first based the class struggle on domination, the second did it on exploitation. In my work, I don’t deal with either theme. I relate the health inequalities that we find in our societies to the different forms of class power, without relating it to exploitation or domination, showing, for example, that countries that are worker friendly (like those governed by the Social Democratic Tradition) have better health indicators than countries governed by capitalist class friendly parties like the US.

Marx has had an enormous influence on Western Europe (particularly in continental Europe) where I come from, and I am gratified that my work has been enriched by that tradition. In that respect, I am pleased to be endowed with that tradition. But that is not the point. I will be glad to be defined in these terms when other authors are identified by the traditions that have influenced them. But, the use of that term in the US is not descriptive; it is used to show specific adversity. I find it, therefore, abusive and offensive to the extreme when the term Marxism is always used to define my work in the US (rarely is my name cited without putting the name Marxism before it). That practice translates to ignorance or malice since it aims at stigmatizing and marginalizing any critical tradition.

I also find abusive that in a tradition where class is practically non-existent, those who continue labouring on that front are called class reductionists, which ignores the extensive work that many of us have done in other areas as well, such as race and gender. When SS–MW write that class analysis is inadequate, they do not mean it is insufficient (which I would agree with), but rather that it is wrong. That is the meaning of their reply, which fits in with their intent of promoting their social capital outcomes. 2 This is in contrast to Barker’s hypothesis of foetal origin of adult chronic disease that is currently receiving greater attention among investigators. 2

The recent study1 in India is a snapshot of the urban elite. Despite the advantage of a longitudinal birth cohort,1 the investigators have not adequately addressed the two potential environmental risk factors for excess weight gain across the life course: altered nutrition and physical inactivity levels. Delhi is experiencing a plethora of fast food outlets, with disposable income in the hands of many. There is also the onslaught of increased mechanization leading to a relatively sedentary lifestyle. Such changing lifestyles compounded with a rapid urbanization may result in the emergence of another paradox—‘affluence’. The authors proposed a ‘growth trajectory’ phenomenon,1 but reverse causality bias cannot be ruled out, particularly when body mass index has been used as a proxy measure for adiposity.

Historical birth cohorts are rich national resources for providing powerful evidence across the life course as regards exposure timing, biological pathways, and potential mechanisms such as ‘inter-generational effects’, ‘intra-uterine programming’,

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Adult chronic disease and childhood obesity: a life course approach in developing countries?
From ZUBAIR KABIR

The developing world is undergoing nutritional transition, along with anthropometrical transition. Many developing countries have been advocating blanket childhood food supplementation programmes for decades. As a public-health doctor, I am concerned about such health promotional activities where short-term benefits may outweigh any long-term harm. A recent study in India also showed that childhood obesity, together with low birth weight, is associated with Type 2 Diabetes in early adulthood among an urban slum community.1

Low birth weight and undernutrition are still considered the two main causes of childhood mortality and morbidity in developing countries. Consequently, an apparent association between low birth weight and childhood obesity seems to be counter-intuitive for the lay public. For reproductive epidemiologists, birth weight (a surrogate measure for intrauterine environment) has been a paradox, because birth weight is argued not to be on the ‘causal’ pathway to population-health

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‘adiposity rebound’ phenomenon, or ‘thrifty phenotype’. While such epidemiological paradigms are fashionable, the narrow framework of Barker’s hypothesis has also been extended to post-natal growth and developmental trajectories. However, it is difficult to disentangle the real culprit of nurture from nurture: junk food, an elusive ‘fat gene’, gene-environment interactions, or the heterogeneity of biological pathways linking early life exposures to later outcomes.

To date, limited life course epidemiological evidence exists in developing countries. Such evidence reported in the developed world cannot be generalized. Therefore, historical birth cohorts and expertise are both necessary for the resource-poor countries where cost-effective public-health policies drawn on the conventional ‘black box’ (risk factor) epidemiology are also firmly established. So, how realistic can long-term untested policies based on interdisciplinary life course epidemiological approaches or utilizing sophisticated ‘Mendelian randomisation’ techniques be in situations where ‘joined-up thinking’ is not even the norm! Should our policy makers and public-health leaders replace ‘prevarication with imagination’ as to empower our future generations with the ultimate health prevention models that are built on life course approaches, and integrated into a macro-environment?

References


Black tea and cardiovascular disease

From SHINKAN TOKUDOME,1* IMAEDA NAHOMI,2 CHIHO GOTO,3 YUKO TOKUDOME3 and MALCOLM A MOORE4

Dr Sesso et al.1 could not detect a significant association between black tea consumption and risk of cardiovascular disease using well-established cohorts for the study on health and physical activity. Dr Poole et al.2 have commented on the paper with regard to the concept of causation. We would like to add another view based on nutritional epidemiology and biological pathogenesis.

Potential protective effects of black tea against cardiovascular disease and cancer are attributed to polyphenol compounds and flavonoids/flavanols, including catechin/EGCG and theaflavin. The authors admit that they lacked a data-based approach for selecting foods/beverages contributory to certain nutrients in order to assess intake of flavonols/theaflavins from black tea.

We can assume from the literature that black tea is a major source of catechin/EGCG, but comparisons within several cups of black tea may not have enough power to detect any favourable effects of catechin/EGCG. In other words, a dose–response relationship could not be proven even after taking into account confounding coffee consumption. Although thus far inconsistent, some beneficial effects have been experienced with large intakes of black/green tea, such as ≥10 cups/day.5–8 We need a wide range of comparisons for cups of black tea to evaluate possible protective effects, if any, on cardiovascular disease.

The concentrations of catechin/EGCG in black tea are rather less than in green tea.4 In addition, antioxidant activity of black tea scored by oxygen radical absorbing capacity (ORAC) is lower than that for green tea. Furthermore, flavonoids are supplied to a greater extent by vegetables and fruit than several cups of black tea. Thus the authors should, at least, adjust for effects of consumption of vegetables and fruit.

Finally, it is known that folate is antiangiogenic because it is a cofactor in the metabolism of homocysteine to methionine. According to our recent study, folate is supplied by green tea along with vegetables and fruit; however, its content in black tea is far less than in green tea. Black tea thus seems generally less anticarcinogenic, antimutagenic, and antiangiogenic than green tea. Moreover, any fluids/beverages, including water, black/green tea, and coffee, may be important in terms of blood viscosity and excretion/dilution of mutagenic and carcinogenic substances.10,11

References