

‘POOR CHILDREN GROW INTO POOR ADULTS’: HARMFUL MECHANISMS OR OVER-DETERMINISTIC THEORY?

SHAHIN YAQUB*
Sussex University, UK

Abstract: Does childhood poverty lead to adult poverty? Evidence shows childhood is a sensitive period for developing cognition, physical vitality and personality. This is traceable to specific behavioural and biological mechanisms. However such science could easily drive over-deterministic views about how childhood affects later life. The paper therefore discusses how damage from childhood poverty can—at least sometimes and partially—be resisted or reversed, both during childhood and in adulthood. As people reach biological maturity, alterations to their developmental trajectories rely increasingly on alterations in behavioural relationships. Opportunities remain vital throughout life for sustained socioeconomic attainment. Copyright © 2002 John Wiley & Sons, Ltd.

1 INTRODUCTION

A widely accepted assumption is that childhood experiences set the stage for lifetime experiences. Childhood is seen as foundational for individual development, both physiologically and psychologically, and is taken to define lifetime socioeconomic potential. Thus: ‘... capabilities that adults enjoy are deeply conditional on their experiences as children’ (Sen, 1999) and ‘there has been a rediscovery in the policy world of the role of early childhood as a lifelong determinant... because issues began to be expressed in a credible vocabulary for modern society, the vocabulary of science... to give credibility to notions long held as common sense’ (Hertzman, 2000). In such perspectives resources to tackle child poverty are justified not only morally, but also as sound investment against future poverty and as propellant for economic development. Apparently traditional policy tensions are eased because child interventions powerfully avert poverty and fit comfortably within the human capital framework of economic growth (Deutsch, 1998; IADB, 1999).

The paper probes empirical justifications for this role accorded to childhood. Section 2 presents supporting evidence. Cognition, physical vitality and personality have strong

*Correspondence to: S. Yaqub, Poverty Research Unit, School of African and Asian Studies, Sussex University, Falmer, Brighton, BN1 9QN, UK. E-mail: s.yaqub@sussex.ac.uk

basis in childhood. And yet, children resist and bounce back from harmful experiences, indicating 'resilience' and 'plasticity' in their functionings. To properly capture this, arguments about childhood foundations of poverty should ground themselves not only in the social sciences but also in the natural sciences of human functionings, and should look critically at empirical literature in each by relating one to the other. 'People are biological organisms, after all, and our activities and thoughts can be understood only by situating us properly with a brain in a body in an eventful world abounding with objects and people' (Dawson and Fischer, 1994). We also find people from disadvantaged backgrounds succeed throughout life. Section 3 contextualizes such ideas with evidence on socio-economic opportunities and individual agency. These are easily under-emphasized by uncritical acceptance of research on childhood 'predictors' of poverty. Socioeconomic attainments require a sound basis *at each life stage*.

Section 4 concludes the paper. Our understanding of the roles of the many stages of childhood in accounting for lifetime achievement needs to be more differentiated both across functionings and age. This will suggest comparisons of costs to obtain *poverty reversals* through adult interventions versus *poverty avoidance* through child interventions. These two are contrasting antipoverty approaches, since partly child interventions are prospective and aim to support 'resilience' against harm, whilst adult interventions are retrospective and rely on 'plasticity' in already failed functionings. Careful empirical research is required because unsubstantiated theory building could mean childhood poverty becomes the stage for something equally harmful, if genetic over-determinism is replaced with childhood over-determinism as political reason to avoid antipoverty interventions.

2 CHILDHOOD FOUNDATIONS: SENSITIVE PERIODS, RESILIENCE AND PLASTICITY

Important developmental foundations are laid early in life. But how early, under what influence, and how permanently? In the following discussion, the term perinatal is reserved for the first postnatal week, neonatal to the first month, and infancy to the first year. The prenatal (or gestational or intrauterine) phase is divided into three sequential trimesters. Key motivating concepts are that: (i) different aspects of human development each have one or more 'sensitive periods' when development is most receptive to influence (Bornstein, 1989); (ii) even during sensitive periods, certain individual and environmental characteristics (perhaps including genes) give 'resilience' against damage from poverty, at least partially—so thresholds into developmental failure may differ between people (Engle *et al.*, 1996; Grotberg, 1995); (iii) even if damage occurs during sensitive periods, there may be 'plasticity' (or reversibility) in some and permanence in others.

