Chapter 3

The First 5000 Days

Making DOHaD, 1989–2003

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3.1 Introduction

In June 2003, the Second World Congress on Fetal Origins of Adult Disease took place in Brighton, the UK. Alongside researchers specialising in fetal development – developmental physiologists, epidemiologists, obstetricians, and paediatricians – the meeting was addressed by a group of illustrious guests: the well-known scientists and science communicators Colin Blakemore and Lord Robert Winston; the Nobel laureate in economics Amartya Sen; and the royal patron, Princess Anne. The latter stressed the importance of this research for global health and presented a silver salver to the Southampton epidemiologist David Barker, in recognition of his pioneering work in the field [1]. At this meeting, the International Society for the Developmental Origins of Health and Disease (DOHaD) was founded, and the global ambitions of the new field were evident in its logo showing a fetus ostensibly peacefully nestled in the globe.

Yet just a decade earlier, this field had not existed. It began at a workshop at Lerici, near La Spezia in Italy, in 1989, in which David Barker presented his retrospective epidemiological research from Hertfordshire, UK, showing that low birthweight was associated with an increased risk of chronic non-communicable diseases in later life. It was at that meeting when fetal physiologists first discussed the plausibility and possible underlying mechanisms of Barker's observations [2].

This chapter, written by a founder of the field and a historian with long-term interest in DOHaD, examines this key (long) decade in the making of DOHaD, bookended by the 1989 La Spezia workshop and the 2003 Brighton Congress. It argues that, for all the attention that DOHaD has received from social and biomedical scientists, its history has not been studied in sufficient depth. Yet to understand the objectives, methods, research questions, and intellectual networks making the field of DOHaD, and the responses that it evoked and that further shaped it, we must appreciate the historical and geographical context in which it was created. For the purposes of this chapter, we focus on three key themes:

1. **Interdisciplinarity.** From its inception, DOHaD was explicitly interdisciplinary, and interdisciplinarity is a source of its intellectual dynamism and breadth. Yet this required

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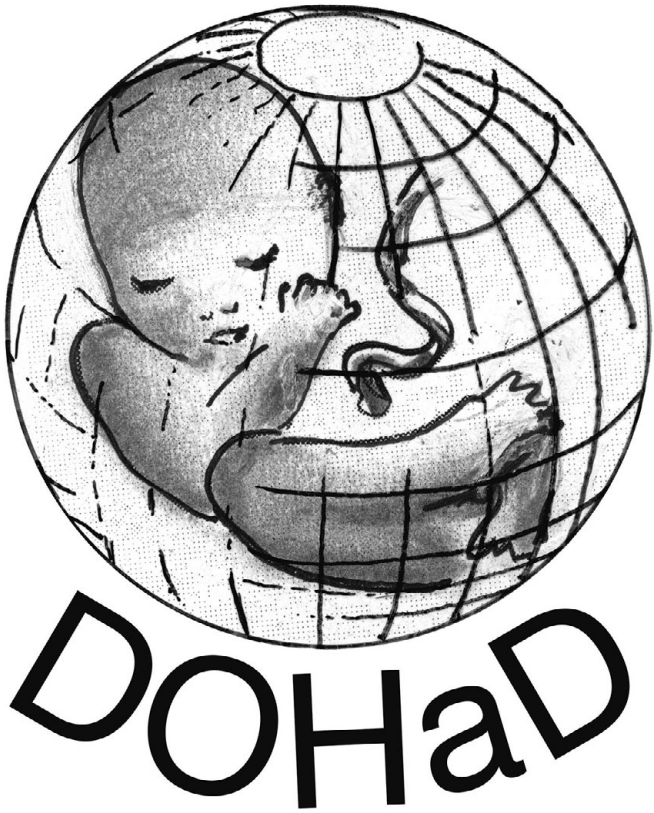

Figure 3.1 Original logo of the International DOHaD Society, created by Mark Hanson.

rendering the concepts of each collaborating discipline intelligible to all participating members [3]. As we will show, while these transformations were productive, in the process some of the context and layers of the original question were lost.

2. **Social class and health inequalities in Britain:** Barker brought to his research a concern with social inequalities in health. We briefly review its long-standing history in British science and policy and then focus on the reasons for the uptake of DOHaD by the Labour Party upon its accession to power in 1997.
3. **Globalisation and health.** DOHaD's international expansion took place during a decade of globalisation. The global interest in DOHaD has been taken for granted, but, as we show in this section, the international networks through which the field spread merit deeper investigation.

