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An Inter-generational Perspective on Social Inequality in Health and Life Opportunities: The Maternal Capital Model

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Introduction

The twentieth century saw increasing bifurcation in the academic study of human variability and its association with social behaviour. On the one hand, biological approaches were profoundly influenced by progress in molecular biology, emphasising the genetic basis of phenotypic variability. This led to interest in the heritability of behavioural traits, including the specific mechanisms through which genes may influence behaviour. Partly in opposition to this approach, social scientists developed a very different perspective, focusing on the sensitivity of behaviour to living conditions in order to understand how the organisation of society impacts biological phenotype. When we come to the study of social inequality, whether we are interested in health outcomes, life opportunities or wealth, these two approaches offer starkly different perspectives. In this chapter, we aim to provide a holistic perspective shedding new light on how social inequality can propagate trans-generational effects that are not the product of genotype.

From the late nineteenth century, eugenicists had argued that societies would become healthier if the 'weaker' members (e.g. those judged to have

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mental or physical illness) were 'selected out' by a winnowing process, thus improving the 'genetic stock' of the remaining members (Pearson 1912; Pearson and Lee 1903). Eugenics itself became increasingly unacceptable following the Second World War, though sterilisation programmes to prevent reproduction on several grounds remained widespread in subsequent decades, and are still evident in a few countries (Dikötter 1998). Nonetheless, the notion that a substantial component of behavioural variability has a genetic basis remains supported by twin and family studies, and the new science of genomics has made possible the search for individual alleles associated with variability in behavioural traits such as addiction, aggression, intelligence and attention span. On this basis, those at the top or bottom of social hierarchies might in theory be there in part because of their genotype. Some argue in favour of this, claiming support for the hypothesis that social rank in animals may be shaped by genetic differences in personality traits such as 'anxiety, agonistic behaviour, motivational processes and… behavioural vigor' (van der Kooij and Sandi 2015). With respect to humans, a combination of environmentally and genetically transmitted personality traits has been proposed to underlie inter-generational correlations in economic position (Bowles and Gintis 2001). Others dispute this approach, arguing that the association of genes with personality traits is too weak to contribute substantially to the perpetuation of socio-economic status across generations (Holtzman 2002).

More generally, social scientists typically reject the notion that social gradients in health and life opportunities derive from genotype. Any notion that individuals are 'captives' of their genetic constitution contradicts hundreds of years of philosophical thought emphasising free will, human rights and democratic governance. Rather, if there is social inequality in health and capabilities, it is primarily because humans have generated it through the structure and functioning of their societies. Taking this view to its extreme, we are born equal, and inequality is imposed by social institutions and practices through the lifespan.

The idea that social rank is assigned within the life-course was already apparent in early medieval thought, as for example in this thirteenth-century chess allegory:

> The world resembles a chessboard which is checkered white and black, the colour showing the two conditions of life and death, or praise and blame. The chess-[pieces] are [people] of this world who have a common birth, occupy different stations, and hold different titles in this life, who contend together, and finally have a common fate which levels all ranks. The King often lies under the other pieces in the bag. (Murray 1913)

If humans are characterised by a 'common birth'—equal standing at the start of life—then social policies must be key to resolving inequalities in health and opportunity, and constraining the negative effects of social stratification. Many different social policies duly attempt to promote equality, targeting different aspects of development or living conditions. Such efforts are directed at public health to reduce inequalities in ill-health, at social institutions in order to equalise access to infrastructure and support, and at schooling in order to equalise the distribution of knowledge, skills and training. The overarching aim is to equalise access to society's benefits, to benefit society at large. If such policies were successful, then the penalties of inequality would diminish, and should ultimately be less evident in biological phenotype and capabilities (Wilkinson and Pickett 2009; Marmot 2000, 2005).

Undoubtedly, the effects of social hierarchy extend back to physiology and morphology. The auxologist (scientist of human growth) James Tanner explicitly expressed this through his argument that the physical growth of children could act as an objective mirror of the level of equality characterising any society:

> If you want to measure the classlessness of a society, and you are not interested in rhetoric but in actual conditions and facts, then looking at the growth of children … is perhaps the best way. (Tanner 1990)

More unequal societies show steeper social gradients in children's growth (Eveleth and Tanner 1976), and even in relatively wealthy countries such as the UK, these gradients are reducing very slowly over time (Kuh et al. 1991). Conversely, in more egalitarian Scandinavian countries, the social gradient in height has progressively narrowed across recent decades, though it has not vanished entirely (Meyer and Selmer 1999; Peck and Vagero 1987). The weight of Tanner's proposal lies in the fact that height is no 'neutral' outcome; rather it is a very powerful marker of health status and human capital, as discussed in more detail below. Poor growth in children and short adult stature are associated with poor educational attainment and increased morbidity and mortality (Victora et al. 2008). Secular trends in height among poorer groups are therefore indicative of underlying trends towards societal equality in health and opportunity.

Unfortunately, however, the analogy provided by the chess game is somewhat simplistic. Though the medieval author referred to the chess pieces being 'levelled in status' at the end of the game, no such levelling actually occurs. At the beginning of every new game of chess, pawns remain pawns, and kings remain kings. Each piece has its allotted social role, and however many times

the game is played, indicative of successive generations, high and low ranks propagate themselves over time. It is clear that social hierarchy also persists across generations in human societies, but the underlying reasons have remained poorly understood. Despite much discussion of social mobility, the inter-generational transmission of social rank is powerful, with recent analyses suggesting that the heritability of wealth is even stronger than that in height (Clark and Cummings 2014, 2015).

