight are defined as an ‘abnormal or excessive fat accumulation that may impair health’. The diagnosis of either of these conditions is dependent on an above-normal BMI, which is calculated by taking a person’s mass (in kilograms) and dividing it by their height (in metres) squared. As BMI relies only on a person’s weight and height it is relatively simple to calculate, and accordingly, given its simplicity, the fact sheet acknowledges that BMI is only a ‘rough guide’.

The creation of BMI by biomedical scientists enabled two different effects: first, it created a standardised scale that could be used to quantify the prevalence of obesity; second, it created a medical category that reproduced normative assumptions regarding what ‘weighing too much’ is. However, the process for determining the boundaries of the BMI categories is more dependent on the deployment of racialised science than one might expect. By this I mean that the BMI categories have been optimised to predict risk of heart disease or diabetes for different ‘races’ or ‘ethnicities’ due to metabolic illnesses occurring at higher rates in ‘thin’ East Indians. A review of the trope of the ‘thin-fat Indian’, which gained prominence with a photograph published by the *Lancet* in 2004 will illustrate how, when breakdowns occur in the usage of the ‘rough guide’ of BMI, racial categories can be invoked to stabilise its existence (Yajnik and Yudkin 2004).

The ‘thin-fat Indian’ trope – or the ‘Y-Y paradox’, as the authors termed it – describes the disparate physiques of the two authors, Yajnik and Yudkin. Both coauthors have a BMI of 22.3 kg/m², yet the white European author has a body fat percentage of 9.1 percent while the Indian author has a body fat percentage of 21.2 percent. The authors use this discrepancy to highlight some of the inconsistencies between bodily form as measured by the BMI metric and ‘true’ body fat percentage as measured through medical imaging (Yajnik and Yudkin 2004). However, the paradox runs somewhat deeper, as the Indian author is described as ‘fat’, despite possessing a ‘thin form’ because upon further biochemical interrogation, his body was shown to metabolically mimic a ‘fat’ body (Solomon 2016, 32; Yajnik 2018). The authors suggest that, while ‘genes’ may have played a role in the

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3 For adults, the ‘normal’ BMI is 18.5 to 24.9 kg/m², the ‘overweight’ BMI is 25 to 29.9 kg/m², and the ‘obese’ BMI is 30 kg/m² or greater.
development of the Y-Y paradox, it was more likely that ‘early life malnutrition’ and a ‘low birth weight’ were the likely origins of the discrepancy (Yajnik and Yudkin 2004). These suggestions led to the hypothesis that the early maternal conditions of foetal development, and the mother’s diet or stress, could profoundly shape a child’s metabolism and bodily form later in life: as the Indian author Yajnik stated, ‘Indians are born “thin-fat”’ (Yajnik 2018, 471; see also Yajnik et al. 2003).

The construct of the ‘thin-fat’ Indian eventually created a new form of racialised overweight that physicians, biomedical researchers, and the global health community deployed in their understanding of obesity in India. Four years after the publication of the Y-Y paradox, a consensus statement by Indian physicians shifted the BMI thresholds for obesity and overweight in India, from 30 to 25 kg/m² and from 25 to 23 kg/m² respectively (Misra et al. 2009; Solomon 2016, 33). According to the consensus statement, the reason for this shift was that many metabolic illnesses, such as type 2 diabetes, were found by Indian epidemiologists to be occurring in ‘thin’ individuals, which they believed meant that the discrepancy between ‘thin’ bodies with ‘fat’ (in other words, pathological) metabolisms should be resolved through a harmonised definition of a ‘fat body’ that correlated with a ‘fat metabolism’ (Misra et al. 2009; Yajnik 2018). These reconfigurations were not isolated; indeed, they were based upon the WHO’s earlier revised standards for India and at least seven additional regions (The Lancet 2004). Unfortunately, the WHO explained these reconfigurations in terms of racial differences ascribed to ‘Indians’ or other racialised categories, as opposed to a more nuanced and biologically grounded account. This reproduced what Anthony Ryan Hatch (2016, 61–75) has described as the ‘scientific racism of metabolism’, by presenting some Indian bodies as possessing mysterious or mystical ‘Indian genes’ that made them ‘thin-fat’.