3.2 The Promises and Challenges of Interdisciplinarity

In 1989, the doyen of fetal physiology, Geoffrey Dawes, invited the epidemiologist David Barker to a meeting in Villa Marigola, a conference centre near La Spezia, Italy. The title of the conference, 'Fetal Autonomy and Adaptation', signalled both continuity and change. Twenty years earlier, Dawes chaired a meeting centred on the key idea that the fetus 'demonstrates its innate capacity for influencing its external and maintaining its internal environment – that is, its autonomy' [4]. The La Spezia meeting was intended to

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mark a new era in fetal physiology in which the preoccupation with the autonomous functions of the fetus would be complemented, if not replaced, with a focus on the interaction between the fetus and its broader environment [2, 5].

To inspire new thinking and draw on the views of the physiologists' collective, Dawes invited David Barker, an environmental epidemiologist from the University of Southampton. Barker had recently published a series of well-received but provocative articles. He argued that chronic non-communicable diseases were not caused (exclusively) by adult lifestyles but by the conditions of early life that set the organism on a path that increased, or reduced, the later disease risk [6, 7].

Barker built his hypothesis by linking historical with contemporary demographic and epidemiological data. His first studies took an 'ecological' approach, by demonstrating a geographical correlation between high infant mortality in the early twentieth century and high morbidity from cardiovascular disease (in men) in the period 1968–1978. The causal link, he proposed, was poor early-life nutrition, caused by maternal malnutrition and poor health, infectious diseases in infancy, and artificial feeding practices. Ecological studies were followed by a retrospective cohort study on a group of men born in Hertfordshire around 1920, whose records of birth and infant weight were, unusually, preserved. Their matched mortality records showed that those born small, and especially those whose growth failed to 'catch up' in the first year of life, had a higher relative risk of death from cardiovascular disease [7].

In the conference proceedings, printouts of presentations were followed by summaries of the discussions after each paper. These records show us physiologists at the La Spezia conference were intrigued by Barker's findings but struggled to imagine how to convert them into a workable experimental programme. The discussants asked about placental size and gestational age at birth, and also about the possible effects of genetic factors, smoking, and breastfeeding. Significantly, in view of later developments in DOHaD, Hanson asked Barker whether correcting for social class might remove the association between birthweight and later disease, in view of the well-known association with cardiovascular disease, which we will discuss in the next section. This correction might distinguish between an underlying mechanism and merely an association. Barker replied that 'Hertfordshire *at the time in question* [emphasis original] was a rural county in which social class was relatively unrelated to health' [2, p. 35]. While Barker noted that future data on social class and early life would become available, the fetal physiologists left with the resolve to devise studies in animal models to investigate possible fundamental mechanisms.

Hanson's group provides an example of such early physiological, animal DOHaD work. When they moved to University College London in 1990, they secured funding to investigate the effect of small reductions in the food intake of ewes during early pregnancy. These reductions were not large enough to produce a sustained reduction in maternal body weight or lambs' birthweight but did produce effects on fetal and neonatal cardiovascular and endocrine function. This experiment, they argued, distinguished between a physiological ('normal') adaptive process, albeit with possible later health consequences, and a pathophysiological process in utero. The physiological proposition was indeed confirmed, although the misconception that developmental processes 'harm' the fetus has been persistent [8, 9]. Barker and researchers investigating the effects of moderate to severe challenges such as the Dutch Hunger Winter imagined the environment of early human development as a complex web of social and economic forces ultimately manifested as the food available to women and girls. Physiologists, in contrast, had in mind specific graded and quantifiable changes in physiological

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parameters such as blood pressure, oxygen level, or concentration of nutrients that could be registered by receptors and which then altered development through plasticity [10].

Experimentalists initially turned to the animals – namely, sheep – that had been traditionally used to model human pregnancy. Indeed, their confidence in this animal model was validated when ultrasound, a technology introduced in the 1960s, confirmed similarities between ovine and human fetal development [8, 11]. But studies testing the effects of specific nutritional modification on developing offspring required larger numbers of animals than pregnancy research. Sheep were expensive, slow to reproduce, and difficult to manipulate nutritionally for such studies. At the same time, animal experimental regulations were becoming much more stringent. DOHaD scientists replaced sheep with smaller animal 'models' – rats, mice, and guinea pigs – that had the advantage of rapid reproductive cycles, lower cost, and simpler regulatory approval. Yet with their large litters and fast development, much of which occurred after birth, they had far less in common with human fetal development. While the replacement of the sheep with small animals was a pragmatic decision, the transferability of observations from small experimental animals to humans was uncertain.