While rejecting 'genetic determinist' accounts of inequality, however, social scientists have themselves proposed theoretical models of its inter-generational transmission that remain deterministic despite not referencing genes. In the mid-twentieth century, for example, one school of thought considered that poverty might replicate itself through cultural transmission. Based on interviews with families from Mexico and Puerto Rico, Oscar Lewis argued that impoverished communities in Latin American societies were characterised by a unique culture that was transmitted across generations (Lewis 1959, 1966a, b). He considered some elements of this culture to be self-defeating, giving rise to disorganisation, resignation and apathy: these characteristics then contributed to the perpetuation of poverty.

> The culture of poverty is not only an adaptation to a set of objective conditions of the larger society. Once it comes into existence it tends to perpetuate itself from generation to generation because of its effect on the children. By the time slum children are age six or seven they have usually absorbed the basic values and attitudes of their subculture and are not psychologically geared to take full advantage of changing conditions or increased opportunities which may occur in their lifetime. (Lewis 1966b)

Later, responding to criticism of his approach, he downplayed the role of culture in perpetuating poverty (Lewis 1969). Nevertheless, the idea proved attractive to policymakers, particularly in the US, and stimulated heated debate over whether the poor were architects or victims of their misfortune (Valentine 1971). The 'culture of poverty' concept contributed to a 'war on poverty' where social programmes sought 'to correct the social, occupational and physical deficits of people born and raised to a life of poverty' (Gladwin 1967). When these programmes, which made little effort to change structural factors, did not succeed, the intractability of the 'culture of poverty' was duly invoked as explanation. Apparently, its inter-generational cultural basis had simply made poverty 'ineradicable' (Seligman 1968).

We need to move beyond such deterministic approaches, in order to improve understanding of how social inequality can arise through

environmental stresses and yet be persistent in the face of efforts to change it. As evidence from 'genome-wide association' studies accumulates, it is no longer possible to deny that genetic factors contribute to variability in key markers of inequality such as health and educational attainment. Nevertheless, these studies typically explain only a small minority of the variance, and there is abundant and compelling evidence that living conditions, lifestyle and the broader structural environment strongly shape health and schooling outcomes. The primary risk factors for chronic degenerative diseases such as stroke, hypertension, type II diabetes and cardiovascular disease are diet, obesity and physical inactivity level. Social gradients in the risk of these diseases can thus be attributed to underlying inequalities in living conditions and access to health care (Wells 2016). Likewise, the component of education that most powerfully expresses social inequality is whether a child is in school at all. Clearly, none of these outcomes can be said to have a primary genetic basis. But in that case, why do these inequalities persist across generations?

Recently, there has been increasing recognition that the inter-generational persistence of social inequality involves mechanisms of plasticity, rather than genomic transmission. This represents something of a 'middle ground' between gene-based and cultural models of variability, and it forms the focus of this chapter. Biological plasticity refers to the capacity of phenotypes to respond to diverse environmental stimuli and stresses. One approach to this middle ground is to focus on how genes are *expressed* according to prevailing ecological conditions, while further acknowledging that experience in early life generates long-lasting imprints on DNA expression (Petronis 2010; Borghol et al. 2012). This focus on 'epigenetic' mechanisms may however lose sight of more fundamental explanatory approaches, which are our priority here.

The aim of this chapter is to elucidate *how* social inequality perpetuates biological effects across generations, and to identify how we can benefit from this understanding with the aim of reducing social gradients in health, education and life opportunities. We will see that despite the role of plasticity, chronic exposure to adversity can induce a cumulative phenotypic condition that may take several generations fully to reverse. In other words, biological penalties may be hard to resolve and yet this does not mean that they are inevitable. This approach goes beyond previous consideration of how social stresses lead to biological 'embedding' or 'embodying' (Krieger 2001; Hertzman and Boyce 2010), by placing unique emphasis on the mediating role of maternal phenotype.

Developmental Plasticity

At a proximate level, the inter-generational propagation of social inequality arises through developmental plasticity, a mode of response to environmental stimuli that operates during early life and generates long-term impact on phenotypes. Although a number of different environmental stresses are important, nutrition merits particular attention.

In the 1960s, classic studies showed that the effect of under-nutrition on rats depended on the timing of the insult. If the animal was underfed directly after birth, it would never fully resolve the deficit in body size, and would remain small in adult life. If the insult occurred several weeks after birth, however, growth would only slow temporarily, and as soon the nutritional constraint was lifted, rapid weight gain ensued, restoring the animal to its original growth trajectory (McCance 1962). This indicated 'critical periods' in growth, and subsequent work has shown that this concept applies not only to body size, but also to a host of specific tissues and organs, including the brain with implications for behaviour (Smart 1986; Smart 1991; Petry et al. 1997; Davison and Dobbing 1968; Davison and Dobbing 1966).

Amongst the underlying reasons is that the nature of growth changes fundamentally through early development. Early growth comprises an increase in cell number through cell division, known as *hyperplasia*, whereas later growth comprises increases in cell size, known as *hypertrophy* (Bogin 1999). The great majority of hyperplasic growth occurs during foetal life and early infancy. In the rat, for example, organ and tissue growth is entirely due to cell proliferation until approximately 17 days after birth, with minimal change in cell size. Detailed studies on rats have found that if nutritional constraint is imposed during this period, the animals develop lighter organs with fewer cells in them. If the nutritional insult is delayed until later, the animals can regain their organ masses and cell numbers after re-feeding (Winick and Noble 1965, 1966; Enesco and LeBlond 1962).

