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CORRESPONDENCE

Biography and biological capital

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Abstract
At the crossroads between sciences, epidemiology brings together the social and the biological to examine social inequalities in health. The concept of biological capital represents the accumulated history of biological experiences, alongside the other forms of accumulated capital, notably cultural, economic and social. The ability to access the three other forms of individual capital and therefore position in life depends on inherited biological health/skills, epigenetic imprinting and the accumulation of embodied biological changes that make an individual more or less successful in life. We present results from analyses carried out within the Lifepath consortium, showing that the socioeconomic environment, from early life and over the lifecourse, is an important risk factor for health and partly works through its effects on biological mechanisms. We show that socially stratified pre-disease states related to ageing may be examined using biomarkers, and help underline areas and mechanisms to promote healthy ageing.

Keywords Life-course epidemiology · Biomarkers · Allostatic load

Epidemiology is at the cross-roads between social and natural sciences. This is particularly evident when we consider social inequalities in health. However, there is still a wide gap between the two fields, both methodologically and conceptually. Natural sciences focus in particular on biological mechanisms and outcomes, i.e. they deal with "zoe", the biological life, while social sciences have to do with "bios", the biographical life, according to the terminology used by Dworkin [1]. It is obvious that what we epidemiologists try to do is to connect zoe and bios in a meaningful way, though this attempt has rarely been made explicit, except e.g. in the work of Nancy Krieger through the concept of "embodiment" [2].

But there is another way—more practical—we can look at the relationships between social and natural sciences, that is the transfer of epidemiological knowledge into the concept of "biological capital" in addition to the better known economic, social and cultural capitals. The French sociologist Pierre Bourdieu, in particular, has explained the role of social and cultural capital in the functioning of societies and in social inequality. While "the social world is accumulated history" [3], so too is the individual life at any particular time/age: it is the accumulated history of all economic, social, cultural and eventually biological experiences that have had an impact on the body. *Biology and biography* meet for example through health status, depending on social position at a given age. According to Bourdieu, "capital can present itself in three fundamental guises: as *economic capital*, that is immediately and directly convertible into money and may be institutionalized in the form of property rights; as *cultural capital*, which is convertible, on certain conditions, into economic capital and may be institutionalized in the form of educational qualifications; and as *social capital*, made up of special obligations ("connections"), which is convertible, in certain conditions, into economic capital and may be institutionalized in the form of a title of nobility" [3]. Students of social inequalities may recognize here a reflection of the three tenets of socio-economic position as they have been investigated and classified in epidemiology, i.e. occupation, income and education. However, how this connects to biology is not explained by Bourdieu, and biological capital is the missing concept. Nevertheless, the ability to access the three other forms of individual capital

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and therefore position in life depends on inherited biological health/skills, epigenetic imprinting and the accumulation of embodied biological changes that make an individual more or less successful in life.

These principles start to be incorporated into epidemiological research, via the integration of social contexts and biomarkers through a life-course approach, as we have done in the Lifepath consortium [4]. The results from analyses carried out within Lifepath show that the socioeconomic environment, from early life and over the lifecourse, is an important risk factor for health and partly works through its effects on biological mechanisms.

Let us consider successful ageing and the impact of socioeconomic position. Malakov et al [5] state the evolutionary "problem" of ageing as such:

> Aging is deleterious for Darwinian fitness, yet is a pervasive feature of most living beings. Given the large number of known repair mechanisms, it is not clear why organisms should senesce. This apparent paradox is resolved by the evolutionary theory of aging, which relies on the fundamental principle that the strength of natural selection declines with age, because of extrinsic (non aging-related) mortality resulting from the cumulative effects of a variety of biotic and abiotic factors [5].