Neurons are brain cells that store and transmit information, and by six months of pregnancy, all the neurons a person ever has are produced (Berk, 1996). By age 3 years the brain is 70 per cent of its adult size, and by 5 years nearly 90 per cent (Berk, 1996). Protein-energy undernutrition (especially marasmus, rather than kwashiorkor) impairs cognition, and if occurring prenatally or in infancy retards brain development and correlates with later functional deficits (Scrimshaw, 1998). In Chile, Ivanovic *et al.* (2000) found that low birth weight infants, as compared to normal infants of similar socioeconomic status, had at age 18 years significantly lower intelligence quotient, scholastic achievement, head circumference, and physical brain development (as indicated by magnetic resonance imaging). Even in affluent countries low birth weight is negatively

associated with childhood, adolescent and young-adult attainments in cognition, education, and labour market outcomes (Bartley *et al.*, 1994; Pollitt and Mueller, 1982). Controlled animal experiments suggest that some (but not all) aspects of brain development damaged by early malnutrition is irreversible (e.g. myelination—insulation of neural fibres—which speeds information transmission) (Levitsky and Strupp, 1995).

Brain growth spurts coincide with functional growth spurts, and this continues into young adulthood. Unfortunately 'almost all relevant studies have investigated either brain growth or behaviour, not both' (Fischer and Rose, 1994). Regions of the brain are functionally specialized, mature at different ages (possibly into young adulthood) and have differently timed growth spurts (in terms of structure and electrical activity). This needs to be related to progress in specific human functions to identify neurodevelopmental foundations. Yet tests of functional development tend to be crude for the complexity they aim to measure (for example, consider the many elements of cognition). Moreover, the role of different (macro- and micro-) nutrients may vary by timing. For example, if iron-deficiency occurs *after age 5 years*, iron supplementation can reverse deficits in learning ability and memory, *but not attention* (Pollitt *et al.*, 1986; Rao and Georgieff, 2000). Reversal is impossible if iron-deficiency occurs in infancy, because then iron assists in permanent structural changes in the brain (Rao and Georgieff, 2000). Prenatal iodine deficiency, especially by the second trimester, produces permanent neurological damage (Scrimshaw, 1998).

Neurons are networked via synapses, allowing motor and mental functions. Initially synapses are formed randomly at a phenomenal postnatal rate, but then are selectively pruned, so that perinatal and mature adult brains contain *fewer* synapses than infant brains. This synaptic overproduction followed by elimination is argued to have functional correlates (Huttenlocher, 1994). First, it is greatest for humans compared with other species, and greatest for brain areas involved in complex tasks. Synaptic elimination in the brain's visual cortex is completed by age 10 years, whereas in the brain's frontal lobe, responsible for thought and consciousness, it continues into adolescence. Secondly synaptic pruning is influenced by environmental stimuli. A practical example is using a patch to cover a *good* eye to prevent visual loss in a temporarily squinting eye due to selective synaptic elimination (from the asymmetric stimuli). Importantly, the patch is ineffective after age 7 years (Huttenlocher, 1994). From this, a general principle is suspected in which functional plasticity coincides with ages when synaptic connections exceed adult values (Huttenlocher, 1994). Remarkable recovery from even major brain lesions is possible for motor functions (especially in the first few months after birth) and language (until age 8 years), as remaining normal brain regions take over (Huttenlocher, 1994). In emotion and behaviour, post-trauma recovery seems greater for older rather than younger children (Fuemmeler *et al.*, 2002).

Obviously it cannot be assumed that physical brain development is identical to functional development. The intelligence quotient (IQ) is a contentious measure of ability because of confounding factors, like access to information, cultural definitions of 'basic knowledge', and test-taking experience (Sternberg *et al.*, 2002; Fagan and Holland, 2002). Intelligence quotients measured at different points in childhood do change (i.e. intraper-sonally). In one study five patterns of longitudinal change in IQ were identified between ages 30 months and 17 years, plus a set of cases with idiosyncratic profiles (Wohlwill, 1980). After age 6 years IQ scores become more stable and predictive of later scores (Siegler and Richards, 1982; Feinstein, 2000). For example, IQ scores between ages 5–7 years show a correlation to IQ scores between 17–18 years of 0.86 (Wohlwill, 1980). Absences from school and poor environment lead to IQ declines (Ceci, 1999; Gorman and