This means that the mammalian body cannot reverse major structural 'decisions' already locked into physiology through foetal or infant growth patterns. Although not all of these studies have been conducted in humans, for obvious ethical reasons, the profile of human growth is essentially similar to that observed in rats. Consequently, exposure to adversity in early life, whether this derives from nutritional or psychosocial stress, induces long-term changes in physiological structure and function, impacting both the body and the brain. The other side of this coin is that long-term improvements in nutrition during early life potentially represent a key opportunity for improving health and human capital. The importance of such early 'critical windows' of devel-

opment is now recognised through the slogan 'the first thousand days of life', and is researched under the Developmental Origins of Health and Adult Disease (DOHaD) hypothesis (Gluckman et al. 2008).

As soon as we consider in more detail what environmental factors matter during the first 1000 days, however, it is impossible to ignore the importance of maternal phenotype (Wells 2010). Critical windows of physiological sensitivity occur primarily during the periods of gestation and lactation, during which all stimuli experienced by the developing organism are transduced by maternal phenotype. Although not all women breast-feed in contemporary populations, we have argued from an evolutionary perspective that windows of plasticity evolved in concert with maternal nutritional care, in other words gestation and lactation (Wells 2003, 2014). In turn, many components of maternal phenotype that impact offspring development are powerfully shaped by maternal social rank. On this basis, we can see something profound: an inter-generational process in which (a) maternal social rank influences development of the offspring, and (b) developmental experience of the offspring shapes its social rank in later life (Fig. 24.1).

To some extent, the influence of maternal metabolism on the developmental trajectory of the offspring represents a form of protection against external ecological insults (Wells 2003, 2016). For example, even when the

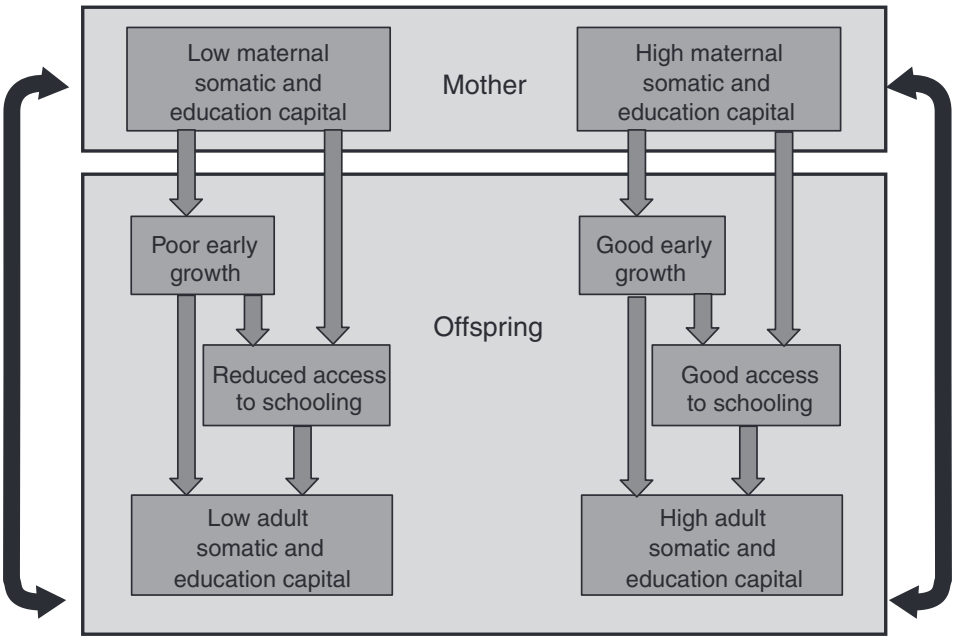

Fig. 24.1 Schematic diagram showing an inter-generational cycle between the level of maternal capital and the acquisition of somatic and educational capital in the offspring. Those receiving poor nutrition and less education in early life embody these traits in adulthood and transmit them to the next generation. Those receiving high investment can transfer more to their own offspring

mother herself is exposed to famine and experiences drastic reductions in energy supply, growth of the foetus is only relatively mildly affected (Stein et al. 2004). Maternal physiology functions to buffer the immature foetus from adversity, and this buffering is of particular value because it shields the period of hyperplasic growth, when the organs and tissues are most sensitive to disruptive ecological stresses (Wells 2003, 2014). However, the very protection that the mother can offer her offspring can become its own constraint when the mother herself has experienced chronic exposure to adversity. We have referred to this as a 'metabolic ghetto' (Wells 2010, 2016), where the developing foetus cannot escape exposure to the mother's long-term experience of deprivation. As in a castle that is besieged using its own defensive walls, layers of maternal physiological protection can lock in their own stresses so that they manifest to the foetus or infant as a developmental constraint (Wells 2016).

Maternal Effects

Small maternal size, poor nutritional status at the time of conception, poor dietary intake during pregnancy and exposure to infectious diseases have all been shown to impact development of the foetus and infant, in different ways (Ozaltin et al. 2010; Dominguez-Salas et al. 2012; Stein et al. 2004; Guyatt and Snow 2004; Ticconi et al. 2003). Maternal psychosocial stress can also generate adverse effects on foetal growth, through the medium of maternal levels of cortisol, a physiological marker of stress (Entringer et al. 2008a, b, 2009, 2011).