These regulatory and economic pressures on experimental physiology were happening simultaneously with the rise of genomics, which culminated with the publication of the human genome at the end of our examined period (2000, officially in 2003) [12, 13]. This co-occurrence was not coincidental but the result of the economic and policy shift in the UK through the 1970s and 1980s. The political pressures to cut costs and modernise science were translated into support for certain scientific fields, while other fields lost funding, institutional footing, and political advocacy. In agriculture, traditional animal genetics that relied on long-term follow-up of generations of farm animals was defunded in favour of genomic biotechnology [14, 15]. Fetal physiology, also using large farm animals, saw funding cuts too. Through the 1980s and 1990s, the spotlight on novel animal 'disease models' developed in genomics laboratories – animal strains genetically modified to carry mutant genes predisposing them to specific diseases – and the first successful experiment in cloning a mammalian organism further increased public concern about animal rights [16].

DOHaD, with its focus on the environment–organism relationship, had no interest in genomics at first; however, the overarching push away from experimental physiology and towards genomics was likely the key reason for DOHaD's entrance into epigenetics in the early 2000s [17]. This disciplinary relationship was mutually beneficial: while epigenetics provided molecular evidence to DOHaD, DOHaD secured policy relevance for epigenetics [18]. This disciplinary relationship between DOHaD and (environmental) epigenetics is so close that many see it as the same field [9]. Yet it is important to note that each field began on its own, roughly simultaneously in the late 1980s, and had over 15 years of independent development [19]. Many scientists who have used epigenetics to explain intergenerational transmission of disease, or, more broadly, inheritance of phenotypic traits, do not see themselves as members of the DOHaD community. Similarly, for many in the DOHaD community, epigenetics is not a core element of the field but rather one of the tools to address the question of 'developmental origins'. As this chapter stops in 2003, the DOHaD–epigenetics relationship is beyond its scope.

A field of particular interest to the emerging DOHaD community was human medicine. Here we must distinguish between epidemiologists and nutrition scientists working inside medical institutions and research groups, who had been interested in

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'Barker's hypothesis' throughout, from practicing clinicians. In particular, specialists in internal medicine – cardiologists, diabetologists, and endocrinologists – who treated adults, and, increasingly, elderly patients, and whose primary objective was treatment rather than early prevention of chronic disease showed little interest in DOHaD [20, p. 47]. In terms of elucidating mechanisms of cardiovascular and metabolic disease, they placed greater trust in genomics, which promised to reveal the basis of risk of disease at the individual level; a promise later captured in the term 'personalised medicine' [21]. In contrast, obstetricians and paediatricians, communities that had already collaborated closely with experimental physiologists in the fields of fetal and neonatal physiology, joined DOHaD in larger numbers. So, while the field was meant to bridge two opposite ends of the human lifespan, in practice, clinical disciplines studying life's beginning took up more space in the field than those at the other end, and this influenced DOHaD's direction. Probably the most significant criticisms came from Barker's own discipline. Epidemiologists argued that his observations were artefacts arising from over-controlling for variables such as BMI and other confounders. They lacked confirmation in studies of cohorts where birthweight was smaller such as twins. The potentially underestimated importance of social factors was also emphasised [22, 23].

The retrospective nature of the early studies made it difficult if not impossible to resolve such issues, and epidemiologists began to use case-control and cohort studies to clarify the causal links [24, 25]. The Southampton group established the Southampton Women's Survey (SWS) between 1998 and 2002 [26]. With the help of general practitioners in the city, researchers recruited women and then followed up the pregnancies and children of those who conceived. The SWS collected rich data, produced many papers, and confirmed and extended DOHaD thinking in many ways.

Through the early 1990s, we can track the process of disciplinary expansion, to incorporate new knowledge, and then its translation. *Mothers, babies and disease* – a 1994 book-length explanation of the 'Barker's hypothesis' – combined Barker's own historical epidemiological studies with a summary of research investigating adult risk factors of chronic disease; animal studies of the long-term impact of nutritional modifications, especially during so-called 'sensitive periods'; and existing clinical data [27]. Although the idea itself was not necessarily new, the disciplinary collaboration was novel, and Barker, with his team, was its tireless champion.

Yet in this interdisciplinary translation and expansion that required 'telescoping' from social conditions to dietary components and then specific molecular pathways, the link with the broader social environment became difficult to maintain. Social scientists have critiqued the ways in which social class, gender, and race are ostensibly erased in DOHaD research [9, 28]. We borrow the term 'telescoping' from Warin and colleagues who criticised the shift from the long-term impact of early-life undernutrition to overnutrition as the key question in the field [29]. In their view, this also meant a move from concerns over social determinants of health to assumptions about individual women's bodies [32]. Here we want to add another meaning: the entrance of experimental and molecular fields and the pressures of interdisciplinarity. This disciplinary structure of DOHaD as a biomedical field – rather than being situated within social medicine or even epidemiology – has profound consequences. As a recent ethnographic study of DOHaD science argued, while researchers are aware of the importance of understanding the structural reasons underlying different early life conditions, current DOHaD studies, with their focus on individual behaviour and measurement of a limited set of variables, make these connections difficult [30].