Pollitt, 1996). Time and effort spent on active learning are important determinants of cognitive performance (Bruer, 1998; Aksoy and Link, 2000; Kagan, 2000). Undernutrition and morbidity, especially in combination, retard attention spans, motivation, memory, and school attendance (Grantham-McGregor *et al.*, 1994; Del Rosso and Marek, 1996; Glewwe *et al.*, 1999; Alderman *et al.*, 2001; Berkman *et al.*, 2002). Proper development of visual and other sensory functions are vital for academic persistence and achievement (e.g. Gomes-Neto *et al.*, 1997).

Various agents (called teratogens) are toxic to the foetus, including caffeine, aspirin, alcohol, nicotine, drugs, and sexually and non-sexually transmitted viruses and bacteria. These cause death, physical malformations and growth retardation. At low levels of exposure, teratogens are believed to cause developmental deficits. Jacobson and Jacobson (2000) found prenatal alcohol exposure correlated with deficits at age 8 years in attention and information processing speed (but not memory), and prenatal exposure to polychlorinated biphenyls (PCBs) correlated with deficits at age 11 years in speech and reading (but not processing speed). Low-quality habitats of poor people likely increase exposure to a broad range of teratogens, but research is surprisingly lacking. For example, the long-term developmental impact of possible teratogens from domestic biomass fuels in developing countries remains unknown. Such fuels are linked to low birthweight and stillbirths (Smith, 2000) and to acute respiratory infections (Ezzati and Kammen, 2002), the prime cause of mortality and morbidity in under 5 year olds (Williams *et al.*, 2002).

Major bodily abnormalities are less likely after three-months of gestation, although development of sensory organs remains sensitive throughout pregnancy. Growth in outer body dimensions continues to age 20 years, and periods of greatest sensitivity are during gestation and infancy (Beaton *et al.*, 1990). Maternal nutrition in the first trimester is critical to avoid miscarriage, and in the last trimester for foetal growth (Norton, 1994). Stature is sensitive to the adolescent growth spurt. Modest catch-up is possible if environments are improved in early years (Rutter *et al.*, 1999).

The 'small but healthy hypothesis' casts doubt on the functional consequences of body size, arguing that people with low height-for-age, but normal weight-for-height, suffer no impairment (Seckler, 1984; Messer, 1986). Various measures of body size are correlated to immunocompetence, physical work capacity, and reproductive health (Payne, 1992; Perez-Escamilla and Pollitt, 1992; Martorell, 1996). Such correlations are clearest amongst those extremely undernourished (Osmani, 1992). At issue is an unresolved controversy over the extent to which body size reduction represents an adaptive response, for example to lower energy requirements: 'moderate stunting without wasting is neither a cause nor a marker of current or *individual* deprivation. It is a marker, though not a cause, of previous *population* deprivation' (Payne and Lipton, 1994). For example, maximum oxygen intake (VO₂max), a common indicator of physical work capacity, is lower for stunted adults partly because of less body mass. This disadvantage is partially offset because physical labouring seldom requires more than 40 per cent VO₂max, often involves movement of one's own body, and biological and ergonomic adaptations may increase stamina (Payne and Lipton, 1994).

Prenatal adversity heightens morbidity *throughout life*—referred to as the 'foetal origins of disease', and observed in many populations (Barker, 1994). People of foetal age in the Dutch famine of 1944–45 were as adults more vulnerable to diabetes, high blood pressure and coronary heart disease (Lumey *et al.*, 1993; Roseboom *et al.*, 2001). Such diseases of affluence are correlated to low weights at birth and infancy. This is thought to depend on intrauterine and infant biological programming of body tissues and systems for

an anticipated—but unrealized—life of scarcity (Scrimshaw, 1997). Similar effects are found for infectious diseases, suggesting immunocompetence impairment. In rural Gambia, people born in the hungry season were ten times more likely to die in young adulthood, mostly from infectious diseases (Moore *et al.*, 1999). Animal experiments reveal foetal programming of immune function (Prentice, 1998).