In turn, variability in foetal and infant patterns is now well established to represent a key component of variability risk of chronic diseases in later life (Leon et al. 1996; Hales et al. 1991; Barker et al. 1989; Li et al. 2015). These diseases now comprise the primary burden of ill-health in high-income countries, and despite a persistent burden of infectious disease in low- and middle-income countries, chronic diseases are also the main source of premature mortality and morbidity in these countries too (Lozano et al. 2012). In England during 2009–2011, for example, living in an area with the highest deprivation (measured in deciles) was associated with seven and nine years shorter life expectancy for women and men, respectively, compared with those in the least-deprived areas (Office for National Statistics 2014). Equivalent differences in healthy life expectancy were twice as large. Elevated burdens of long-term ill-health and premature mortality are thus key aspects of social inequality.

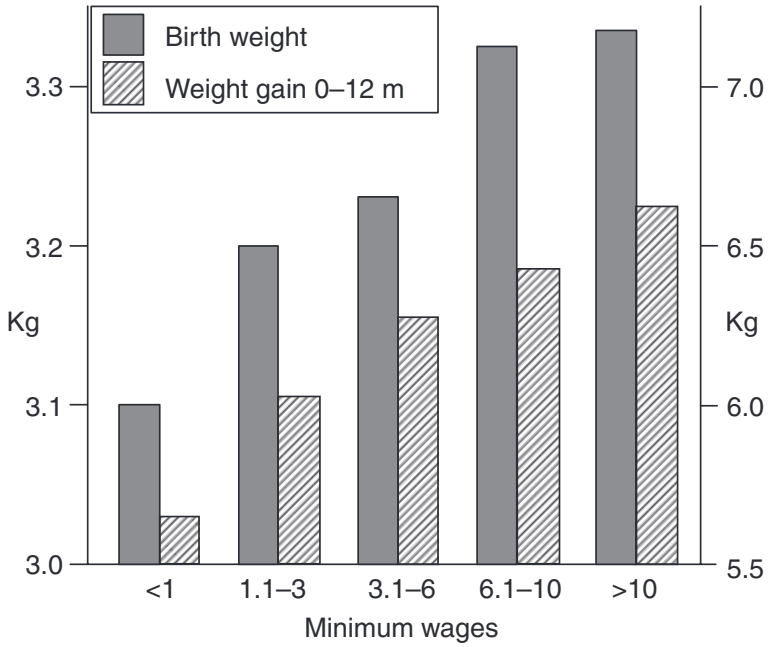

Fig. 24.2 Association of birth weight and infant weight gain with family income, assessed in 'minimum wages', in the 1982 Birth Cohort from Pelotas, Brazil. Data from Victora et al. (1987)

Early growth variability feeds directly into such health inequalities. In most populations, birth weight and infant weight gain show profound social gradients, such as those illustrated in Fig. 24.2 for the population of Pelotas in southern Brazil (Victora et al. 1987). Even where low birth weight is followed by compensatory catch-up growth (Ong et al. 2000), the recovery of deficits in size occurs at the expense of long-term health (Metcalfe and Monaghan 2001). Catch-up growth is an independent risk factor for adult chronic disease, and exacerbates the effects of low birth weight (Eriksson et al. 1999).

Such inter-generational associations help understand social gradients in adult chronic disease risk, but to fully explain patterns of variability we also need to take into account the effect of social inequality in later life. Just as the risk of low birth weight is greater in those of low social rank, in high-income populations the risk of obesity is also greater in those of lower socio-economic position (Giskes et al. 2008; Sobal and Stunkard 1989; McLaren 2007). This means that the poor, in affluent societies, have two independent risk factors for chronic disease: low birth weight imposed by their mother's deprivation, and an unhealthy lifestyle and physiology imposed by their own experience of deprivation in later life. However, in low- and middle-income countries, risk of obesity is greater in those of higher socio-economic position (Subramanian et al. 2011; Neuman et al. 2011). In these populations, therefore, chronic diseases remain clustered amongst more wealthy groups, whilst those of low

social economic position are most vulnerable to under-nutrition and infectious disease.

We have termed our model of developmental plasticity the 'maternal capital' hypothesis (Wells 2010, 2012a) to emphasise that the offspring initially calibrates its developmental trajectory to its allocation of maternal investment. Building on Kaplan's concept of 'embodied capital' (Kaplan et al. 2003), maternal capital is defined as 'any aspect of maternal phenotype, whether somatic or behavioural, which enables differential investment in offspring' (Wells 2010). As we have seen above, those receiving less 'capital transfer' in early life through the medium of maternal nutrition are more susceptible to ill-health, though the relevant diseases depend on the level of economic development.

When maternal physiology provides a stable metabolic signal for the offspring, that signal carries the imprint of maternal rank (Wells 2010). The offspring is exposed to the sum total of maternal capital, which has accumulated through the mother's life-course. That such signals elicit a response by the offspring is shown by correlations between many stable components of maternal phenotype and those of the offspring. For example, maternal birth weight, adult height and leg length, and body composition at the time of conception all show correlations with the offspring's birth weight (Wells 2016).

