

REVIEW

Telescoping the origins of obesity to women's bodies: How gender inequalities are being squeezed out of Barker's hypothesis

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Aim: This paper traces the genealogy of the Barker hypothesis and its intersections with popular representations of scientific discourses about pregnancy and maternal obesity.

Method: Drawing on Foucault's genealogical method, this study examines the historical 'descent' of the developmental origins of adult disease and its initial grounding in structural factors of gender inequality and low socioeconomic status.

Results: In the more recent reproductive medicine literature, Barker's hypothesis has been used to understand the causes and consequences of foetal over-nutrition and has shifted its focus from social determinants to individual, gendered bodies. The print media has gainfully employed this conceptualization of obesity and, in doing so, placed women, and mothers in particular, as causal agents in the reproduction of obesity across generations. Such a 'common sense' understanding of obesity production and reproduction means that both the scientific literature and the public understanding of science has inadvertently assisted in putting women forward as the transmitters of obesity across generations.

Conclusions: This powerful telescoping of the origins of obesity to women's bodies and their appetites is in stark contrast to earlier foci on gender inequalities and changing women's circumstances.

Keywords: Maternal obesity, the Barker hypothesis, foetal origins of adult chronic disease, Foucault's genealogy, gender inequality

INTRODUCTION

The French philosopher Michel Foucault used the concept of genealogy to connect histories of knowing across different times. Tracing the history of knowledge involves the history of power and struggles over how knowledge is constructed and what types of knowledge are valued, excluded or

ignored. In *Nietzsche, Genealogy and History*, Foucault (1971) argued that every concept has a genealogy or history. Genealogy is, however, not a linear unfolding of historical events but an investigation of the subtle intersections that form a network of knowledge. Tracing the descent of genealogy does not search for origins or truths, but follows the complex course of descent by 'identifying the accidents, the minute deviation... the errors, the false appraisals and the faulty calculations that give birth to those things that continue to exist and have value for us' (p. 81). The goal is not to trace the evolution of a concept, but to 'isolate the different scenes where they engage in different roles' (p. 76).

Intimately connected with this approach is the ways in which genealogy, as an analysis of descent, attaches itself to 'the body' (Foucault 1971: 82). Bodies and histories are 'situated within the articulation of body and history; in the nervous system, in temperament, in the digestive apparatus;... in improper diets; in the debilitated and prostrate bodies of those whose ancestors committed errors' (p. 82). The task of a genealogist is to shorten [her] vision to those things nearest and expose a body totally imprinted by history (p. 83). As McPhail (2009: 1043) argues, 'obesity is not biologically self-evident, but an historically articulated, worked-through and worked-on category infused by power, politics and positioning'.

This paper takes Foucault's concept of genealogy to unravel the 'origins' (in a Foucauldian sense) of the foetal origins of adult chronic disease. 'Origins' thus has two distinct meanings in this paper; as an analytical frame to interrogate the history of ideas that accompany discourses of maternal obesity; and secondly, the taken-for-granted meaning of origin in the medical science literature on obesity. The first section examines the scientific literature on Barker's hypothesis in relation to the developmental origins

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of adult disease. Barker's early work focused on the gendered socio-economic effects of maternal under-nutrition in pregnancy and this has more recently extended to maternal obesity and foetal over-nutrition. This shift from under-nutrition during historical periods of acute economic hardship to over-nutrition during widespread economic transition to affluence has occurred in the context of rising concerns about the 'obesity epidemic' and the resultant economic, social and public health costs of chronic disease. We argue, however, that this shift to over-nutrition has led to an emphasis on the individual bodies of women—and in particular the interiority of mothers' bodies, their individual choices and mothering roles, as the point of origin for obesity. There has been an associated shift away from the embodiment of social inequalities in the starved population of women to the individual woman with a body formed by consumption. It is the moral judgements and imperatives attached to such thinking that we problematize here.