In this section I make explicit how the alteration of the BMI-defined risk groups of ‘obesity’ and ‘overweight’ occurs by describing how risk categories are formed from a continuous variable. One of the main goals of the formulation of a statistical category is ensuring that the category matches its description (and vice versa), such that a given ‘low risk category’ actually corresponds to a ‘real’ lower likelihood of developing the characteristic one is ‘at risk of’; the extent to which these two properties overlap is described as a given risk group’s predictive value. The problem with BMI in India was that the categories of interest had a sufficiently low predictive value, so much so that it challenged the usefulness of the current BMI categories and potentially the utility of the BMI metric itself. Given that the numeric

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4 It is worthwhile to note that in this sense, having a BMI of any number constitutes a given ‘risk state’ and the ‘diagnosis of risk’ (cf. Aronowitz 2015, 16, 21–43).
thresholds for overweight and obesity are significantly determined in relation to their predictive value for epidemiologists, the BMI thresholds for the diagnosis of ‘overweight’ and ‘obesity’ could be (and eventually were) shifted to more accurately represent the probability of being diagnosed with a metabolic illness. While this process of shifting the categorical boundaries of risk on the BMI scale did maximise the predictive value of the BMI metric, it did not necessarily challenge the epidemiologists’ belief that measuring BMI is the best way to predict metabolic illness. Instead, that belief was taken as the premise of the entire process of optimisation and enabled the epidemiologists to alter the BMI categories to align them with their beliefs that BMI should track a risk of metabolic disease or a ‘fat metabolism’. Unfortunately, the premise that ‘large bodies’ should be ‘risky’ continues to be taken for granted by the WHO, the global health community, and the biomedical community, regardless of how well suited it is for that task, the human costs of marginalising non-normative bodies, and the racialising premises of the science justifying its validity.

Examining the WHO’s obesity ‘fundamentalism’

Having provided an overview of the construction of BMI, the metric used to diagnose obesity, I wish to return to the WHO’s representation of obesity and its causes in its ‘Obesity and Overweight’ fact sheet (World Health Organization 2018). I do so in order to explore how the premise that ‘large bodies should predict metabolic illness’ enables a problematic narrative of the global ‘obesity epidemic’. These problems are immediately visible in the ‘key facts’ section that begins the document, whose urgent tone is meant to compel belief and action. The sheet leads with the claim that ‘worldwide obesity has nearly tripled since 1975’ and that ‘in 2016, more than 1.9 billion adults, 18 years and older, were overweight. Of these over 650 million were obese’. Next, it states that in many countries ‘overweight and obesity kills more people than underweight’. It concludes with the brief sentence ‘Obesity is preventable’. However, in the section entitled ‘What are common health consequences of overweight and obesity?’ it describes obesity and overweight as merely a ‘major risk factor for noncommunicable diseases’, such as cardiovascular disease, diabetes, musculoskeletal disorders, and some cancers. These two rather different portrayals of obesity – first as the cause of more deaths than underweight, and then second as a ‘major risk factor’ with no direct symptoms of its own – highlights a tension within biomedical discussions of obesity arising from the assumption that obesity should cause metabolic disease or directly ‘kill’ without sufficient biological evidence to substantiate a statistical correlation.

The fact sheet then presents a simple model of how larger bodies are produced: ‘[t]he fundamental cause of obesity and overweight is an energy imbalance between calories consumed and calories expended’ (World Health Organization 2018). This framing is similar to BMI in its simplicity, and by presenting an atomised biochemical understanding of
‘obesity’ as the ‘fundamental’ account, it renders other historical, social, or economic causal pathways secondary.\footnote{While both BMI and calories are quantitative constructs, it is important to note that being numerical does not prevent them from having dynamic social lives. In particular, the notion of a ‘calorie’ presents a level of constancy and clarity regarding how energy is metabolised by bodies globally, which may not always be the case (Camacho and Ruppel 2017; Hargrove 2007).} Given obesity’s ‘fundamental cause’, the WHO claims that ‘an increased intake of energy-dense foods that are high in fat; and an increase in physical inactivity’ are the origins of the obesity epidemic. The WHO adds that ‘[c]hanges in dietary and physical activity patterns are often the result of environmental and societal changes associated with development’, presenting ‘development’ as a naturally obesogenic process (World Health Organization 2018).