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3.3 Social Class, Health Inequalities, and Government Policy in the UK

The relationship between social class and health has long been a key preoccupation of British scientists and policymakers. Francis Galton's eugenic ideas were a defence of the existing social order based on innate and fixed biological characteristics [31]. Yet by the interwar period the practitioners of the new discipline of genetics began to insist on the precise delineation and description of heritable traits and to criticise the ambiguity of eugenics. Studies such as Lionel Penrose's Colchester Survey, which examined the heritability of 'mental retardation', pointed to a range of congenital (i.e. associated with birth or pregnancy), but not heritable, factors influencing the characteristics of the new individual [32]. The economic depression of the 1930s further exposed problems with the eugenic argument, showing that poverty rather than heritable traits was the main cause of many diseases. Soon thereafter, the Second World War strengthened social and political support for the emerging welfare state. Simultaneously, eugenics was replaced by social medicine – a new field that joined together the commitment to redistributive economic policies, public health concerns with living conditions, and the interest in 'lifestyles' [33].

Between the 1940s and 1960s, social medicine flourished at British universities [34, 35]. David Barker received his PhD in 1967 under one of the leaders of the field, Professor Thomas McKeown at the University of Birmingham. McKeown's research programme investigated the relationship between human reproduction, social conditions, and mental health [36]. Even though Barker subsequently worked in or led departments of epidemiology rather than social medicine, his methodology resembled McKeown's in its blending of historical with contemporary epidemiological data and his enduring preoccupation with social inequalities.

Barker collaborated with epidemiologists who studied links between social class and disease. He worked with Geoffrey Rose, a lead investigator in the longitudinal 'Whitehall Study' that interpreted coronary heart disease as an outcome of class-based inequalities rather than a disease of 'affluent lifestyles' [37]. Barker participated in the debate on health inequalities through his series of investigations into the links between contemporary geographical distributions of chronic diseases and the patterns of predisposing factors in earlier generations. In the 1970s, he mapped the occurrence of Paget's disease (of the bone) in the elderly onto the incidence of childhood rickets in earlier generations. This research can be understood as a precursor to his more famous 1980s studies [38]. But in contrast to McKeown, Barker was operating not in the context of a rising welfare state but in the neoliberal response to the 1970s economic crisis: a political and economic environment in which the elements of the 'welfare state' were progressively eroded.

An explicit contribution to the debate was written in 1987, when Barker quoted the report on inequalities in health by the committee led by Sir Douglas Black – a report commissioned by the Labour government but issued by the new Conservative government under Margaret Thatcher in 1980 – which explained inequalities as 'the more diffuse consequences of the class structure' [39, 40]. Barker argued that 'specific explanations may be found in the environmental influences that determined past differences in child development. These explanations may allow a national strategy for reducing inequalities in health to be developed' [39].

This quote, Barker's intellectual networks, and his epidemiological research indicate that a contribution to policymaking aimed at reducing health inequalities had long been

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one of his objectives, although perhaps out of reach through the 1980s under the Thatcher government. But by the early 1990s, conditions for health and social policy in the UK started to change. In his 1994 book *Mothers, Babies and Disease in Later Life*, Barker stressed the profound implications of emerging interdisciplinary research – his earlier epidemiological work and the incipient physiological and clinical research on 'Barker's hypothesis' – for government policy [27, pp. 170–171]. He referred to the UK's *Health of the Nation* strategy, based on the WHO's *Health for All* (1978) and launched in 1992 as 'the first attempt by a British government to develop a strategy explicitly to improve the health of the population' [41]. Yet although broadly welcomed, this strategy was also criticised, both for its disease-based model and, importantly, for not considering the socio-economic determinants of health [41, p. 4].

The Labour Party's historic accession to power in 1997, after 18 years in opposition, meant a renewed focus on socio-economic determinants of health, within a broader commitment to marshalling scientific evidence into public policy. The new government immediately commissioned a report on 'inequalities of health', with the objective of identifying priority areas for policies to 'develop beneficial, cost effective and affordable interventions to reduce health inequalities' [42]. Chaired by Donald Acheson, Barker's predecessor as Director of the MRC Environmental Epidemiology Unit in Southampton who was then appointed Chief Medical Officer of the UK, the working group included scholars who would become synonymous with research into inequalities and health, namely the epidemiologist Michael Marmot and the sociologist Hilary Graham, alongside David Barker.