We present findings from a recent discourse analysis of obesity in the Australian press to demonstrate how Barker's hypothesis has influenced a 'common sense' understanding of maternal obesity as dangerous and corrupting the foetus and newborn child. The anthropologist Clifford Geertz argues that 'common sense' is a cultural system, in that it is historically constructed and 'can be questioned, disputed, affirmed, developed, formalized, contemplated, even taught, and can vary dramatically from one people to the next' (Geertz 1983: 73–93). For Geertz, 'common sense' is a stock of shared knowledge that is assembled over time and appears as unquestionable 'truths' because common sense 'lies so artlessly before our eyes, it is almost impossible to see' (Geertz 1983: 92).

A common sense understanding of maternal obesity did not appear without a history that is both interpretative and judgemental. Women's reproductive bodies are entangled in theories of how pregnancy, appetites and harm to the foetus are intimately connected (cf Kukla 2005). When coupled with a political shift to individual responsibility and moralizing discourses on obesity, maternal obesity becomes a potent signifier for blame and public scrutiny. The dangers associated with women's reproductive bodies have a powerful hold in the public imagination and it is these vestiges that resurface in recent discourses about maternal over-nutrition. The concluding section of the paper reflects on why responses to the intra-uterine environments of under- and over-nutrition might be so different.

BARKER AND THE ORIGINS OF ADULT CHRONIC DISEASE

From the late 1980s, research conducted by Professor David Barker and his colleagues advanced the theory that chronic disease originated, at least in part, in early life. The central argument was that organs and systems of the body were especially vulnerable during critical pre-natal formative periods and their development could be perturbed by adverse intrauterine conditions, with permanent consequences for the baby's physiology and metabolism.

This process, known as 'programming', made an individual susceptible to chronic disease in later life, most notably cardiovascular disease and diabetes mellitus. Of particular relevance to the Barker hypothesis was the focus on poor maternal nutrition in pregnancy.

The theory became the focus of a major international research effort. Critics initially referred to the theory as 'the Barker Hypothesis', an expression that Barker rejected in favour of 'the foetal origins hypothesis'. It was soon recognized that the period of developmental vulnerability extended into infancy and perhaps beyond, so the terms 'foetal and infant origins' or 'early origins' or 'developmental origins' were preferred. It has also become appreciated that maternal nutrition status commences prior to pregnancy and is reflected in the maternal body fat and other nutritive stores laid down prior to conception.

The paper that heralded the theory was by Barker and Osmond (1986). The paper drew attention to the similarity in the pattern of infant mortality rates in 212 regions of England and Wales in 1921–1925 and the pattern of death rates for ischaemic heart disease in those regions in 1968–1978. Epidemiologists refer to this type of study as a geographical or ecological study. The findings are understood to be open to many interpretations, but are considered to provide a good starting point for hypothesis generation and more definitive research. Barker and Osmond (1986: 1080) speculated that '... adverse influences in childhood, associated with poor living standards, increase susceptibility to other influences, associated with affluence, encountered in later life'. Causes of ill-health were suggested to be poor living standards, invoking structural constraints on health.

The following year, Barker and Osmond (1987) presented a comparison of three neighbouring towns in the north of England that had different mortality profiles, so that the likelihood of death from chronic disease at ages 55–74 years was quite different in each town. Barker and Osmond argued that these differences were related to life-long differences in the living conditions of the inhabitants.

The present similarities of the towns belies the large differences that formerly existed and led to large differences in mortality among infants and young children. These differences included the health and physique of mothers, infant feeding practices, housing, and sanitation... (Barker and Osmond 1987: 752).

Here, the root causes are suggested to be structural, related to life-course (history of growth) and to maternal behaviour (breast feeding), although that was seen to be economically governed (since women needed to return to work in the cotton weaving mills).

The first paper by Barker and his colleagues that presented data concerning individuals (not regions or towns) and linked early growth to cause of death was published in 1989 (Barker et al. 1989). That research was based on the archived records of midwives (who attended home births) and 'health visitors' (who went to the home periodically over the next year) in Hertfordshire in 1911–1930. The records were used to assemble a cohort of

men who would have been 58–77 years old if they were living when the research was conducted. Deaths that had occurred among the men were ascertained from the UK National Health Service Central Register. Low birth weight and, more strongly, low weight at 1 year, was associated with increased risk of death from ischaemic heart disease. In the discussion, Barker and his colleagues point to the growth of the mother, particularly in her early childhood, as an underlying influence on birth weight.