Energy stores are undoubtedly important, but there are many other components of maternal capital, including nutrients (vitamins, minerals, macronutrients), social capital, and indices of completed growth such as stature or pelvic dimensions. Beyond nutritional resources themselves, another crucial component of maternal capital comprises her capacity for metabolic homeostasis. In contemporary populations, mothers with hypertension or gestational diabetes expose their offspring to perturbed metabolism (Wells 2007). Such effects can continue in infancy, when the breast milk of diabetic mothers has high sugar levels (Plagemann et al. 2002). Just like maternal starvation, such metabolic dysfunction can be considered a depletion of maternal capital, and it can impose long-term metabolic penalties on the offspring. Now the 'walls' lock in too much fuel, rather than too little, and the offspring again experiences metabolic penalties (Wells 2016).

Each offspring continues to process ecological cues throughout development, resulting in a chain of 'decision nodes' that collectively constitute its life history strategy (Wells 2012b). These 'decisions' are generally not expected to involve conscious thought, and instead are generated through physiological or subconscious mechanisms shaped by natural selection. Even where behaviours (e.g. sexual activity) do involve conscious decision-making, it is possible

that such decisions may simply provide post-hoc rationalisation—more a consequence of behaviour than cause. For example, similar behaviours in other species would not be assumed to involve any conscious deliberation.

Each of these decisions reflects the expression of its genotype under the influence of the quality of the environment. The earliest decisions occur through calibration to maternal phenotype (Wells 2010), whereas later decisions are elicited directly by the environment. For example, studies of migrants from Bangladesh to the UK show that reproductive physiology retains plasticity throughout childhood and adolescence (Nunez-De La Mora et al. 2007, 2008). Since later decisions are shaped by earlier ones, however, the whole chain maintains the initial maternal imprint (Wells 2010).

Figure 24.3 shows how offspring tailor their life history strategy to the magnitude of investment they receive, using data from a study of young South Asian women living in the UK (Wells et al. 2016). A lower level of nutritional

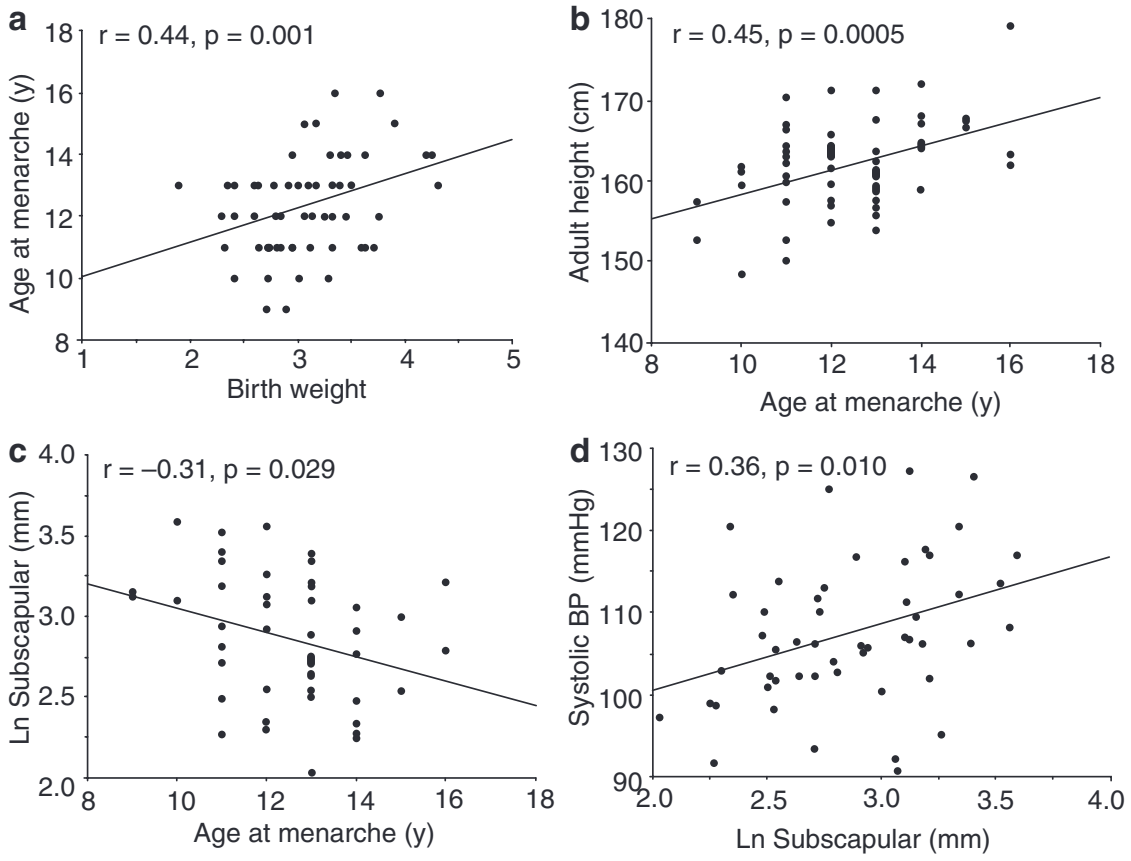

Fig. 24.3 Associations of maternal investment (proxied by birth size) with maturation rate and adult phenotype in young healthy South Asian women living in the UK. (**a**) Birth weight is inversely associated with age at menarche. (**b**) Earlier menarche is associated with lower adult stature. (**c**) Earlier menarche is associated with higher adult subscapular skinfold. (**d**) Subscapular skinfold is positively associated with systolic blood pressure. Reproduced with permission from Wells et al. (2016)

investment, indicated by lower birth weight, was associated with an accelerated pattern of development, as indicated by earlier puberty. However, this accelerated maturation reduced the total period of growth, and was associated with shorter adult stature and with elevated adiposity. Finally, high levels of body fat were associated with higher blood pressure. This study is important because it shows that daughters who received less investment from their mothers adopted a 'fast life history', which prioritised the acquisition of energy stores for reproduction at the cost of investment in somatic growth and health. Other studies show that adult women may propagate these traits to the next generation, perpetuating the cycle (Ong et al. 2007).