DOHaD influenced the report and the British policy. Both nutrition and gender would have likely received attention, with or without DOHaD. But the explicit statement on the nutrition of women before and during pregnancy, influencing the long-term health of the next generation, and an entire discussion on the risks of reduced fetal growth, referenced by the recent work of the Southampton group, were most likely contributed by Barker [42]. Correspondence kept in the National Archives confirms this hypothesis: in a letter to Acheson dated 4 September 1997, Barker explained the policy implications of inequalities in fetal growth, and Acheson wrote on the margins, 'Thank you very much for your interesting and important letter which will be duly fed into our process.'

An early outcome of the report's – and DOHaD's– impact on British policy may be the cross-departmental programme Sure Start, which brought together social services, health, early childhood and primary education, and social justice, to improve the 'physical, social, emotional and intellectual status of children' [43]. While the original remit was children under seven years of age, as the review developed 'there was an accumulation of evidence that successful intervention in the earliest years offered the greatest potential for making a difference' [43, p. 260]. This text did not explicitly reference Barker or Acheson, but it named the Health Secretary Tessa Jowell who oversaw both the Acheson Report and Sure Start and who steered the 1997 Comprehensive Spending Review in which the Sure Start programme originated 'towards services for families and their children aged nought to three, including the pre-natal period' [43, p. 260].

In conclusion, although the reception of DOHaD in the science community in the late 1990s was not fully settled, the historical moment in British politics, with the election of a party explicitly committed to using the latest evidence to reform social and health policy, created the conditions for DOHaD to enter public policy early in the history of the field.

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3.4 Developmental Origins in the Era of Globalisation and Global Health

DOHaD began in the UK, indeed in England: at Barker's home institution, Southampton; at University College London, where Hanson led a fetal physiology group; with Alan Lucas' research team at the Childhood Nutrition Centre of the Institute of Child Health in London; and in Cambridge where Nicholas Hales' group studied clinical biochemistry and metabolism. But the field almost immediately began to expand internationally. In this section, we show how early collaborative networks were created along the established intellectual networks in the British Commonwealth. Then we show how multilateral global organisations became key proponents and advocates of DOHaD.

In 1994, Barker published a report of the 'first international study group' meeting in Sydney in October 1994, bringing together scientists from the UK, Australia, Canada, and New Zealand [44]. The geographical location is significant as it maps onto the leading centres of fetal physiology and medicine. And while these were relatively new fields, launched in the mid-twentieth century, they built on the long-standing networks of research and practice of agriculture, and especially sheep breeding, in the British Empire [45–47]. These scholarly communities had been studying animal growth for decades; they had developed sophisticated research methodologies and had easy access to animals. DOHaD provided a new framework for their research, by putting the emphasis on environmental (nutritional) influences on fetal growth, and new relevance, by linking their work to adult clinical medicine. In turn, these research communities responded enthusiastically. DOHaD groups sprung up in the UK beyond the southeast: in Bristol, Nottingham, Aberdeen, and Edinburgh; in Toronto, Adelaide, and Auckland; and in US centres with a strong tradition of animal agricultural research – Cornell and Portland, Oregon [48].

But it was human studies in the Global South that provided the key missing piece. As the previous section discussed, human prospective studies were central to the confirmation of the DOHaD hypothesis developed on retrospective historical data. And while the Southampton Women's Study was important for its rich insight into the everyday lives of 'Western' women – now conceptualised as developmental environments – prospective human studies in the Global South were important for three reasons. At least since the 1950s, biomedical scientists trying to explain and predict the impact of a rapidly changing environment upon humankind have taken the Global South populations – more likely to subsist on sparser diets than the late twentieth-century people of Europe and North America – as a window into the recent global past [49, 50]. DOHaD researchers further wanted to understand differences between human populations: do they all respond to developmental nutritional fluctuations in the same manner? Are the risks of cardiovascular and metabolic diseases the same for all? Finally, the 1990s were the heyday of the economic, cultural, and technological transformation termed 'globalisation', which helped spread the 'nutritional transition' – a shift to a diet high in ultra-processed, high-fat, and high-sugar foods – from the Global North to the South [51, 52]. By launching human studies in the Global South just as this transition was starting, DOHaD researchers hoped to capture this fleeting moment, when generations raised under 'old' nutritional regimes were bearing children into new ones.