In one of the first of many papers in which Barker summarized and interpreted the findings available thus far, he concluded:

A new model of adult degenerative disease is developing. The old one was based on the interaction between genes and an adverse environment in adult life. The new one will include the programming of structure and function by the environment in foetal and infant life. It may lead to a new national strategy for reducing inequalities of health in Britain . . . The new one will need to address differences in the growth of babies and in the nutrition and health of their mothers . . . The seeds of inequalities in health in the next century are being sown today—in inner cities and other communities where adverse influences impair the growth, nutrition and health of mothers and their infants (Barker 1991: 67).

Again, attention is drawn to the geographical and socio-economic constraints on the health of women and their children. Central themes of the theory at this point are that the challenges to the mother are pervasive, enduring and socially located; and that maternal hardship constitutes an insult to the developing foetus with complex and profound diminution of basic physiological functioning that takes decades to deteriorate to the point of clinical disease.

Throughout the 1990s, evidence of associations (statistical connections) between low birth weight and increased risk of chronic disease in adulthood accumulated. Cardiovascular disease and type 2 diabetes were the most prominent outcomes. The high levels of scepticism with which the 'early origins' theory had initially been regarded began to abate with the publication of supportive findings from several very large cohort studies (Rich-Edwards et al. 1997; Leon et al. 1998). Sporadic jousting about the strength of the evidence continued (e.g. Kramer and Joseph 1996; Susser and Levin 1999). However, by the end of the decade several consensus statements in support of the theory had been issued (e.g. by the International Dietary Energy Consultancy Group, Grivetti et al. 1998) and the Royal College of Obstetricians and Gynaecologists (UK) had convened a Study Group and published the collected papers (O'Brien et al. 1999).

Perhaps most significant was the UK Report of the Independent Inquiry into Inequalities in Health (1998, known as the Acheson Report) which endorsed a link between low birth weight and chronic disease, clearly locating low birth weight in deprivation over the life of the mother. The Report made various recommendations to improve health and nutrition and reduce poverty (especially

food poverty) in children and women of childbearing age. So influential was Barker's hypothesis that New Labour instrumented a raft of policies and national strategies (National Childcare Strategy, the Sure Start programme and the Working Families Tax Credit) that aimed to support parents (e.g. mothers) and children and childcare services.

This emphasis on mothers, their reproductive bodies and child welfare has a long history in UK policy. Wainright (2003), for example, details how a specific relationship between infant deaths and working mothers in Victorian and Edwardian Dundee led to programmes of reform to reduce infant mortality which were attached to certain 'classed' meanings of 'mother' and 'motherhood'. The Acheson Report and the raft of New Labour policies in a neoliberal political climate extend this history of reform, but instead of deep structural change, it is mothers and their *in utero* environments (with the biological environment now taking priority) that are constructed as a risk to the foetus. Women's reproductive capacities embody the future (cf Evans 2010) and it is the risk to future generations that must be managed in the present.

This legitimization of the foetal origins of disease was a critical turning point in what Foucault would call the 'conditions of possibility'. It was now possible to speak about foetal origins as an entity and such a discourse was legitimated through the power and knowledge of medical institutions (including the scientific community) and government frameworks and public policy. However, Foucault's genealogical method is not simply concerned with 'discoveries' or technologies of power, but with the operations of power at its destination, that is the ways in which ideas about foetal origins of disease flow out into the capillary extremities and enter a machinery of production in which certain objects and people become entangled in a new web of scrutiny.

Thus, the focus on low birth weight and the undernourished baby was accompanied by considerable interest in the gendered socio-economic context in which women became mothers. Arguably, this was facilitated by medical understanding that nutrition of the foetus does not simply correspond to maternal nutrition, but reflects 'a long and sometimes precarious 'supply line'' (Harding 2001: 16) that involves many factors, including the adequacy of the blood flow to the uterus and of placental function. A small but long-standing literature pointed to locational, historical and even intergenerational influences on a woman's biological capacity in relation to the supply line (Harding 2001). This seems to have resonated with a larger literature on women's struggles for material necessities, including food.