Sensitive periods in behaviour-traits, self-esteem, temperament and personality, are more difficult to ascertain, and may well be culture specific. Language acquisition is important for subsequent learning and psychological development (Walker *et al.*, 1994). Adults continually improve language skills and master considerable grammar and vocabulary in second languages. Nevertheless a sensitive period in early childhood is suspected for language development—as suggested by research on children deprived of stimulation, and research on second language acquisition by migrants of different ages (Berk, 1996). Studies in industrial countries suggest self-esteem undergoes a radical period of change after first contact with peers (say at kindergarten). High self-esteem is argued to be associated with 'mastery-oriented attributions', in which success is attributed to ability and failure to effort or environment (in contrast, low self-esteem is associated with 'learned helplessness'). Attribution retraining to correct for this can occur in middle childhood, but becomes progressively harder (Berk, 1996). Emotional temperament seems to show modest longitudinal continuity. Some studies have found scores on activity level, rhythmicity (regularity of body functions), attention span, irritability, sociability, and shyness are correlated between infancy and childhood, and in some studies, into adulthood (Berk, 1996).

The most pressing results on plasticity in human development are longitudinal studies on those suffering extreme deprivation and abuse. Adoptions from Romanian and Russian orphanages after communism provided an unusually large study sample. Extremely developmentally impaired children showed major reversals in failures in functionings after adoptions in affluent countries. Cognitive and anthropometric status of those adopted *after* age six months improved (mostly to within normal ranges), but at age 4 years, lagged behind those adopted *before* age six months (Rutter *et al.*, 1999). The latter were indistinguishable from a control group of UK adoptees. Similar results were obtained for adoptions into Canada and the USA (Johnson, 2000).

More generally, those restored to their natural parents after periods in institutional care show *lower* intellectual, scholastic and emotional outcomes than those adopted. Clarke and Clarke (1999) argue that this reveals the importance of 'chain effects' in the way improved environmental factors affect the path of attainments. Such ideas suggest the basis for saying '... whatever stresses an individual may have encountered in early years, he or she need not be forever more at the mercy of the past... children's resilience must be acknowledged every bit as much as their vulnerability...' Schaffer (1992). The simple model of an environmental input leading to a functional output in people is wrong, not least because people's developmental experiences are mediated via many other people (contrary to an earlier matricentric assumption), inputs are experienced together and/or in particular temporal sequences, and people have their own individuality (Schaffer, 1992; Pilling, 1992).

3 LIFETIME FOUNDATIONS: OPPORTUNITIES, AGENCY AND TURNING POINTS

Three types of correlations aim to provide measures of lifetime socioeconomic opportunities: (i) intergenerational, (ii) sibling, and (iii) intrapersonal correlations. In these, a

person's attainments in income, class, education, health, and employment have been shown to correlate to attainments of their parents, siblings, and themselves at a prior time *respectively*. Welfare correlations between people that shared similar socioeconomic backgrounds (i.e. family members) suggest that socioeconomic background influences lifetime attainments. Sibling correlations are stronger tests to the extent that natural siblings share genes, culture, community and household characteristics, and unobservable factors like parenting, etc.—although birth order, sex and birth spacing may condition these. All these add weight to views that childhood experiences determine adult poverty.

The correlations mentioned can be interpreted as 'large'. Earnings advantages to offspring with well-off parents, implied by contemporary intergenerational correlations in Britain, for example, are comparable to advantages gained through tertiary education (O'Neill and Sweetman, 1995). Moreover such correlations may be resilient to quite fundamental changes to the economy.¹ The point here, however, is that in these correlations a lot of welfare variance remains unexplained, and regression to the mean exists. Some of this is probably due to errors in obtaining measures of lifetime attainments from data that in most countries is considerably shorter than actual lifetimes (thereby requiring statistical adjustments when estimating correlations) (Solon, 1989). Of all countries with available estimates, *R*-square statistics in regressions of offspring attainments against parental attainments never exceed 0.50 and elasticities—the percentage change in offspring attainment for percentage change in parental attainment—never exceed 0.75 (depending on country, period, statistical method, and whether the attainments indicator is earnings, income, wealth, or socioeconomic status)—and these figures can be as low as 0.10 (reviewed in Yaqub, 2000). Clearly socioeconomic attainments can differ considerably between parents and offspring, and even between siblings.