The new DOHaD human medicine centres in the Global South were established through Commonwealth networks too, but in former British colonies rather than settler

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societies. An early and highly significant collaboration flourished between Southampton and Chittaranjan Yajnik who set up the Pune Maternal Nutrition Study in 1994. The aim was to study the long-term, especially metabolic, effect of maternal malnutrition on children born to women in villages around Pune, in the hope of explaining the much higher risk of insulin resistance and diabetes mellitus among the subcontinent's population [53, 54]. Similarly, a study in Jamaica led by Terrence Forester linked birthweight with the later response to famine: children who were born small tended to respond with the wasting illness, marasmus, and those born larger were more likely to develop the more life-threatening kwashiorkor [55]. At the Medical Research Council Unit in The Gambia, DOHaD research was integrated into an existing programme of human nutrition research in West Africa [56].

DOHaD was new, but, as we show in this section, it built upon the existing disciplinary and institutional networks largely within the British Commonwealth, with histories dating back to the British Empire. Whether these were fetal physiologists whose animal models and research methodologies built upon the structures of settler agriculture, or medical institutions and knowledge networks that traditionally prioritised diseases of greatest economic significance to the empire, their legacies and assumptions influenced the new field of DOHaD. Further research is needed – and in particular case studies focusing on specific countries or research fields and institutions – to elucidate the specific forms and impacts of these influences.

3.5 Conclusions

The field that became DOHaD started in the intimate environment of an academic workshop, but just over a decade later it had sufficient appeal and reputation to hold a world congress bringing together the research community with celebrity scholars and royalty. This chapter argued that to understand this trajectory we must situate the field in the geographical and historical context in which it was created and flourished. We then identified three key themes to help us explain both its success and the controversies: effective interdisciplinarity; ability to offer a new solution to the long-standing problem of social class in Britain; and the ability to recruit existing international knowledge and institutional networks to build a novel approach to emerging global health problems. We summarise our argument in the following way:

First, the interdisciplinarity of DOHaD was its central feature from the start: a source of innovation, intellectual richness, and an effective way to broaden the field's appeal and recognition. Yet at the same time it was a source of challenges and controversies, with the field having to reconcile diverse methodologies and data types and also respond to criticisms from different disciplinary corners. Furthermore, for all its rapid global spread, DOHaD was deeply marked by its British origins. The long-standing concern with the effect of social class on health not only influenced Barker in the formulation of his original hypothesis but also provided the opportunity and context for DOHaD to influence public policy relatively early in its history. This track record in British social and health policy, right at the time when the New Labour government was gaining international interest for its conscious attention to scientific evidence in policymaking, became important in the twentieth century [57]. This period marked the entrance of DOHaD into global health policy, at first through the established scientific connections of the former British Empire. While this could have sounded its death knell in times of

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wider recognition of the harmful legacy of this past, in fact it gave DOHaD new life as the realisation grew in the early years of the current millennium that inequalities in health affect all societies, and none more so than those passing through the nutritional and economic transitions associated with globalisation.