Possible pre-natal origins of obesity were not a clear theme in the early origins research during the 1990s. A key publication on the topic, by Whitaker and Dietz (1998), contained cautious speculation and many caveats about gaps in knowledge and emphasized a role for maternal gestational diabetes rather than maternal weight. Research in a similar vein had been occurring for quite some time (e.g. Kramer et al. 1985; Aerts et al. 1990), but generally not under the 'early origins' banner. By the early 2000s, however, the idea

was part of the child obesity lexicon. Ebbeling et al. (2002), in a landmark paper on the childhood obesity ‘crisis’ reported an

intriguing hypothesis that prenatal overnutrition might affect lifelong risk of obesity. According to this hypothesis, maternal obesity increases transfer of nutrients across the placenta, inducing permanent changes in appetite, neuroendocrine functioning, or energy metabolism... The implications of these findings are formidable: the obesity epidemic could accelerate through successive generations *independent* of further genetic or environmental factors (p. 475) [our emphasis].

The point in tracing the development of Barker’s ideas through a genealogical method is to identify the ways in which the field of early origins has become constituted as a reality, expanded and ‘praised as representing a paradigmatic shift in medicine’ (Vagero and Illsley 1995: 219). Yet, while this field has been growing, there has been a simultaneous telescoping of foci away from wider social determinants of health to the individual bodies of women. The medical gaze that Foucault (1973) wrote about in *The Birth of the Clinic* is narrowed to women and their potentiality to become mothers; and this gaze is now penetrating deep into female bodies. The uterus has become ‘the environment’ of scrutiny; and the social environment is an independent and secondary context.

An extreme example of this narrowing is offered by Kral (2004), a US surgeon who argues that all women, even ‘newborn girls’ (p. 1544), have the potential to become ‘doubly damaging’ because of their childbearing capacity. As fat is passed on through the female body the only way to curb the obesity epidemic is to target the female sex: ‘[f]rom birth to menarche, behaviour modification in mothers and children should be the first choice’ in obesity prevention (Kral 2004: 1544). In Kral’s stepped care strategy, this intervention should then progress to drug treatment and, in selected cases, laparoscopic surgery for young women of childbearing age. The current thinking that the obesity epidemic could accelerate through successive generations *independent of wider factors* is a significant shift from Barker’s early work. Obesity is no longer thought of as an embodiment of social inequalities (such as gender and social class) but a biomedical disease that has its origins in the interiority of individual women’s bodies. This shift or telescoping, we argue, has gone unnoticed since the 1990s.

THE POPULARIZATION OF THE OVER-NUTRITION HYPOTHESIS

While Kral’s statements might be regarded as representing one end of a spectrum on how to ‘curb the obesity epidemic’, the over-nutrition hypothesis and the central role of women in reproducing obesity has not only entered, but is now embedded in, public discourse. This, to follow the Foucauldian genealogical method, is the productive force of capillary power. Women and their bodies are entangled in

a machinery of production in which the media is the main conduit of scientific ‘discoveries’.

In relation to public understandings of obesity, a number of commentators (Rich and Evans 2005; Boero 2007; Saguy and Almeling 2008) have already noted that ‘the media is simultaneously a crucial source of scientific and public health information, and a key contributor to the shaping and definition of public health issues as social problems’ (Maher et al. 2010: 236). Monaghan et al. (2010) refer to the media as ‘amplifiers/moralizers’ in terms of sensationalizing, stereotyping and repeatedly focusing on ‘dramatic’ or ‘moralizing’ aspects of obesity. The remainder of this paper examines how maternal over-nutrition is ‘amplified’ in media discussions of obesity, showing how, across the life course, women are portrayed as entirely responsible for passing obesity to their children and across generations as a matter of their biology and ill-informed ‘lifestyle choices’.