Maternal Capital and Education Outcomes

It is clear therefore that variability in growth patterns in early life contributes to health inequalities in later life, and recently the same approach has been used to explore variability in education outcomes. As with chronic disease risk, educational attainment has been associated with patterns of growth during the period before children are of school-going age.

In a large cohort study of 8362 children in Brazil, Guatemala, India, the Philippines and South Africa, for example, lower birth weight, slower linear growth and lower relative weight at 2 years were all independently associated with an increased risk of not completing secondary school (Martorell et al. 2010; Adair et al. 2013). Similar findings have been reported from other studies in Ghana and Tanzania (Beasley et al. 2000; Fentiman et al. 2001; The Partnership for Child Development 1999). Although faster growth at later points in the life-course may also benefit cognitive performance (Horta et al. 2009; Cheung 2006), there are substantial 'trade-offs', for faster weight gain from mid-childhood onwards is associated with increased risk of chronic diseases (Victora et al. 2008). In other words, growth is most beneficial for *both* health and education if it occurs during the early critical windows that are under the influence of maternal metabolism. Breast-feeding is an important part of this process, because in addition to providing optimal nutrition, including many nutrients critical for brain development (Isaacs et al. 2010; Anderson et al. 1999), it also reduces the likelihood of acquiring infectious diseases, which can stunt growth and cognitive development (Walker 2010).

While these observational studies link poor growth in early life with lower educational attainment, they do not provide robust evidence of the causal linkage. One exception however is a longitudinal study in Guatemala, which found that in comparison to children who received a different nutritional supplement, those who had received a high protein nutritional intervention

between birth and 36 months completed 1.2 more years of schooling and performed better on cognitive tests (Maluccio et al. 2009).

Collectively, these studies provide insights into the mechanisms through which early life nutrition is associated with educational outcomes. Growth retardation, indicating a lack of nutrients at the cellular level in early life, has been associated with poorer brain and neurological development (Martorell et al. 2010). For example, a study of Scottish school children broadly showed that the shorter the duration of foetal development, indicated by gestational age at birth, the higher their risk of cognitive impairment (MacKay et al. 2010). Whether and how this cognitive 'deficit' is then directly associated with educational outcomes require more research, including data on school-based factors. However, some studies imply such associations. In their first year of school, under-nourished children from Mexico performed poorly on cognitive tests and participated less in the classroom with 38% repeating a grade (Chavez et al. 2000). A longitudinal study in Guatemala also suggested that psycho-social factors such as lower levels of stimulation and social interaction may delay the age at school entry for stunted children, thereby increasing their risk of repeating or dropping out of school (Brown and Pollitt 1996).

Although growth in early life is associated with educational success, a key aspect missing from these studies is the influence of maternal phenotype during the period of children's early growth. Further research is also required on how maternal nutritional status directly predicts children's educational outcomes (Walker et al. 2011). An analysis of a longitudinal biomedical study of 838 children since birth in Dhanusha, Nepal, adopted a more comprehensive approach, testing the independent associations of different components of maternal somatic and educational capital and family economic capital with children's educational attainment (Marphatia et al. 2016b). The results showed that children aged 8.5 years were at a higher risk of completing fewer years of schooling if their mothers had lower levels of capital defined by lack of education and poor nutritional status, especially anaemia, and that these associations held after adjusting for broader components of family capital.

Figure 24.4 shows independent associations of different components of maternal phenotype with poor educational attainment at age 8.5 years, expressed as odds ratios for categories of maternal capital. In this model, the association of maternal low haemoglobin (anaemia) with children's educational attainment was mediated by poor rates of growth between birth and 2 years of age, adjusting for size at birth. Overall, maternal lack of education, a proxy of the social capital offered to children throughout their life-course, had the strongest magnitude of association, followed by different maternal biological markers for the two sexes, high land ownership for boys and rural location for girls.

