



Productive simplification in the use of anthropometric nutritional status

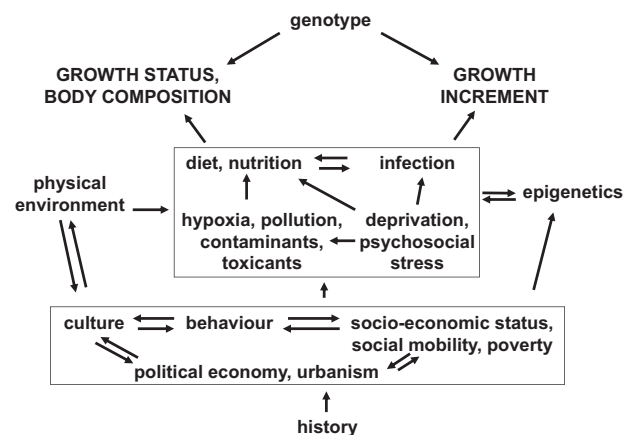
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The article by Scheffler et al. [1] demonstrates again, with an analysis of cross-sectional anthropometry and some clinical measures of undernutrition in Indonesian children aged 6–13.2 years, that malnutrition may lead to growth stunting, but that stunting by itself does not indicate malnutrition. The letter of Tanjung et al. [2] questions Scheffler et al.'s conclusion, citing evidence from the Multicentre Growth Reference Study Group [3]. The response of Hermanussen et al. [4], with a much more limited set of authors, questions the validity of this reference study group to represent ideal patterns of child growth. Much of the argument on both sides sits on the use (or non-use) of observations in often small populations past and recent to support (or refute) the idea that growth stunting is not (or is) an indicator of undernutrition. This exchange of views highlights two issues. The first is that the use of anthropometric nutritional status relies on productive simplification for its ease of use. The second is that the data used to build the case either side is not big enough for the purpose they are using it, although Tanjung et al. [2] draw on the largest systematically organised dataset available. Both issues will be returned to, after an examination of the type of problem that undernutrition is seen to be, according to the type of practice whose job it is to intervene in its production.

That malnutrition can lead to growth stunting, and stunting by itself does not indicate malnutrition, is far from new knowledge. Various studies across many years have shown that there are many factors associated with the depression of linear growth, including infection, poor diet quality, low socioeconomic status, low birthweight, hypoxia, environmental pollution, deprivation, psychosocial stress, social and economic status, poverty, and structural

violence in some countries [5]. Fig. 1 illustrates this, incorporating also epigenetics [6]. A recently published study [7] has examined the relationships between change in growth stunting and distal, intermediate, and proximal level factors using multilevel pooled trend analysis of data from 50 Demographic and Health Surveys since 2000 in fourteen countries (Bangladesh, Cambodia, Ethiopia, Haiti, Kenya, Malawi, Mali, Nepal, Nigeria, Rwanda, Tanzania, Uganda, Zambia, and Zimbabwe). These authors found that at a distal level, a decrease in the Gini coefficient, an improvement in women's decision-making, and an increase in urbanisation were each significantly associated with a lower probability of within-country stunting. Intermediate-level factors associated with change in stunting rates were observed to be improvements in access to improved sanitation and drinking water, and access to vaccination. The proximal factors associated with declines in rates of stunting were increased early initiation of breastfeeding within a population, and a decreased prevalence of low birthweight. These observations map well onto the biocultural ecology of child growth shown in Fig. 1. The gini coefficient and urbanisation represent country-level political economic



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Fig. 1 Biocultural ecology of child growth (modified from Uljaszek [6]).

factors, socioeconomic status, social mobility and poverty, while women's decision making reflects cultural and political economic factors. The intermediate-level factors identified by Argaw et al. [7] map onto exposure to infectious disease, while the proximate factors of birthweight and breastfeeding map onto epigenetics, nutrition, and exposure to infection, respectively. The Argaw et al. [7] analysis allows priorities to be set for intervention for reduction in rates of stunting in low and middle income countries. In order, they are to decrease the rates of low birth weight, increase rates of vaccination, to improve water and sanitation provision, to further promote early initiation of breastfeeding and to reduce economic inequality, to further promote urbanisation, and to increase levels of female empowerment. The infrastructure changes needed to bring these interventions into greater effect are overwhelmingly political-economic—with respect to taxation and welfare regimes, education, urban planning, and health service provision.

That the incorporation of clinical signs, indicated by Jelliffe [8] and others more than half a century ago, are of value for medical approaches to undernutrition is important without doubt, but it is less clear whether such incorporation would benefit most other approaches to it. The analysis of Argaw et al. [7] shows that reducing growth stunting at the population level can be more effectively approached from social, economic and political perspectives than from the medical one. Medicalisation of social issues more broadly has not proved most fruitful in the past [9], although medicine will always be of help for those with extreme undernutrition.