In 2009 we undertook a discourse analysis of obesity reporting in three metropolitan Australian newspapers—*The Advertiser*, *The Australian* and *The Sydney Morning Herald*—between 1 January 2009 and 31 March 2009. In order to collect data on visual images we opted against using text-based databases such as Factiva or LexisNexis and manually searched microfilm of the newspapers in our sample. We sourced 181 articles related to obesity, made copies of each and conducted a thematic analysis of text and visual images (see Zivkovic et al. 2010 for a comprehensive methodological description). When obesity was constructed in terms of parental responsibility, the onus was on the parent (and *always* the mother) to help their child lose weight for the specific purpose of reducing overweight-associated health problems.

Obesity was repeatedly presented as ‘a problem’ and ‘a danger’. In March 2009 an article in the daily State newspaper of South Australia (*The Advertiser*) featured a large photograph of a smiling mother and her newborn baby, warning that ‘health problems are passed on through generations’. The headline to the story—‘Mothers’ smoking gun’—referred to a cohort study conducted by researchers at the University of Adelaide (including two authors of this paper) that implied that overweight pregnant women are more likely to have children, even grandchildren, who are overweight. A similar news item in Australia’s national circulation paper (*The Australian*) in the previous month claimed that ‘obese women are more likely to have children with a range of birth defects’ (Taor 2009a). The warnings are clear—obesity in pregnancy is potentially damaging and it is mothers who are responsible for their children’s ill health.

The media reporting on scientific studies, however, not only constructs fat mothers as putting their newborns and future generations at risk, but also positions them as a danger to themselves. Obese pregnant mothers are ‘at risk of diabetes, high blood pressure and delivery by caesarean section, while cancer and heart disease could also develop’ (Harper-Erini 2008). Hospitals are having to adjust to ‘increasing numbers of obese mothers’ by ‘spending more than \$100 million on equipment to deal with overweight

patients, including \$45 million on stronger beds' (*The Advertiser*, January 2009). Obesity in pregnancy is constructed as dangerous and as Keenan and Stapleton (2010: 370) suggest, 'the medicalization and moralization of large bodies in pregnancy creates women as subjects "at risk" to themselves and their foetus/infant'.

The emphasis, however, is very much on the culpability of mothers. In a context where foetus' have personhood and rights (cf Petchesky 1987; Ruddick 2007), mothers can be 'constructed as antagonistic towards their foetus, who becomes an object of collective concern, with its own public identity as the "potential [healthy] citizen"' (Longhurst 1999, cited in Fox et al. 2009: 62). Foetuses and children are victims in need of protection from irresponsible parents and in some cases mothers have been prosecuted for neglect and abuse of their obese children (Zivkovic et al. 2010).

Media headlines amplify a failure of duty of care in terms of women's biological and social roles as mothers. Childhood obesity, it is claimed 'might start in the womb' (Brown 2009) and lies in the 'improper' nutrients supplied to foetuses by their mothers. In response to scientific reports in early 2009, Australian broadsheets had stories with headlines such as: 'Obese mums-to-be urged to diet' (Hall and Davis 2009), 'Weighty problems born of bad diet in pregnancy' (Brown 2009), 'Overweight mums putting newborns at greater risks' (Shepherd 2009), 'Breastfed children least likely to be abused by mothers' (Taor 2009b) and 'Child neglect linked to [breast] feeding' (Medew 2009). In these stories the proximity of the maternal–foetal/maternal–child relationship is central to childhood obesity—from pre-pregnancy, pregnant bodies as foetal containers and good 'breastfeeding' mothers as neoliberalism's 'healthy citizen'. If women do not accept their responsibilities as caregivers (both biologically and through social roles) they place the unborn baby, their children and future generations at risk.

