We are developing a more detailed appreciation of the variability in health potential with which we enter the world. Outcomes that once may have been viewed as serendipitous or pre-determined are now being examined with regard to aetiology and opportunities for primary prevention or mitigation. For example, we know that the poor carry a higher burden of chronic disease than the rich, and that subtle differences apparent at birth mark our developmental trajectories for life.1 If the latter is more than coincidence, we may expect some relationship between maternal deprivation and fetal growth to birth. Further, if we could then identify the point of departure in fetal development between social groups, we may be able to identify a critical period for framing interventions to reduce health inequalities in adulthood.

However, this task becomes quite challenging on several fronts. The first is that fetal growth is but a proxy for underlying processes. While major defects at birth and failure to survive to birth can often tell us something definitive about antecedent events, subtle variations in anthropometry are often difficult to relate to underlying metabolic processes or historical exposures, thereby eroding our analytic ability and raising questions of residual confounding. A second major question is the choice of indicator for disadvantage, as the precise mechanisms for any relationship between deprivation and fetal growth may vary accordingly. A third difficulty is that apparent effects at one time point may in fact be part of pre-established and long unfolding of developmental potential. Finally, there are limitations in the resolution to any instrumentation, no matter the degree of their contemporary relative sophistication, which means that we tend to focus on what we have in front of us, rather than what might be there from a theoretical perspective.
These rather complex matters are at the heart of a recent paper in this journal by Silva et al., in which they relate fetal growth by ultrasound to maternal education. They chose maternal education as it had previously been shown elsewhere to offer the greatest contribution from social indicators to birth anthropometry. There was no particular theoretical justification. At first glance, this analysis may appear a puzzling exercise, as education is extraordinarily broad in definition and potential effects on health-related factors, either as a mediating variable or due to multiple potential confounders. The analysis observed differences in fetal growth of the head, abdomen and femur from mid-gestation across levels of maternal education. How this might occur is good question. Let us first consider education as a global defining state.

Formal education is accumulated across 10–25 years of life, and is not merely the acquisition of knowledge or literacy, but the development of a ‘literacy of ideas’ that are internalized, forming the basis for interpreting and acting upon the world, and engaging with others. Through its application, education becomes embodied in what has been called habitus. This rather phenomenological use of habitus is distinct from the medical use of habitus used to describe physique, although the two coincide to inform our understanding of how, for example, ‘lifestyle’ factors may give rise to obesity.

As part of our habitus, education plays an important role in establishing our perceived ‘life choices’ and ‘life chances’, and our responses to them. From an epidemiological perspective, habitus as a ‘way of being’ plays an important mediating role during our trajectory across the life course, potentially giving rise to self-perpetuating social inequalities in health. Of course, Silva et al. controlled for the confounding effects of financial hardship, smoking and various other potential confounding factors and still found an effect. What is left may be residual confounding due to factors such as diet, which was not assessed, but there may also be other upstream factors to consider. Who gets an education anyway? Is this a form of familial habitus? Perhaps another option is that education represents a form of transgenerational material and social ‘capital’, reflected in a nurturing environment for maternal growth, and subsequent capacity for supporting fetal growth. We know that maternal stature and body composition correlate with fetal length and head size. Testing this was undertaken by modelling maternal height and education together. What does this mean? The positive effect of maternal height is interpreted as suggesting that women raised in circumstances conducive to becoming tall are also more likely to get an education—and not that women with an education get taller in pregnancy. This may reflect historical family circumstances not adequately captured in the present study—there is, for example, no data on familial wealth. A family effect therefore makes sense socially, and is also biologically plausible—either cumulatively, or due to a distant event that is reflected in more than one generation. It is not commonly appreciated that when a grandmother (F0) is pregnant with her daughter (F1), the fetal ovaries of F1 contain the oocytes that will become the F2 granddaughter. Hence, grandmaternal well-being can have a direct and independent effect on the developmental potential of both her daughter and grand daughter. In this context, unravelling the proximal and distal effects of maternal education on fetal growth are problematic. The interplay of the social and biological across generations is exquisitely complex, with the Silva et al. paper touching on some of the potential complexities.

Finally, I wish to express a note of caution when interpreting fetal growth data. The precision of ultrasound in determining a point in departure in fetal growth trajectory, and by implication, a critical period for intervention is highly problematic. The errors in resolution by ultrasound are tiny, but variability in growth rate in very early life is well below that discernable by ultrasound. There may well be departures in place by the time they reach the resolution limits of ultrasound and that apparent increases in differences are simply the continuation of an established trajectory. We also know that factors can alter growth tempo immediately post-fertilization, and even pre-fertilization as substances such as super-ovulation drugs appear to alter oocyte factors related to fetal growth to birth resulting in outcomes, such as lower birth weight for children born after in vitro fertilization, or the number of cells in the 3-day embryo correlates with birth weight. That these effects become apparent in later pregnancy may therefore lead us to ineffective strategies for intervention as the critical period for the setting of growth trajectory may have elapsed. Possibly worse, inducing late fetal growth may induce disproportion to placental growth, thereby placing the rapidly growing fetus at increased risk of exceeding nutrient supply with adverse consequences for the fetus, mother or both.

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References