This highlights the "universal" and cross-species nature of ageing, and its relationship with evolutionary theory. The demands and challenges of living each day result in a depletion of biological resources to restore organisms to full function. Adaptive biological strategies to maximise survival and fitness occur from conception onwards. The literature on developmental origins of adult health and disease is rife with instances of biological adaptive responses to external conditions. These are proxies of ageing, just occurring very early in the maturation process. Evidence stemming from animal studies supports the proposal that a biological event occurring during a critical period of animal development can permanently 'program' the organism. Lucas provides a working definition of this notion of *programming*, whereby "an early stimulus or insult, operating at a critical or sensitive period, results in permanent or long-term change in the structure or function of the organism" [6]. By being 'programmed' an organism responds to stimuli from its environment by optimally adapting itself to the prevailing conditions, thus potentially prolonging its survival in such an environment. However, this adaptation is only beneficial to the individual in the short-term because, by fixing its adaptation so early, the organism is accommodating to one set of circumstances, but is not necessarily adapted if those circumstances should change.

Our environment is highly variable requiring the permanent adaptation of physiological systems. This adaption through changes is crucial for survival and refers to *allostasis* [7]. Multiple physiological systems, nervous, endocrine and immune, are involved in the allostasis processes, all of which mature during the postnatal period into adulthood. Chronic exposures to stressors but also interindividual differences in the susceptibility to environmental stressors are associated with a prolonged activation of allostatic systems. This may lead to an allostatic overload with potentially detrimental health consequences. Allostatic load (AL) is therefore the price paid by the body over time for adapting to challenges. It refers to the concept of biological multisystem wastage, whereby "the strain on the body produced by repeated ups and downs of physiologic response, as well as by the elevated activity of physiologic systems under challenge, and the changes in metabolism and the impact of wear and tear on a number of organs and tissues, can predispose the organism to disease" [8]. Various scores of AL have since been proposed and have been shown to be better predictors of mortality and functional limitations than the metabolic syndrome or any of the individual components used to measure AL when analysed separately. The AL score is also associated with an increased incidence of cardiovascular disease, and poorer cognitive function. Recent research also suggests a link between early environment and AL and AL-related outcomes [9].

Epigenetics, specifically DNA methylation modifications, has recently been proposed as a source of biomarkers of biological ageing and as one of the plausible mechanisms through which social exposures become biologically embodied, affecting physiological systems and cellular pathways leading to disease susceptibility [10]. The 'epigenetic clock' is one of the main mechanisms contributing to age-related methylation changes [11]. It refers to specific sites on the genome where methylation levels constantly change as the body ages and can therefore be used to predict chronological age with high accuracy [12].

Disadvantaged socio-economic position (SEP) in early life may shape lifestyle and health-related behaviours, which then affect health in adulthood (Fig. 1). In fact, it may be associated with poor early life nutrition; tobacco exposure in utero, infancy and childhood; foetal growth restriction or premature birth. A child's development is clearly sensitive to the surrounding environment in early childhood and to the availability of sufficient economic resources. Socioeconomic disadvantage in childhood may result in educational disadvantage, which in turn drives economic disadvantage in adulthood. As a whole, this evidence has important implications for health and social policy.

In the *Lifepath* consortium we found that disadvantaged SEP can lead to physiological wear-and-tear involving inflammatory responses, impaired immune function, i.e. what has been called "Inflammaging" [14], and epigenetic acceleration of ageing [15]. One approach taken by *Lifepath*