MATERNAL APPETITES

The ways in which women's bodies and behaviours are situated as the cause for 'the obesity epidemic' links in with a long history of association between pregnancy and danger (cf Warin et al. 2011). In her study of 17th and 18th century midwifery texts (and Rousseau's political writings as well as contemporary guides to pregnancy and breastfeeding), Kukla (2005: 14) outlines the logic of maternal imagination, in which 'frights, craving and sexual arousals . . . were by far the most [common] cited passions attributed with marking the foetus' (cf Roodenburg 1988). Cravings for shellfish, for example, might cause grotesque facial deformities and cravings for strawberries or other fruits caused birthmarks resembling those fruits. It is, in particular, the passions bound up with maternal appetites that posed the greatest threat to the assumed permeability of pregnant bodies. A pregnant woman's appetite (including the ingestion of foods, drinks and other sensory experiences such as fear and lust) was the explicit mechanism that transferred the effect from maternal environment to foetus (Kukla 2005: 14).

Wombs were considered to have appetites too and were constructed as having the potentiality for dual power as they could both form and deform; create and nurture the human form, yet also create monsters (Kukla 2005). Hippocrates had earlier claimed that the 'womb is the origin of all diseases in women' (Hippocrates, cited in Kukla 2005: 5) and this agency was attributed to emotional disturbances like hysteria. In the 18th century, pregnancy was thus a dangerous process, for women's appetites increased, which in turn increased the possibility of harmful passions and appetites corrupting the foetus.

While knowledge and practices surrounding pregnancy have significantly changed through time, Kukla suggests that pre-occupations with the pregnant bodies and potentiality to harm the foetus still govern our imagination. Maternal obesity is presented in the news media as harming foetuses and it is the uncontrollable appetites of mothers to be that are blamed for the obesity epidemic.

THE SOCIAL ACCEPTABILITY OF OBESITY STIGMA

Inability to control appetite is morally loaded and underpins why under-nutrition and over-nutrition are responded to and constructed so differently in both scientific and public understandings of maternal obesity. Maternal and foetal under-nutrition is understood to be the result of interconnections between deprivation and gender inequalities (cf Osmani and Sen 2003) and located in wider environments of cultural and economic transitions. Hampshire et al. (2009), for example, describe how childhood under-nutrition is not straightforward and can be driven by an array of social, political, cultural and economic processes. Over-nutrition, however, does not carry the same broader implications, compassion or understanding of life-long disadvantage.

This difference between understandings of over- and under-nutrition are related to a powerful moral discourse associated with fatness that draws upon historical traces of appetite and danger. Evans (2006: 262) argues that 'weight is a proxy for ill-health, whereby there are two mutually exclusive possibilities: fat, unfit and unhealthy or thin, fit and healthy'. Yet, in the foetal origins field, underweight and overweight mothers and babies can both be considered 'at risk' and not healthy. Under-nutrition, however, is seen as outside of the mother's control, whereas over-nutrition is viewed as an individual 'choice' and morally culpable. The historical construction of obesity as a deviant or discredited attribute means that maternal over-nutrition is understood through a different cultural lens.

As an embodied state, obesity is constructed as gluttonous, slothful, lazy and lacking in willpower (even in UK policy documents; Evans 2006). An analysis of obesity in Swedish newspapers found that overweight people were presented as 'stupid', 'ugly', 'naïve', 'irresponsible', 'lazy', 'greedy', 'without manners' and 'repugnant' (Sandberg 2007). Weight was also presented as a female problem and overweight women were described as 'too big and sloppy', 'sweating' and 'disgusting'. Fatness provokes disgust,

contravenes the standards of ideal beauty and is at the core of our dietary restrictions and understandings of bodily purity (Murray 2005). Women who are pregnant and obese thus carry a double moral failure, for they have not only contravened societal standards of bodily control, but passed fat onto their foetus and future generations.

A systematic review of the stigma of obesity published in 2009 (Puhl and Heuer 2009) found that the news media often frames obesity in terms of personal responsibility (Lawrence 2004; Bonfiglioli et al. 2007; Kim and Willis 2007), focusing on individual causes of obesity (such as a poor diet) and individual-level solutions (e.g. changing one's diet). This focus on personal responsibility enables mothers-to-be blamed for the misfeeding of their children, even *in utero*.