References

1. Hanson M, Gluckman P, Bier D, Challis J, Fleming T, Forrester T, et al. Report on the 2nd World Congress on Fetal Origins of Adult Disease, Brighton, UK, June 7–10, 2003. Pediatric Research 2004; 55: 894–897.
2. Dawes GS, Borruto F, Zacutti A, Zacutti A Jr. (eds). Fetal Autonomy and Adaptation. Chichester, UK: Wiley; 1990.
3. Kluger MO & Bartzke G. A practical guide how to tackle interdisciplinarity: A synthesis from a postgraduate group. Humanities and Social Sciences Communications 2020; 7: 47.
4. Wolstenholme G & O'Connor (eds). Fetal Autonomy. Basel: CIBA Foundation; 1968.
5. Buklijas T & Al Gailani S. A fetus in the world: Autonomy and environment in the making of fetal origins of adult disease. History and Philosophy of the Life Sciences [accepted].
6. Barker DJP & Osmond C. Infant mortality, childhood nutrition, and ischaemic heart disease in England and Wales. Lancet 1986; i: 1077–1081
7. Barker DJP, Winter PD, Osmond, C, Margetts B, & Simmonds SJ. Weight in infancy and death from ischaemic heart disease. Lancet 1989; ii: 577–580.
8. Hawkins P, Steyn C, McGarrigle HH, Calder NA, Saito T, Stratford LL et al. Cardiovascular and hypothalamic-pituitary-adrenal axis development in late gestation fetal sheep and young lambs following modest maternal nutrient restriction in early gestation. Reproduction Fertility and Development 2000; 12(8): 443–464.
9. Richardson S. The Maternal Imprint: The Contested Science of Maternal-fetal Effects. Chicago: University of Chicago Press; 2021.
10. Hanson M. 1990. Fetal baroreflexes and chemoreflexes: Adaptation and autonomy. In Dawes GS, Borruto F, Zacutti A, Zacutti A Jr, eds. Fetal Autonomy and Adaptation. Wiley, pp. 5–13.
11. Nicolson M & Fleming JEE. Imaging and Imagining the Fetus. Baltimore: Johns Hopkins University Press; 2013.
12. Fox Keller E. The Century of the Gene. Cambridge, MA: Harvard University Press; 2000.
13. Hood L & Kevles E (eds). The Code of Codes: Scientific and Social Issues in the Human Genome Project. Cambridge, MA: Harvard University Press; 1993.
14. García-Sancho M. 2015. Animal breeding in the age of biotechnology: The investigative pathway behind the cloning of Dolly the sheep. History and Philosophy of the Life Sciences 2015; 37 (3): 282–304
15. Myelnikov D. Cuts and the cutting edge: British science funding and the making of animal biotechnology in 1980s Edinburgh. British Journal of History of Science 2017; 50(4): 701–728.
16. Balls M. The ethics of research involving animals: the report of the Nuffield Council on Bioethics. *Alternatives to Laboratory Animals* 2005;33(6): 649–649.
17. Brawley L, Poston L, & Hanson MA. Mechanisms underlying the programming of small artery dysfunction: review of the model using low protein diet in pregnancy in the rat. Archives of Physiology and Biochemistry 2003 Jan; 111(1): 23–35.
18. Kenney M, & Müller R. Of rats and women: Narratives of motherhood in environmental epigenetics. BioSocieties 2017; 12(1): 23–46
19. Buklijas T. Histories and meanings of epigenetics. In: Meloni M, Lloyd S, Fitzgerald D, Cromby J, eds. Handbook of

https://doi.org/10.1017/9781009201704.005 Published online by Cambridge University Press

Biology and Society. London: Palgrave; 2018, pp. 167–187.

20. Reynolds LA & Tansey AM. Cholesterol, Atherosclerosis and Coronary Disease in the United Kingdom, 1950–2000. London: Wellcome Witness Seminar; 2005, no. 27.
21. Prainsack B. Personalized Medicine: Empowering Patients in the 21st Century. Cambridge, New York: New York Press; 2017.
22. Paneth N & Susser M. Early origin of coronary heart disease (the 'Barker hypothesis'). BMJ. 1995, 18 Feb ;310 (6977): 411–412.
23. Rich-Edwards JW & Gillman MW. Commentary: A hypothesis challenged. BMJ. 1997, 22 Nov;315(7119): 1348–1349.
24. Elford J, Shaper AG, & Whincup P. Early life experience and cardiovascular disease. Ecological Studies. Journal of Epidemiology and Community Health 1992; 46(1): 1–8.
25. Elford J, Whincup P, & Shaper AG. Early life experience and adult cardiovascular disease: Longitudinal and case-control studies. International Journal of Epidemiology 1991; 20(4): 833–844.
26. Inskip HM, Godfrey KM, Robinson SM, Law CM, Barker DJ, Cooper C. Cohort profile: The Southampton women's survey. International Journal of Epidemiology. 2006 Feb 1;35(1): 42–48.
27. Barker DJP. Mothers, Babies and Disease in Later Life. London: BMJ Publishing Group; 1994.
28. Valdez, N. Weighing the Future: Race, Science, and Pregnancy Trials in the Postgenomic Era. Berkeley: University of California Press; 2021.
29. Warin M, Moore V, Zivkovic T & Davies MJ. Telescoping the origins of obesity to women's bodies: How gender inequalities are being squeezed out of Barker's hypothesis. Annals of Human Biology 2011; 38(4): 453–460
30. Penkler M. Caring for biosocial complexity. Articulations of the environment in research on the Developmental origins of health and disease. Studies in History and Philosophy of Science 2022 Jun; 93: 1–10.
31. Bland L & Hall L. Eugenics in Britain: The view from the metropole. In: Bashford A, Levine P, eds. The Oxford Handbook of the History of Eugenics. Oxford, UK: Oxford University Press; 2012, pp. 212–227.
32. Kevles, D. In the Name of Eugenics: Genetics and the Uses of Human Heredity. Cambridge, MA: Harvard University Press; 1985.
33. Oakley A. Eugenics, social medicine and the career of Richard Titmuss in Britain 1935–50. The British Journal of Sociology 1991; 42(2): 165–194.
34. Porter D. Social medicine and the new society. Medicine and the scientific humanism in mid-twentieth century Britain. Journal of Historical Sociology 1996; 9(2): 168–187.
35. Porter D. The decline of social medicine in the 1960s. In: Porter D, ed. Social Medicine and Medical Sociology in the 20th Century. Amsterdam: Rodopi; 1997, pp. 97–119.
36. McKeown LI. Record and the epidemiology of malformations. Paediatric and Perinatal Epidemiology 1996; 10(1): 2–16.
37. Clark P. "What else can you expect from class-ridden Britain?": The Whitehall studies and health inequalities, 1968 to c. 2010. Contemporary British History 2021; 35(2): 235–257.
38. Barker DJP & Gardner MJ. Distribution of Paget's disease in England, Wales and Scotland and a possible relationship with vitamin D deficiency in childhood. British Journal of Preventive & Social Medicine 1974; 28(4): 226–232.
39. Barker DJP & Osmond C. Inequalities in health in Britain: Specific explanations in three Lancashire towns. BMJ 1987; 294: 749–752.
40. Black D. Report of the Working Group on Inequalities in Health. London:

https://doi.org/10.1017/9781009201704.005 Published online by Cambridge University Press

Department of Health and Social Security; 1980.

41. Hunter DJ, Fulop N, & Warner M. From "Health of the Nation" to "Our Healthier Nation". Policy Learning Curve Series No 2. Copenhagen: Regional Office or Europe, WHO; 2000.
42. Acheson D. Independent Inquiry into Inequalities in Health: Report. London, UK: The Stationery Office; 1997.
43. Glass N. Sure Start: The development of an early intervention programme for young children in the United Kingdom. Children & Society 1999; 13: 257–264.
44. Barker DJP, Gluckman PD & Robinson JS. Conference report: Fetal origins of adult disease – report of the First International Study Group, Sydney, 29–30 October 1994. Placenta 1995; 16 (3): 317–320.
45. Franklin S. Dolly Mixtures: The Remaking of Genealogy. Durham, NC: Duke University Press; 2007.
46. Buklijas T. Food, growth and time: Elsie Widdowson's and Robert McCance's research into prenatal and early postnatal growth. Studies in History and Philosophy of Science Part C 2014; 47: 267–277.
47. Gluckman PD & Buklijas T. Sir Graham Collingwood (Mont) Liggins. 24 June 1926–24 August 2010. Biographical Memoirs of the Royal Society 2013; 59: 193–214.
48. Fall C & Osmond C. Commentary: The developmental origins of health and disease: An appreciation of the life and work of Professor David J. P. Barker, 1938–2013. International Journal of Epidemiology 2013; 42(5): 1231–1232.
49. Ventura Santos R, Lindee S, & Sebastião de Souza V. Varieties of the primitive: Human biological diversity studies in Cold War Brazil (1962–1970). American Anthropologist. 2014 Dec;116(4): 723–735.
50. Santos RV, Coimbra Jr CE, & Radin J. 'Why Did They Die?' Biomedical narratives of epidemics and mortality among Amazonian indigenous populations in sociohistorical and anthropological contexts. Current Anthropology. 2020 Aug 1;61(4): 441–470.
51. Popkin B. Nutritional patterns and transitions. Population and Development Review 1993; 19(1): 138–157.
52. Labonté R, Mohindra K, Schrecker T. The growing impact of globalization for health and public health practice. Annual Review of Public Health 2011; 32: 263–283.
53. MRC Lifecourse Epidemiology Unit. The Pune Maternal Nutrition Study [Internet]. Southampton: University of Southampton [Cited 2022 November 1]. Available from: www.mrc.soton.ac.uk/web2/cohorts/studies-in-india/the-pune-maternal-nutrition-study/
54. Metabolic Health Digest. Q & Session with Dr. C. S. Jaynik: the one who coined the term "thin-fat" [Internet]. [Cited 2022 November 1] Available from: www.metabolichealthdigest.com/q-a-session-with-dr-c-s-yajnik-the-one-who-coined-the-term-thin-fat/.
55. Forrester TE, Badaloo AV, Boyne MS, Osmond C, Thompson D et al. Prenatal factors contribute to the emergence of kwashiorkor or marasmus in severe undernutrition: Evidence for the predictive adaptation model. PLoS One 2012; 7(4): e35907.
56. Green A. A new paradigm of the MRC units in The Gambia and Uganda. The Lancet 2018, 3 Feb; 391(10119): P418.
57. Wells P. New Labour and evidence-based policy making: 1997–2007. People, Place & Policy Online 2007; 1(1): 22–29.

https://doi.org/10.1017/9781009201704.005 Published online by Cambridge University Press