Commentary: Biological Freudianism and the quest for understanding of the social origins of health

Vivienne Moore

Dubos and colleagues’ stated purpose1 was twofold, to demonstrate that the early environment could have a lasting influence on biological characteristics and to illustrate the ability of laboratory models to address ‘socio-medical’ problems occurring in human populations. These twin purposes were drawn together at the end of the paper in the call for ‘an experimental science that might be called biological Freudianism’.

Three years later, in 1969, René Dubos won the Pulitzer Prize for his book So Human an Animal, which had the unifying theme ‘that all experiences leave a stamp on both physical and mental characteristics’, with special emphasis placed on ‘very early influences, prenatal as well as postnatal, because their effects are so profound and lasting’ (p. x). 2 Other writings reveal an even broader appreciation of the connectedness of human experience with the evolutionary past and the ecological present. 3-5

Acclaim as a great thinker followed earlier career success at the forefront of microbiology in the 1930s, when Dubos identified soil microbes that were precursors of antibiotics (and Selman Waksman, under whom he studied at Rutgers, went on to receive a Nobel Prize for the discovery of streptomycin). Amidst the promise of antibiotics, Dubos keenly recognized the social origins of health and disease. He wrote, “The belief that disease can be conquered through the use of drugs [is a] fallacy [in] that it fails to take into account the difficulties arising from the ecological complexity of human problems ... does not solve the problem of disease in the social body’ (p. 83). 3

The paper by Dubos et al.1 reprinted here predates an eight-part series on ‘Lasting biological effects of early environmental influences’ which Dubos published with other colleagues, presenting data on a wider range of exposures. 6 The experiments reported by Dubos et al.1 largely concern the manipulation of postnatal nutrition or infection, with growth in weight the primary outcome. There are some striking features of this work which foreshadow current preoccupations in the field of the early life origins of adult disease and further illustrate the tidal history of underpinning concepts. 7

(i) Not only do Dubos et al. present animal experiments investigating main effects on growth, they also demonstrate an interaction: mice bred in a relatively pathogen-free environment showed greater weight advantages over control mice when both groups were fed an inadequate diet. This is a perceptive approach to research concerning environmental influences on health. Exploration of synergistic combinations of exposures has been limited in more recent research concerning the early environment, although this is changing, 8 partly under the aegis of a lifecourse perspective.

Nutrition and infection, the interacting factors examined by Dubos et al., were recognized as a potent compound by the World Health Organisation in the early 1970s, when improved nutrition was urged as a strategy for reducing infectious disease in developing countries. Around that time, some commentators believed it ‘likely that the interaction between nutrition and infection [is] more important in animal and human populations than one would predict from the results of laboratory investigations’ (p. 39). 9 Interestingly, despite an accumulation of supportive ecological data, good evidence at the individual level remains elusive. 10

(ii) Dubos et al. attempted to identify a critical period, a time window in development during which an exposure has an enduring effect, a relationship that is absent outside of this window. Dubos et al. reported that the growth of rat pups was permanently depressed when the nursing dams were fed ‘Diet E’, but not when the inadequate diet was introduced more than five days following the birth of the pups. Critical periods are a long-standing notion in the developmental sciences, to which attention has once again returned. 11 Additional sophistication in conceptualizing critical periods (yet to be realized in research) has followed the resurgence of the lifecourse perspective, which recognizes that critical periods may not operate universally but may depend on the prior experience of the individual. 12

(iii) Dubos et al. concentrated on mild adverse exposures: ‘natural’ variability in mouse mothering, a slight reduction in dietary nutrients, a single dose of pathogenic material. (This has implications for sample size which they bear resolutely, in a single day reassigning 240 mice pups to foster mothers!) In concert with the focus on mild exposures, Dubos et al. were aware that consequences may be visible only under conditions of challenge, as encapsulated in their penultimate paragraph. Renewed appreciation of subtlety in exposures and in manifestation of consequences is now being seen in work on the early life origins of adult disease. 5, 13, 14

Dubos et al. 1 argued for systematic expansion of their work and sought to pay intellectual tribute to Freud, who was esteemed at the time for recognizing ‘continuity in the mental life of the individual, [so] symptoms were not mysterious incursions from without but rather exaggerated expressions of processes common to everyone which revealed the specific stresses of the patient who developed them’ (p. 3). 15 The wish to acknowledge Freud is somewhat curious because in other writings Dubos draws from a rich and recurrent theme of...
legacies of past experience, spanning the arts and sciences over several centuries. The choice may reflect Dubos’s concern that contemporary biology perpetuated the separation of the body from the mind, and may be a gesture towards overcoming this problem. Also, although Freud is now remembered largely for his work on psychoanalysis, like Dubos he articulated a role for archaic heritage and wrote expansively about problems of civilization.16

To a social epidemiologist, the disappointing aspect of the paper was the call by such an impressive thinker for a narrow way forward. Dubos’s championing of animal models ignores rather than addresses the complexity of human experience. It is an approach that poorly acknowledges multiple pathways or differences in the relative importance of factors, and it provides relatively meagre insight into the social production of health.