Shallow and limited press coverage of obesity and mother blame is not confined to the sample we used in Australia. Similar narrow discourses can be found in UK and US (Saguy and Almeling 2008) print media. Keenan and Stapleton's (2010) study of media reporting in the UK found very similar results of regular reporting of the perils of obese mothers. Headlines focused on the scale of the problem ('Alarming surge in the number of obese women having babies', Fletcher 2007) and the rising number of obese infants ('The big millennium babies: Quarter are overweight by three', Elliot 2007). Keenan and Stapleton also found that most articles emphasized the dangers obese women posed to their infant's health across the life course with 'obese mum's a risk to "tot"' (Obese Mum 2007) and 'Obese mums harm baby's fertility' (Roberts 2005).

In a neoliberal environment, being fat and having fat kids 'is framed as the product of unhealthy choices' (Saguy and Almeling 2008: 57) and plump babies are now a literal embodiment of the wrong choices and failed mothering. 'Poor' and 'bad' choices can be made at any stage across the life course and rather than distinguish between reproduction and care giving, this frame makes women seamlessly responsible for the future health of children. Women are held responsible for the obesity of their children if they do not prepare their bodies for pregnancy, do not maintain their bodies during pregnancy, do not breast feed, do not put the right choices in lunchboxes or make nutritious, home cooked meals (Malik 2007: 45–46; Fox et al. 2009). Levels of responsibility attributed to mothers in relation to obesity thus travel from the 'placenta to breast, from breast to lunchbox, from lunchbox to the dinner table' (Malik 2007: 46).

CONCLUSION: THE TELESCOPING EFFECT OF ORIGINS

Historical constructions of pregnant bodies and public understandings of the foetal origins hypothesis have informed common sense approaches of causality in which mothers are bio-culturally constructed as the origin and source of obesity. Drawing upon Foucault's genealogical approach, we have traced how the development origins of health and disease has transformed from a concept that highlighted the interaction between a country's socio-

economic status and the disproportionate exposure of women to obesogenic factors (Wells 2010) to a narrow understanding that locates the reproduction of obesity in the interiors of women's bodies. The societal environment has telescoped into a bodily environment of the womb.

This is not to suggest that there is no attention given to the relationship between obesity and wider societal factors; the evidence, for example, points to women from lower socio-economic circumstances being the most obese (Wardle et al. 2002). Poor women are thus statistically more likely to be obese and obese women are more likely to be impoverished as a result of their body size (Viner and Cole 2005). Wells (2010) argues that higher obesity prevalence in women arises for different reasons in different global regions, relating variously to issues such as gender bias in children's nutrition, foetal exposure to maternal work during pregnancy or even constraints on adult behaviour for religious reasons. Education, social independence and economic independence (as well as the role of fathers in intergenerational obesity; Cole et al. 2008), are all important factors in understanding obesity. Yet, despite this evidence, the explanations for maternal obesity, and obesity more generally, ignore these circumstances and blame poor people for their bad eating habits and poor health.

Foucault was sceptical of searching for origins. He argued that a conventional historian might look for origins from which to construct tidy and linear narratives, but he was more interested in the things that were overlooked (ignored) in order to fit a coherent story. Feminist scholars like Fausto-Sterling (1992; 2000) and Fine (2010), who investigate scientific claims about sex, gender and bodies, similarly note that 'when we follow the trail of contemporary science we discover a surprising number of gaps, assumptions, inconsistencies, poor methodologies and leaps of faith—as well as a more than one echo of the insalubrious past' (Fine 2010: xxvii). Our attention has also been to the echoes and subtle shifts that have been overlooked in the foetal origins hypothesis and its public consumption. These shifts illuminate a number of historical and contemporary moral and political discourses, which intersect to entangle women, their appetites and their reproductive bodies into simple 'sound bite' explanations. The health of future generations is compressed into an understanding of women's reproductive bodies as both the origin and future of obesity, in which space and time is collapsed into a material presence of maternal risk. Representations of the foetal origins hypothesis and obesity in the news media should call upon a 'profusion of entangled events' (Foucault 1971: 89), not an 'ultimately simple configuration' (p. 89) where events possess 'essential traits', 'final meaning' or 'final value' (p. 89).

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