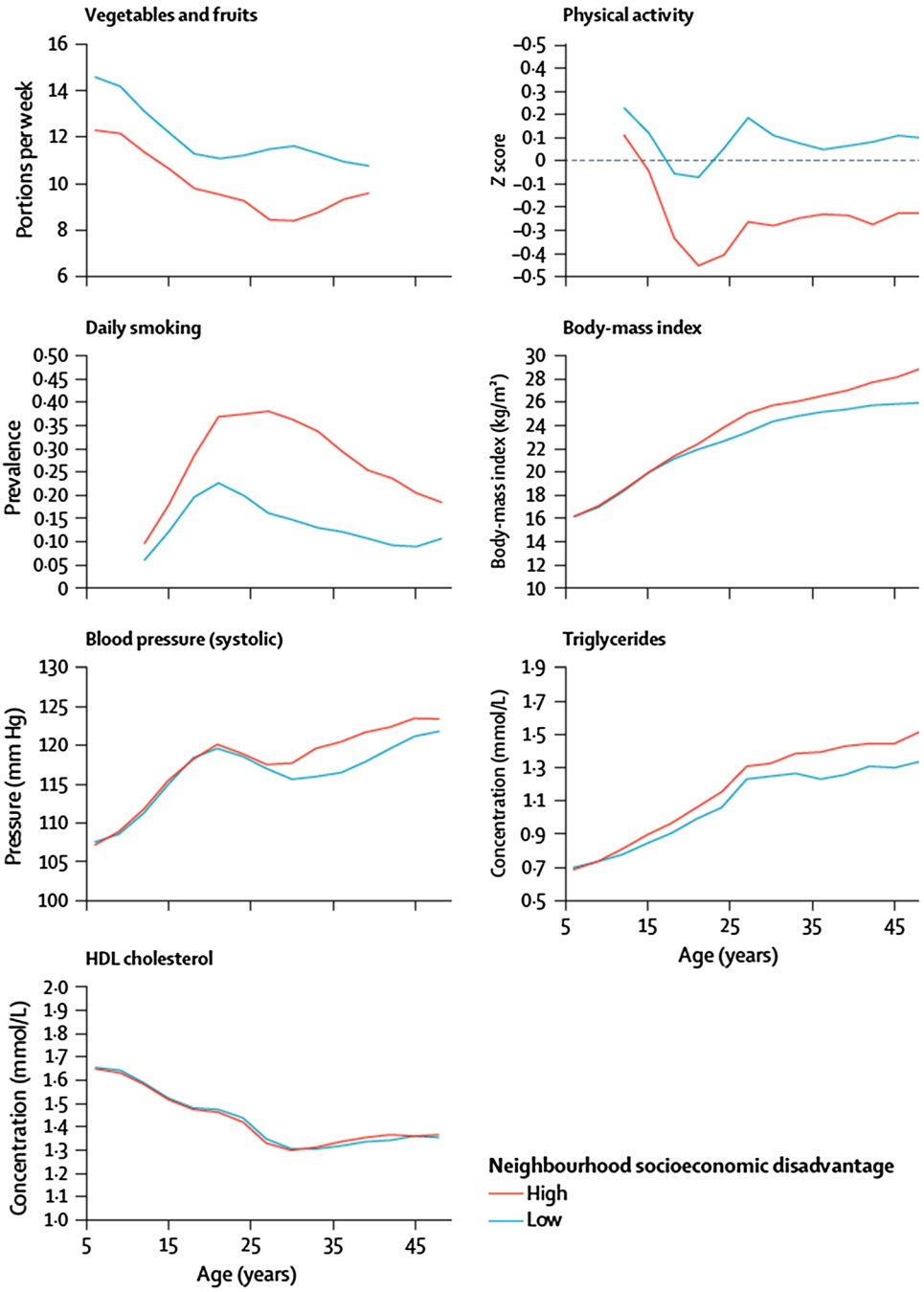

Fig. 1 Risk factors of cardiometabolic health by age and cumulative neighbourhood socioeconomic disadvantage (25) (blue = less disadvantaged; red = more disadvantaged) (from ref. 13)

was to examine allostatic load as a composite measure of overall physiological wear-and-tear. For example, data from the 1958 British birth cohort indicated that lower maternal education and paternal manual occupation were associated with a higher (meaning worse) allostatic load at 44 years [16]. The research suggests a pathway whereby parental occupation and education affect children's education and this then impacts on later life. Other *Lifepath* analyses focused specifically on how differences in SEP are revealed in the DNA of our cells. DNA methylation is used to represent overall biological ageing and has been linked with educational attainment of individuals [17]. Disadvantaged SEP was associated with accelerated ageing. The results suggest that biological ageing was more rapid in individuals with fewer years of education.

These early but consistent findings show the value of using biological markers to understand the relationship between social factors and health, and materialize the concept of biological capital in epidemiology and the social sciences. Pre-disease states can be identified using markers such as DNA methylation and composite indicators like the allostatic load, picking up on the phenomenon of socially

patterned accelerated ageing before the onset of diseases. Biomarkers can be used to explore the impacts of income inequality: more unequal societies are thought to produce higher levels of biological damage via chemical and physical stressors (including unhealthy behaviours), but also via psychosocial stress in response to 'status anxiety' at the individual level, and a growing amount of evidence highlights the role of chronic inflammation in this connection.

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