*Journal of Developmental Origins of Health and Disease* (2015), **6**(5), 475–476.

doi:10.1017/S2040174415001294



Susan L. Prescott, *Origins: Early Life Solutions to the Modern Health Crisis*. University of Western Australia Press, Crawley, Western Australia, 379 pages, ISBN: 9781742586700, 2015

*First published online 15 July 2015*

Professor (and Doctor) Prescott has given us a remarkable book that summarizes research on the sensitivity of human health to diet, lifestyle and the environment, from womb to tomb. She writes engagingly for a lay broad audience, but also cites evidence in depth for biomedical insiders. The synthesis and coverage is wide and insightful, ranging from studies of mummies that show atherosclerosis is an ancient disease to modern environmental toxic influences on the fetus from tobacco. The clinical examples are illuminated by citations of examples from the animal literature – for example, that captive marmosets also show the impact of maternal obesity on their offspring. Environmental influences begin at least as early with the health and exposures of each of our parents, and conceivably back into our grandmothers, where our natal eggs formed in our mothers' ovaries before *her* birth.

Early exposure to infections can lower allergies, as shown by Prescott's landmark *Lancet* paper in 1999,[1] proposing the fetal programming of allergic disease. Remarkable developments in this frontier field are summarized in her widely cited 2011 book, *The Allergy Epidemic, a Mystery of Modern Life*.[2] Prescott's analysis of inflammatory processes is further extended here as the core of most chronic conditions of aging, from adult obesity to atherosclerosis.

The present book begins with a concise history of the 'developmental origins of adult disease', a theory identified with David Barker and the MRC labs in Southampton, England. She describes the momentous meeting in Mumbai in 2001 organized by Barker, with Caroline Fall and Mark Hanson, which I also attended. We all perceived that a scientific frontier was being advanced. A decade later in 2011, these concepts were reified by the UN General Assembly in the Shanghai declaration on non-communicable diseases (NCDs), which the UN Secretary General Ban-Ki Moon described as 'a public health emergency in slow motion'. This global imperative seeks to improve early nurture as a pragmatic approach to costly interventions for premature chronic diseases. Not all first world governments are on board.

In the face of today's obesity epidemic, it is worth pondering the major nutritional improvements from a century ago. Even in 1900, hunger was rampant within the industrialized nations. Hear from London in 1903, when the British Empire was at a pinnacle of wealth, where a poor man tells, '… Look at my scrawny arm … it's too late to make up for what I didn't eat when as a kiddy. Mother died and there were six of us and dad. Older children starve more. By the time the younger ones come along, the older ones are starting to work, and there is more money coming in, and more food to go around'.[3] The illustrated monograph this comes from[3] gives first-hand observations of human misery in the East End of London; the author is *the* Jack London, best known for novels, whose keen and quantitative observations on the London poor can be ranked with Henry Mayhew's of fifty years before.[4] The parents and grandparents in Barker's seminal studies of Hertfordshire may have transmitted such privations to the low birth weights during 1911–1930, which was linked to proportionately more coronary disease.[5]

Back to the 21st century, Prescott notes the new developmental hazards from air pollution that were derived from automotive traffic. Not available when her book went to press are associations of air pollution with childhood obesity. The Southern California Children's Health Study showed that children living close to freeways were more obese, moreover, with additive effects of household tobacco smoke.[6] Exposure to traffic pollution and tobacco is more prevalent in poor households, as Prescott discussed. Despite the decrease in adult smoking during the past decade, in 2011–2012, 40.6% of US children aged 3–11 were exposed to secondhand smoke.[7]

In a work of this scope, minor errors can creep in such as overstating the benefits of caloric restriction '… Rodents generally live 50% longer with caloric restriction' (p. 255). In fact, most studies of inbred rodents find <20% increase, with major differences by genotype and sex.[8,9] Caloric restriction shortened lifespan of some genotypes, particularly those with the greatest fat reduction. A QTL analysis identified loci for fat maintenance during caloric restriction that favored longevity,[9] whereas wild-derived mice (true wild-type!) had minimal increase of lifespan.[10] Intriguingly, the long-lived Ames dwarf mouse, which lives beyond most calorically restricted mice, is on the chubby side, with benefits to insulin signaling that depend on its visceral fat.[11] Although early-life obesity remains a risk factor for NCD, we know little about the role of specific fat depots in later life health.

Another recent book merits mention: Laura Dawes' *Childhood Obesity in America*[12] covers the social and political history of obesity, which was even a target of psychoanalysis. Both books discuss the politics of government and industry in food production and marketing that continue the toxic engorgement of sugary, fatty foods. The global scourge of obesity is likely to persist for several generations.

C. E. Finch
*Davis School of Gerontology, University of Southern California*
*Los Angeles, CA, USA*

## References

1. Prescott SL, Macaubas C, Smallacombe T, *et al*. Development of allergen-specific T-cell memory in atopic and normal children. *Lancet*. 1999; 353, 196–200.

2. Prescott SL. *The Allergy Epidemic, a Mystery of Modern Life*, 2011. UWA Publishing: Crawley, Western Australia.
3. London J 1903. *The People of the Abyss* (reprinted, 1995, L Hill Books: Boston, Massachusetts).
4. Mayhew H 1851. In *London Labour and the London Poor* (ed. Douglas-Fairhurst R), 2010; 472pp. Oxford University Press: Oxford, UK.
5. Barker DJ, Winter PD, Osmond C, Margetts B, Simmonds SJ. Weight in infancy and death from ischaemic heart disease. *Lancet*. 1989; 2, 577–580.
6. McConnell R, Shen E, Gilliland FD, *et al*. A longitudinal cohort study of body mass index and childhood exposure to secondhand tobacco smoke and air pollution: the Southern California Children's Health Study. *Environ Health Perspect*. 2015; 123, 360–366.
7. Homa DM, Neff LJ, King BA, *et al*. Vital signs: disparities in nonsmokers exposure to second hand smoke – U.S, 1999–2012. *CDC Morb Mortal Wkly Rep*. 2015; 64, 103–108.
8. Swindell WR. Dietary restriction in rats and mice: a meta-analysis and review of the evidence for genotype-dependent effects on lifespan. *Ageing Res Rev*. 2012; 11, 254–270.
9. Liao CY, Rikke BA, Johnson TE, *et al*. Fat maintenance is a predictor of the murine lifespan response to dietary restriction. *Aging Cell*. 2011; 10, 629–639.
10. Harper JM, Leathers CW, Austad SN. Does caloric restriction extend life in wild mice? *Aging Cell*. 2006; 5, 441–449.
11. Menon V, Zhi X, Hossain T, *et al*. The contribution of visceral fat to improved insulin signaling in Ames dwarf mice. *Aging Cell*. 2014; 13, 497–506.
12. Dawes L. *Childhood Obesity in America*, 2014. Harvard: Cambridge, Massachusetts, US.