To try to understand why extensive evidence—to the effect that malnutrition may lead to growth stunting, but that stunting by itself does not indicate malnutrition—is repeatedly ignored by public health practitioners and policy makers, a detour into a parallel debate may be helpful. This is in relation to another form of malnutrition, of obesity, in relation to environmental factors that may associate with it [10]. Using machine reading and network analysis of the published scientific literature on obesity and environment, Jensen et al. [10] identify internal divisions in this research field not obvious from traditional readings of the literature. The divisions in this case are outcomes of a number of productive simplifications that use obesity measures for a range of purposes—institutional feeding, biomedicine, epidemiology, and policy. In the generation of productive simplifications, it is acknowledged that truths held about the phenomenon in question are partial, but are good enough for the purposes in hand. In the case of undernutrition, the purposes to which the simplification, that linear growth represents nutritional status, is productive includes epidemiological, public health, and governmental ones. Each interest group holds a different stake in this issue, each

framing the issue with different rationalities [11]. This productive simplification thus deliberately ignores factors other than nutrition and infection that are known to associate with growth faltering, making the matter in turn measurable, treatable, controllable and governable, respectively.

For obesity, the deployment of particular simplifications, assumptions, or standards has facilitated the development of complex understandings of particular obesity-related phenomena [10]. So it is with undernutrition also. The metrics of the epidemiology of undernutrition underpin policy and intervention debates at high and low levels, and anthropometry is an easy measure to undertake. It is easily intuited, has a long history of use, is cheap to carry out, and has a deep interpretive base generated from considerable research across many decades. In the case of undernutrition, anthropometry, using standardised metrics in very regulated ways, is the simplest way to universalise from science to policy to intervention. The meanings of such measurements are under constant review, however, from their use in assessing human biological quality in the eighteenth and nineteenth centuries, to anthropological enquiry in the nineteenth century, and in the twentieth and twenty first centuries as measures of social welfare in public health, economics and national statistics [12]. Simplifications can always be queried, but it is important that their productiveness for different uses beyond the medical should be evaluated in parallel. In the case of the growth of the Indonesian children of Scheffler et al.'s study [1], the question left unanswered is the extent to which reducing simplification by adding clinical measures can increase overall productiveness for the control of undernutrition at the population level.

That people in different places grow differently, and that many factors contribute to this, is clear. The factors and actors interact to produce linear growth are not easily teased apart, however. As with obesity [10], there are broader questions about the configuration of the field of undernutrition research, as selective simplification (for example in the production and use of international growth references) and complication (as argued by Scheffler et al. [1]). When seeking to make the field more complicated, the importance of genetics in human stature should not be ignored. While just over 12% of variance can be explained by 396 common gene loci [13], around 45% of variance can be explained by just over 300,000 common single nucleotide polymorphisms. The genetic contribution to a further 35% variation in stature is presently impossible to detect because the effects of several hundreds of thousands of additional SNPs is too small to be statistically significant [14]. Larger studies and future big data analyses are likely to show that the genetics of stature is greater than is possible to detect at present. Discussion around the environmental

effects (including those of nutrition and infection) on the development of human stature should acknowledge that the proportion of total variation that it can explain lies between 20 and 55% of variation that cannot presently be attributed to genetics. Of equal importance will be understanding the environmental factors that influence gene expression associated with growth and development—thus far, such factors have been mostly nutritional and immunological in nature. Such understanding could have profound impact on how growth faltering is understood, and on policies associated with the alleviation of undernutrition.

In their response letter to Tanjung et al. [2], Hermanussen et al. [4] fail to see any good reason why the World Health Organisation (WHO) Reference 2007 should be valid for Southeast Asian populations, given that it is a reconstruction of the 1977 United States (US) National Center for Health Statistics/WHO growth reference. As one among several scientists involved in the United Nations University/WHO/Food and Agriculture Organisation led International Growth Standard for Children and Adolescents Project (IGSCAP) of the mid-2000s [15], I observed the process and can offer two possible explanations for this. The aim of IGSCAP was to develop a new more globally-relevant set of growth references. Collecting new and appropriate population-based data, at a time when genomics was developing powerful ways of understanding human health through population biology, offered a way to potentially resolve the generations-old issues that are considered in the article by Scheffer et al. [1] and the published discussion that has followed it. One possible reason why IGSCAP did not go forward was the unresolved debate, over which nations and populations therein would be best represented in this new survey. Another is that it was too expensive. The cost of development of such new international growth references with big data capabilities was placed at significantly over 20 million US dollars in 2007 terms, too great for any agency or set of agencies to commit to at that time.

Forms of evidence that are acceptable for science, policy and intervention vary [11, 16]. The simplification of undernutrition, by the use of growth stunting as one proxy for it, serves many purposes and is productive for disciplines that have it as an object of interest—medicine, public health, epidemiology, education, economics and policy-making among them. The productive simplification—that growth stunting is an adequate proxy for malnutrition—is good enough for its many purposes for now, at least until big data approaches might shed further light on this issue.

Compliance with ethical standards

Conflict of interest The author has no conflicts of interest.

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