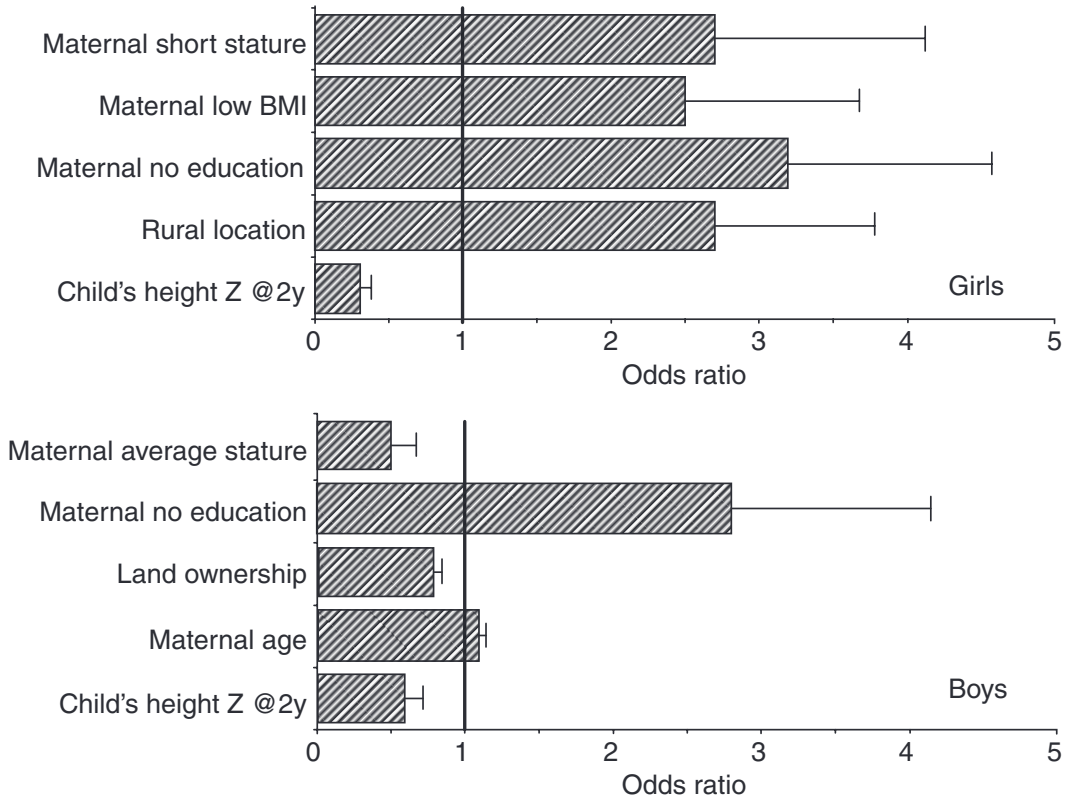

Fig. 24.4 Odds ratios for the risk of Nepali children completing less than 3 years education by ~8.5 years, for a variety of parental or family risk factors, or conditional growth of the children themselves. Based on data of Marphatia et al. (2016b)

These results suggest that, as with health inequalities, educational inequalities are shaped early in the life-course, and are strongly associated with maternal phenotype. Given such trans-generational perpetuation of disadvantage (Schell 1997), school-based efforts may arrive too late to support their participation in education.

Crucially, low birth weight is often followed by some form of catch-up growth, which as we saw above accelerates the pace of maturation. In turn, this may trigger a social response—identifying fast-maturing girls as ready for early marriage. This is important because studies find that adolescent girls who marry at an earlier age are more likely to drop out of school, experience early and repeated pregnancies and have children of low birth weight, who are under-nourished and also more likely to complete less schooling (Fall et al. 2015; Godha et al. 2013; Santhya 2011).

Here, therefore, we see the full trans-generational cycle of disadvantage, in which the pattern of development experience in one generation shapes that in the next generation. None of these associations need derive from genotype, indicating that interventions could aim to improve outcomes if conducted

over lengthy periods. Greater focus on building the educational and nutritional capital of girls and women is likely to be mutually beneficial for both mothers and children, as discussed further below.

Societal-Level Gender Inequality and Children's Outcomes

Although many components of maternal capital reflect the individual circumstances of particular women, we can also consider how the organisation of society influences maternal capital at the population level. One way in which this can be done is by assessing women's status in society relative to men, for example, using the Gender Inequality Index (GII), a measure of women's reproductive health and their participation in education, the labour market and political representation relative to men. Recent ecological studies have used this index to investigate associations between societal gender inequality and child malnutrition and survival.

For example, across 138 countries societal-level gender inequality was associated with neonatal, infant and childhood mortality rates (Brinda et al. 2015). Another ecological analysis investigated whether two countries with similar national wealth, but with different status of women, have different levels of low birth weight, child malnutrition and survival across 96 countries (Marphatia et al. 2016a). Figure 24.5 shows a linear, dose-response between these factors with an increase in gender inequality associated with increases in adverse child outcomes.

These associations were still present even after controlling for markers of economic growth, measured by gross domestic product (GDP). This suggests that the current paradigm of addressing child malnutrition and mortality through promoting GDP is unlikely to be successful unless and until women's status in society also improves (Marphatia et al. 2016a).

Conversely, simulations based on statistical models of these data suggest that reducing gender inequality would have major reductions in child survival and malnutrition globally. These analyses suggest that efforts to promote women's ability to participate, at an equal level, with men in society are likely to have substantial benefits for children's health and survival, especially in low- and middle-income countries. The value of such composite indices is that they identify specific capabilities and opportunities of women that interventions could target in order to improve both their well-being and the health of their children.