The intrapersonal correlations reveal another interesting fact, in that 'windows of opportunities' for lifetime success may have age-related openings. Countries with sufficiently longitudinal data show a strikingly narrow age-range for lifetime economic success (perhaps until mid-30 years). Geweke and Keane (2000) found that in the USA, at age 30 years, low earnings strongly predicted low earnings persistence *throughout life*. They estimated the 'present value at age 25 years' of lifetime earnings was halved if a person was in the poorest quintile at age 30 years, than if not. Björklund (1993) found that in Sweden only after age 30 years did single year income inequality converge on lifetime income inequality: 'income up to around 30 years of age accounts for the marked discrepancy between the dispersion of annual and lifetime income. From around 35 years of age up to 65 years the correlations between annual and lifetime income are high and the dispersions are more or less of the same magnitude'. Table 1 shows that in Denmark, Finland, France, Germany, Italy, Sweden, UK, and USA, earnings mobility was lower at older ages. Two measures are reported (see table notes), and in both a value of one indicates no mobility.

Such data could imply an early-career sorting process characterized by instability, in which subsequently, people follow more closely their 'true' (and more stable) lifetime income paths. Lifetime paths may represent some continuation of processes initiated in

¹Preliminary estimates for my DPhil. show stability in intergenerational earnings and incomes correlations in the British National Child Development Study (NCDS) and British Cohort Study (BCS). NCDS tracks individuals from birth in 1958 to age 41 years, and BCS from birth in 1970 to age 29 years. Interestingly transitions into labour markets occurred 'before' (NCDS) and 'after' (BCS) Britain's New Right monetarist revival, as implemented by governments of Margaret Thatcher after 1979.

Table 1. Earnings mobility by age-group, 1986–91

	Pearson correlation coeff.				Shorrocks index using Theil0			
	<25 yrs	25–34	35–49	50–64	<25 yrs	25–34	35–49	50–64
Denmark	0.23	0.56	0.72	0.78	0.75	0.85	0.91	0.94
Finland	0.12	0.33	0.46	0.48				
France	0.29	0.64	0.80	0.83	0.71	0.85	0.91	0.92
Germany	0.39	0.73	0.87	0.89	0.52	0.88	0.93	0.93
Italy	0.39	0.70	0.83	0.84	0.70	0.84	0.91	0.90
Sweden	0.65	0.48	0.70	0.82				
UK	0.48	0.65	0.75	0.76	0.81	0.85	0.91	0.91
USA	0.52	0.63	0.73	0.70	0.73	0.85	0.91	0.91

Note: Ages refer to 1986. The lower the Shorrocks or Pearson, the greater the mobility. The Shorrocks (1978) index is the following ratio: inequality of earnings aggregated over all years, divided by a weighted sum of inequality in each year (the weights are set equal to the share of yearly earnings in aggregate earnings). The Shorrocks was calculated using the Theil0 inequality index. The Pearson is the simple correlation coefficient between the two years. Björklund (1993) reports similar results for Sweden using the Shorrocks index (not shown because of different age groupings).

Source: OECD (1996); OECD (1997).

childhood, if they are dependent on irreversible characteristics determined in childhood. However the earlier discussion left many empirical issues open on sensitivity, resilience and reversibility with regard to the way that different developmental inputs generate particular functionings at each age. Importantly Geweke and Keane (2000) estimated that 'about 60 per cent of the variation of lifetime earnings [in the USA] not explained by education and race is attributable to permanent individual characteristics that are unobserved and uncorrelated with education, age and race'. A better interpretation than a simple sorting process may be that the determinants of sustained individual development may shift over the lifetime, as people follow shifting opportunities. For example, Hauser *et al.* (2000) found in the USA that for men the correlation between years of schooling and occupational status at first job was 0.77, but by age 54 years, the correlation fell to 0.54—for women the correlations respectively were 0.50 and 0.37.

This view suggests it important to examine, in a similar vein to childhood experiences, persistence (or otherwise) of developmental outcomes in adolescence and young-adulthood. Hobcraft (1998) found in the UK a wide range of adolescent outcomes were correlated to outcomes at age 33 years in incomes, education, employment, housing, and police contact. Burgess *et al.* (1999) found in the UK that only for low-skilled workers did early career unemployment experience have adverse effects on subsequent employment. Burgess and Propper (1998) found that, amongst males in the USA, consumption of hard drugs and violent behaviour in adolescence (age 16–22 years) predicted lower employment, earnings levels and earnings growth over the subsequent decade. Adolescent consumption of alcohol and soft drugs had no such effects.