By presenting the solution to obesity as learning to be balanced in a ‘developed world’, the WHO locates the problem within an abstract sedentary and overconsuming body in an ‘obesogenic environment’. However, a significant body of literature suggests that this model is too simplistic. As some critical researchers have suggested, it discards the roles that endocrine disruptors, chronic stress, and food insecurity play in altering metabolic homoeostasis (Wells and Siervo 2011; Wells 2013; Stenvinkel 2015; Darbre 2017). The WHO’s account ignores these other influences or renders them subordinate to the abstract notion of ‘energy’. If this model is uncritically taken as fact in the creation of public health policy, it could ultimately support treatments for ‘obesity’ that view development as unchangeable, denying the possibility of effective social, economic, or political interventions, and instead favouring individualistic treatments that aim (often ineffectively) to promote weight loss (Warin et al. 2017).

Making obesity in Guatemala

Rigorous ethnography can compellingly complicate and challenge narratives such as those presented by the WHO with strong explanations and direct evidence. Emily Yates-Doerr’s ethnography of obesity and overweight in Guatemala is one such example. In 2001, following various free trade agreements and economic adjustments, Coca-Cola began transforming the environment, economy, social relations, and inhabitants of Todos Santos, a highland village (Yates-Doerr 2015). As Coca-Cola representatives came to Todos Santos and painted their red-and-white logo everywhere in the village, the availability and consumption of Coca-Cola rapidly increased. In 2006, when Yates-Doerr returned, following the ratification of the Central American Free Trade Agreement she found that a large subsidiary of Wal-mart opened in the closest city, where its mass-produced food products
were entering rural markets and homes. Yates-Doerr (2015, 12) notes that each time she returned to Guatemala more “healthy cooking classes” appeared as reported rates of previously unfamiliar metabolic illnesses accelerated. Indeed, while many members of the global health community label metabolic illness ‘diseases of modernity’ or a product of development, Yates-Doerr (2015, 13) argues that ‘such a label is far too simple’.

In her later fieldwork in Xela, Guatemala’s second largest city, Yates-Doerr observed that in the face of increasing rates of metabolic illnesses, the physicians and nutrition scientists who worked there increasingly endeavoured to instil in the residents of Xela and the surrounding areas the ‘objective knowledge’ that their bodies, understandings of health, and social relations were diseased, and hence needed to be changed (Yates-Doerr 2015, 137–52). For many of Yates-Doerr’s informants, health was constituted by phenomenology, relationality, and care rather than numbers (Yates-Doerr and Carney 2016). Healthy meals were produced to taste, using intuitive embodied measurements learnt over years of practice (Yates-Doerr 2015, 8). They were also produced for families or communities, not individuals (Yates-Doerr and Carney 2016). Tastes and the timing of meals were negotiated and had to be socially coordinated; individual ‘diets’ were simply impossible. Health also was corporeal, as Yates-Doerr notes: ‘Many people told me that a fullness of stomach as well as of figure had been desirable for as long as they could remember. A decade earlier, women had taught me how to wrap a skirt around my waist in such a way as to appear plump’ (Yates-Doerr 2015, 6).

Nonetheless, reliant on BMI, health care providers diagnosed larger bodies in Guatemala as ‘overweight’ or ‘obese’. As Yates-Doerr has shown in her research in Guatemala, understandings of bodies, health, and healthy food ran, at times, against biomedical notions of ‘health’. Therefore, difficulties arose in Guatemala as the dietary strategies implemented there to treat ‘overweight’ and ‘obesity’ were designed for application in ‘Western’ countries and presupposed certain arrangements of society and certain social norms (Yates-Doerr 2015; cf. Mintz 1986 for a discussion of how food shapes social configurations). Some Guatemalan health care providers working in diet and nutrition research centres believed that dietary rearrangement was the best solution to obesity. Accordingly, physicians provided obesity patients with individualised prescriptions listing prescribed quantities of ingredients that could only be purchased at the new supermarkets (Yates-Doerr 2015, 162). These interventions generally failed.