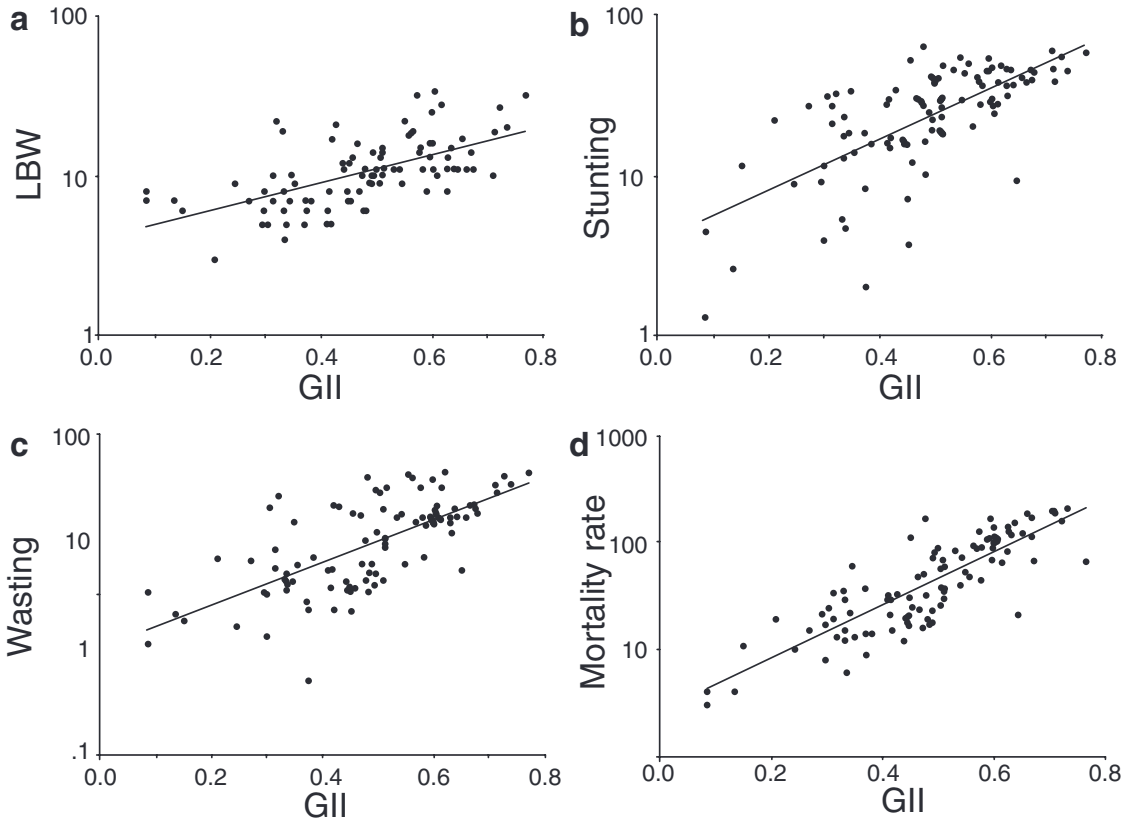

Fig. 24.5 Associations of the Gender Inequality Index (GII) and the prevalence of (**a**) low birth weight (LBW), (**b**) stunting, (**c**) wasting and (**d**) child mortality rate in 96 countries (2 missing data points for LBW). Reproduced with permission from Marphatia et al. (2016a)

Maternal Capital as an Opportunity for Intervention

If maternal capital generates such profound long-term impacts on the developmental trajectory of offspring, then it appears as a key opportunity for interventions intended to break the 'cycle of disadvantage'. Of particular interest, we have demonstrated that each of health and education is associated with growth patterns in early life, which in turn demonstrates the importance of both maternal nutrition and educational attainment.

Paradoxically, efforts to promote maternal nutrition during pregnancy and infancy have had mixed success. A number of supplementary nutrition programmes targeted at pregnant women achieved relatively modest increments in birth weight, averaging around 40 g, although larger increments occurred in particularly under-nourished groups (Kramer 1993). However, one challenge is that most of these programmes were only initiated midway through pregnancy, and thus missed the period during which the placenta develops,

along with the earliest periods of organogenesis. A unique study that supplemented women across two pregnancies achieved a substantially greater increment in birth weight of 150 g (Villar and Rivera 1988). Studies that have supplemented during infancy have also tended to have greater benefits (Conlisk et al. 2004; Kinra et al. 2008), though the challenge remains that the ideal route is to supplement the mother so that the infant benefits through breast-feeding.

These interventions also do not address the critical importance of simultaneously increasing maternal education, which studies find improves both maternal and child well-being. Incorporating training on literacy through women's groups often used in non-formal education programmes may thus enhance the success of these interventions. On their own, these initiatives also have their limitations, the trade-offs of which may be better understood by adopting a holistic understanding of both biological and social factors as highlighted in this chapter (Marphatia and Moussie 2013).

Using maternal phenotype as a medium through which to operationalise interventions targeting the offspring might be considered unethical, on account of treating the mother as a 'passive vehicle' without taking her own needs or identity into account. We believe this perspective is unhelpful, because as we have discussed above, the health of the offspring is fundamentally associated with the health of the mother. In the vast majority of situations, the interventions that are crucial for transmitting benefits to the next generation (e.g. promoting healthy maternal metabolism) will also benefit women in their own right. Our data on the harmful consequences of societal gender inequality highlights the importance of improving the circumstances of women.

How successful might this approach be? Returning to our analogy of the mediaeval chess game, how many games (generations) would it take for public health interventions to dissolve the inequalities in health that currently characterise pawn, queen and king? The available evidence suggests that each generation can accumulate phenotypic improvements, but that a number of generations are required in order to shift phenotype substantially (Wells 2012a). Whilst this may make progress seem hard to achieve, we should also look at the long-term benefits: when mothers have accumulated substantial capital, they are resistant to short-term stresses and can buffer their offspring during early critical windows. This is the 'natural advantage' already enjoyed by those at the top of hierarchies, and it could be shared across the population if societies organized themselves to achieve this aim, as the evidence from gender-equal and egalitarian societies attests.

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