The fact that Dubos saw animal experiments as the key to research progress would seem to reflect his original training in microbiology, the intellectual forebears he emulated, the research climate in the United States in the 1960s, and his particular views on science. To elaborate a little, the practice of research using animal models owes much to Claude Bernard,17 who in the nineteenth century strove to make medicine more scientific by that means. Other scientists admired by Dubos included Pasteur (about whom he wrote two books) and Faraday, whom he described as ‘a peerless experimenter’ (p. 163).3 Dubos spent most of his working life at the Rockefeller University; the third quarter of the twentieth century has been described as a ‘golden age’ for funding of basic sciences in the United States and saw the spectacular rise of molecular biology.18 Dubos, perhaps inevitably, held that ‘much of scientific knowledge is concerned not with objects and events as they occur naturally, but with fragments of nature produced artificially by analytical breakdown. Indeed, this analytical approach to knowledge is perhaps the one characteristic that most sharply differentiates science from art in the attempt to apprehend reality’ (p. 163).3 Almost in the next breath, Dubos concedes that ‘In reality, however, some of the great leaps in the scientific advance have come not from the detailed analysis of mechanisms but rather from the total “intuitive” apprehension of reality.’ For Dubos, in this passage and more generally, there seems to be no bridge of rigorous inquiry between this creative dimension and experiment—no path through epidemiology, sociology, anthropology, or other disciplines. This is odd in some ways, as Dubos elsewhere expounded upon the limitations of science and was dissatisfied with the ‘machine view of living things’ (p. 105).3

The position of Dubos et al. on the utility of animal experiments contrasts strongly with views held by epidemiologists. For example, participants in a 1997 workshop on the nature of the evidence required to demonstrate nutritional effects and to form the basis of recommendations concluded that ‘no other type of nutritional science can equal epidemiology in the relevance of either the dietary exposures or the health outcomes’.19 Several papers from that workshop detail the role of animal experiments. Potischman and Weed,20 for example, find that animal models contribute to evidence of biological plausibility (for which need varies), one of five criteria found to be used in practice to judge causality (in the tradition of the US Surgeon General and Bradford Hill). The contrast between these positions highlights the tension between pragmatic and manipulative bases for attributing causality.21 The former offers a means to identify distal societal causes by applying a system of thinking to observations and using a process of elimination (of confounding), whereas the latter is founded on controlled perturbation of a single factor of interest and emphasizes proximal causes. Perhaps Dubos, with his interest in the history and philosophy of science, would have been more persuaded of the limitations of animal experiments by flaws identified within the logic of the hypothetico-deductive framework, in particular the argument that proceeding by analogy (an animal model) is an inherently weak means of demonstrating causation in humans and suffers from the problem of ‘causal-functional asymmetry’ (similarity of causal properties cannot be inferred from similarity of functional properties, although differences in causal properties may be inferred from differences in functional properties).22

Just as a ‘rapprochement with biology’ has been upheld as a way to strengthen the discipline of epidemiology,23 linkage with other forms of research might have been part of Dubos and colleagues’ path forward. In the absence of this, Dubos et al. remain fundamentally limited in their ability to integrate the social, despite a strong desire to do so. Contemporary epidemiologists also need to be aware of methodological conceit. Although randomized controlled trials (RCTs) are powerful tools, in the words of Ezzy, ‘the underlying worldview that privileges RCTs as the “gold standard” against which all other methodologies must be assessed results in a failure to properly research, or understand, the dimensions of health and illness that cannot be studied utilising RCTs. It is difficult and quite unusual, for example, to conduct randomised controlled trials of the effects of clean water, or of poverty, or of international debt repayments on the health of people’ (p. 52)24 and observational epidemiology likewise ‘privileges the aspects of social life that can be measured and statistically summarised’ (p. 54).24 Respecting and supporting research in other disciplines, including those with different epistemological bases, must be part of our approach to improving the health of populations.

Acknowledgements

My thanks to Michael Davies, John Moss, and Megan Warin for helpful discussions and comments.

References