The labelling of ‘obesity’ as a disease of development or modernity takes for granted the assumption that these processes occur in a naturally neoliberal or ‘Western’ manner (Escobar 2011). But it is not clear that deregulating markets, allowing Coca-Cola to aggressively market its products, and providing space for Wal-mart are necessary features of development; they could instead be reframed as potential ‘disease-causing agents’ (Packard 2016, 261–64). By taking economic policies as ‘natural’ rather than pathological and focusing
on individual-level energy imbalance as the fundamental cause of obesity, we are left with a worldview that ignores how deeply integrated human beings are with economic policy (Gálvez 2018). Indeed, the WHO’s worldview prioritises the targeting of the individual with little hope of efficacy, in place of working directly for healthier trade policy. In doing so the WHO takes for granted the belief that large bodies are the problem. It also presents a simplistic model that ‘obesity’ is fundamentally caused by an energy imbalance because individuals fail to adapt to the inevitably ‘obesogenic’ social configurations necessitated by ‘development’.

Multisited, multiauthor obesity research

The WHO’s account of obesity has remained influential despite relying on racialist explanations of biological variance, a flat and simplistic understanding of ‘development’ as obesogenic, and an abstracted model of metabolism. In contrast, critical accounts by anthropologists and geographers Gálvez (2018), Solomon (2016), Yates-Doerr (2015), and Guthman (2011, 2012; Mansfield and Guthman 2015) locate obesity’s causes beyond the body proper. These authors identify ‘metabolic shifts’ both within the bodies of their interlocutors and in the societies that they study (Moore 2017).

As their findings suggest, the WHO’s account is too reductive to explain the cultural or historical emergence of obesity. In part, this is because it lacks the temporal scope to explain the multigenerational causation of obesity suspected to be occurring in India, for example (Krishnaveni and Yajnik 2017; Yajnik 2018). Chronic diseases such as type 2 diabetes or ‘obesity’ are not caused in a moment, but appear to emerge, as Yates-Doerr (2015) notes, over longer stretches of time – sometimes decades. To document the effects of changing conditions on obesity and related conditions, research needs to prioritise deep longitudinal data. Anthropologists are well positioned to expand their temporal scope of their ethnographic field research and have already productively engaged with archival and other historical sources. Given the necessity of contextualising local changes in relation to larger global trends, anthropologists could also continue to engage with historical scholarship, such as world history, the history of global health, or economic history, to enhance the insights derived from their ethnographic and archival research.

Both historians and anthropologists have noted that the implementation of trade agreements, biomedicine, and global health interventions are not everywhere the same (Anderson 1998, 2014; Livingston 2012; Packard 2016; Street 2014). Moreover, transformations in one location are often deeply linked to changes elsewhere. For example, the North American Free Trade Agreement (NAFTA) transformed not only Mexico but also Canada and the United States of America; such changes require analyses that extend beyond
‘the local’ (Gálvez 2018). However, there is still a tendency in the ethnography of obesity to only study it in one location, which is then contextualised in terms of global-local relations. This represents an important limitation in anthropology’s capacity to study the relational production of ‘obesity’ globally.6 While this limitation arising from studying a single location could be resolved through single-author multisited ethnography, doing so might sacrifice the depth of knowledge and connections gained from long-term fieldwork in a single location.7 Multiauthor, multisited ethnographic projects would more directly study how deeply interconnected ‘obesity’ is with international trade agreements, local environments, and individual lives without sacrificing the sophistication and depth acquired from ethnography in a single site.

Without a greater commitment to collaboration involving multiauthor, multisited ethnographic projects,8 our ability to develop better understandings of the global/local production of obesity and other chronic diseases will likely remain problematically patchy and uneven. This may undermine anthropologists’ ability to comprehensively critique more simplistic understandings of ‘obesity’ and to develop more ethical and effective public policies. Indeed, to ethnographically observe how ‘obesity’ emerges in several different interconnected contexts requires the sophistication and patience of long-term, multiauthor fieldwork. Collaborative and coordinated multiauthored ethnographies will achieve important and unforeseeable insights. Ultimately, they would allow us to document how ‘obesity’ or other chronic diseases are coproduced in global-local transformations brought on by new trade agreements or disease definitions, without sacrificing sensitivity to how exploitative work conditions, food insecurity, biomedicine, and trade agreements locally cluster into relations that necessitate new and potentially toxic social and metabolic transformations.

6 However, we should be mindful that what is ‘global’ or ‘international’ in one case may not be for another. For example, in the case of NAFTA, its three signatory countries were Canada, Mexico, and the United States of America, and therefore a multiauthored ethnography of NAFTA might be more limited in scope than a multiauthored ethnography of a new policy of the World Trade Organization, as it has more than one hundred member countries.

7 Feasibility is also a concern given (amongst other possible complications) the time required to learn new languages.

8 This could also include a commitment to multidisciplinarity through collaborating with historians or economists.
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References
Anderson, Warwick. 1998. ‘Where Is the Postcolonial History of Medicine?’ Bulletin of the History of Medicine 72 (3): 522–30. https://doi.org/10.1353/bhm.1998.0158.
Anderson, Warwick. 2014. ‘Making Global Health History: The Postcolonial Worldliness of Biomedicine’. Social History of Medicine 27 (2): 372–84. https://doi.org/10.1093/shm/hkt126.
Aronowitz, Robert. 2015. Risky Medicine: Our Quest to Cure Fear and Uncertainty. Chicago: University of Chicago Press.
Biehl, João. 2016. ‘Theorizing Global Health’. Medicine Anthropology Theory 3 (2): 127–42. https://doi.org/10.17157/mat.3.2.434.
Camacho, Salvador, and Andreas Ruppel. 2017. ‘Is the Calorie Concept a Real Solution to the Obesity Epidemic?’ Global Health Action 10 (1): 1289650. https://doi.org/10.1080/16549716.2017.1289650.
Darbre, Philippa D. 2017. ‘Endocrine Disruptors and Obesity’. Current Obesity Reports 6 (1): 18–27. https://doi.org/10.1007/s13679-017-0240-4.
Escobar, Arturo. 2011. Encountering Development: The Making and Unmaking of the Third World. Princeton, NJ: Princeton University Press.
Gálvez, Alyshia. 2018. Eating NAFTA: Trade, Food Policies, and the Destruction of Mexico. Oakland, CA: University of California Press.
Garcia, Angela. 2017. ‘The Ambivalent Archive’. In Crumpled Paper Boat: Experiments in Ethnographic Writing, edited by Anand Pandian and Stuart McLean, 29–44. Durham, NC: Duke University Press.

Guthman, Julie. 2011. Weighing in: Obesity, Food Justice, and the Limits of Capitalism. Oakland, CA: University of California Press.

Guthman, Julie. 2012. ‘Opening Up the Black Box of the Body in Geographical Obesity Research: Toward a Critical Political Ecology of Fat’. Annals of the Association of American Geographers 102 (5): 951–57. https://doi.org/10.1080/00045608.2012.659635.

Hargrove, James L. 2007. ‘Does the History of Food Energy Units Suggest a Solution to “Calorie Confusion”?’ Nutrition Journal 6 (December): 44. https://doi.org/10.1186/1475-2891-6-44.

Hatch, Anthony Ryan. 2016. Blood Sugar: Racial Pharmacology and Food Justice in Black America. Minneapolis, MN: University of Minnesota Press.

Krishnaveni, G. V., and C. S. Yajnik. 2017. ‘Developmental Origins of Diabetes – an Indian Perspective’. European Journal of Clinical Nutrition 71 (7): 865–69. https://doi.org/10.1038/ejcn.2017.87.

Livingston, Julie. 2012. Improvising Medicine: An African Oncology Ward in an Emerging Cancer Epidemic. Durham, NC: Duke University Press.

Mansfield, Becky, and Julie Guthman. 2015. ‘Epigenetic Life: Biological Plasticity, Abnormality, and New Configurations of Race and Reproduction’. Cultural Geographies 22 (1): 3–20. https://doi.org/10.1177/1474474014555659.

Mintz, Sidney Wilfred. 1986. Sweetness and Power: The Place of Sugar in Modern History. New York: Penguin.

Misra, Anoop, P. Chowbey, B. M. Makkar, N. K. Vikram, J. S. Wasir, D. Chadha, and Shashank R. Joshi. 2009. ‘Consensus Statement for Diagnosis of Obesity, Abdominal Obesity and the Metabolic Syndrome for Asian Indians and Recommendations for Physical Activity, Medical and Surgical Management’. JAPI 57 (2): 163–70.

Moore, Jason W. 2017. ‘Metabolic Rift or Metabolic Shift? Dialectics, Nature, and the World-Historical Method’. Theory and Society 46 (4): 285–318. https://doi.org/10.1007/s11186-017-9290-6.

Packard, Randall M. 2016. A History of Global Health: Interventions into the Lives of Other Peoples. Baltimore, MD: Johns Hopkins University Press.

Solomon, Harris. 2016. Metabolic Living: Food, Fat, and the Absorption of Illness in India. Durham, NC: Duke University Press.

Stenvinkel, Peter. 2015. ‘Obesity – a Disease with Many Aetiologies Disguised in the Same Oversized Phenotype: Has the Overeating Theory Failed?’ Nephrology Dialysis Transplantation 30 (10): 1656–64. https://doi.org/10.1093/ndt/gfu338.
Street, Alice. 2014. *Biomedicine in an Unstable Place: Infrastructure and Personhood in a Papua New Guinean Hospital*. Durham, NC: Duke University Press.

The Lancet. 2004. ‘Appropriate Body-Mass Index for Asian Populations and Its Implications for Policy and Intervention Strategies’. *The Lancet* 363 (9403): 157–63. [https://doi.org/10.1016/S0140-6736(03)15268-3](https://doi.org/10.1016/S0140-6736(03)15268-3).

The Lancet Diabetes & Endocrinology. 2017. ‘Should We Officially Recognise Obesity as a Disease?’ *The Lancet Diabetes & Endocrinology* 5 (7): 483. [https://doi.org/10.1016/S2213-8587(17)30191-2](https://doi.org/10.1016/S2213-8587(17)30191-2).

Warin, Megan, Tanya Zivkovic, Vivienne Moore, and Paul Ward. 2017. ‘Moral Fiber: Breakfast as a Symbol of ‘a Good Start’ in an Australian Obesity Intervention’. *Medical Anthropology* 36 (3): 217–30. [https://doi.org/10.1080/01459740.2016.1209752](https://doi.org/10.1080/01459740.2016.1209752).

Wells, J. C. K. 2013. ‘Obesity as Malnutrition: The Dimensions beyond Energy Balance’. *European Journal of Clinical Nutrition* 67 (5): 507–12. [https://doi.org/10.1038/ejcn.2013.31](https://doi.org/10.1038/ejcn.2013.31).

Wells, J. C. K., and M. Siervo. 2011. ‘Obesity and Energy Balance: Is the Tail Wagging the Dog?’ *European Journal of Clinical Nutrition* 65 (11): 1173–89. [https://doi.org/10.1038/ejcn.2011.132](https://doi.org/10.1038/ejcn.2011.132).

World Health Organization. 2018. ‘Obesity and Overweight’. WHO fact sheet. 16 February. [http://www.who.int/mediacentre/factsheets/fs311/en/](http://www.who.int/mediacentre/factsheets/fs311/en/).

Yajnik, Chittaranjan. 2018. ‘Confessions of a Thin-Fat Indian’. *European Journal of Clinical Nutrition* 72: 469–73. [https://doi.org/10.1038/s41430-017-0036-3](https://doi.org/10.1038/s41430-017-0036-3).

Yajnik, Chittaranjan S., and John S. Yudkin. 2004. ‘The Y-Y Paradox’. *The Lancet* 363 (9403): 163. [https://doi.org/10.1016/S0140-6736(03)15269-5](https://doi.org/10.1016/S0140-6736(03)15269-5).

Yajnik, C. S., C. H. D. Fall, K. J. Coyaji, S. S. Hirve, S. Rao, D. J. P. Barker, C. Joglekar, and S. Kellingray. 2003. ‘Neonatal Anthropometry: The Thin–Fat Indian Baby. The Pune Maternal Nutrition Study’. *International Journal of Obesity* 27 (2): 173–80. [https://doi.org/10.1038/sj.ijo.802219](https://doi.org/10.1038/sj.ijo.802219).

Yates-Doerr, Emily. 2015. *The Weight of Obesity: Hunger and Global Health in Postwar Guatemala*. Oakland, CA: University of California Press.

Yates-Doerr, Emily, and Megan A. Carney. 2016. ‘Demedicalizing Health: The Kitchen as a Site of Care’. *Medical Anthropology* 35 (4): 305–21. [https://doi.org/10.1080/01459740.2015.1030402](https://doi.org/10.1080/01459740.2015.1030402).