Hobcraft and Kiernan (1999) found that in the UK, for females, the birth of a first child before the age of 23 was predictive of adverse outcomes at age 33 in terms of lone-parenthood, welfare-dependency, educational attainment, income, and physical and emotional malaise—even after controlling for a number of indicators for childhood poverty. 'For young women in particular, it is probable that early parenthood is directly implicated in the genesis of adverse outcomes later in life, through limiting opportunities and choices' (Hobcraft and Kiernan, 1999). Buvinic (1998) reviewed studies on the effects of adolescent childbearing in Barbados, Chile, Guatemala, and Mexico, and found later

marriage chances to be unaffected, subsequent fertility to be higher in Barbados and Guatemala but not in Chile and Mexico, and lower socioeconomic status and earnings.

However three further studies caution against deterministic conclusions. Weed *et al.* (2000) found great diversity in attainments of adolescent mothers five years postpartum in the USA, with large proportions completing at least secondary schooling and having good psychosocial status. Hotz *et al.* (1999) 'constructed' a control group from adolescents experiencing miscarriages (presumed randomly distributed), and found that a sizeable portion of negative effects of adolescent parenthood in the USA was attributable instead to their pre-existing poverty and low socioeconomic status (i.e. delaying childbearing would not have greatly enhanced later attainments). 'Teen mothers may actually achieve higher levels of earnings over their adult lives than if they had postponed motherhood. While teenage childbearing does seem to increase public aid expenditures immediately after the birth of their first child, this 'negative' consequence of teenage childbearing is not a permanent one, in that teen mothers use less public aid in their late-20s as their earnings rise and their children age' (Hotz *et al.*, 1999). This could be an example of public safety-nets working. Similarly in Jamaica, Degazon-Johnson (2001) showed beneficial effects 10 years later (compared with a control group) from a programme targeted on teen-mothers to promote school completion, marketable skills and parenting knowledge.

4 CONCLUSION

This paper investigates whether childhood experiences set the course of lifetime achievements. Empirical literature is presented showing developmental sensitive periods, when certain types of damage to functionings can—*but not always*—result from childhood poverty, and some—*but not all*—damage may be permanent. The caveats indicate, respectively, resilience and plasticity in human functionings. The role of genes in determining human accomplishment suggests to some that antipoverty interventions are futile. Without careful empirical assessment of behavioural *and* biological mechanisms that affect resilience and plasticity in human functionings, research into the lifetime impact of childhood poverty may simply add weight to such 'over-determined' theory.

Much of the research presented relates to sensitivity, resilience and plasticity in physiological development (neurology, anthropometry). The ultimate interest is of course in sensitivity, resilience and plasticity in functional development (cognition, vitality, personality). The incongruence between physiological development and functional development is of prime policy interest. The incongruence first moderates claims about child poverty determining lifetime achievements, and secondly defines possibilities for reversing poverty via interventions amongst adult populations. The former recognizes that childhood physiological damage does not equate to damaged functionality (for example, if brain physiology imperfectly determines trajectories of cognitive growth, because all is not biological). The latter recognizes that beyond ages when physiological maturity is reached (which is in fact staggered in its various dimensions), adult antipoverty interventions rely for success on at least the possibility of continued functional development, albeit perhaps at rates varying across people. As people age, alterations to their developmental trajectories rely increasingly on alterations in behavioural relationships.

Over the past two decades, antipoverty interventions have become increasingly targeted. These are triggered by poverty itself, require 'proof of poverty' for participation, and therefore, make a fundamental assumption of reversibility of failures in functionings.

There are good theoretical reasons, adapting Amartya Sen's concepts of functionings and capabilities, to think that more explicit consideration of the timing of antipoverty interventions would improve impact (Yaquub, 2001). Antipoverty interventions may be premature or delayed in people's lives, affecting how a person takes advantage of resources to convert them into functionings. Antipoverty interventions should be prioritized to when the worst damage from poverty can be avoided, when the most gains in functionings can be obtained, and when the fastest poverty-reversals occur. These are, respectively, damage, size and speed criteria for timetabling antipoverty, and offer a policy-useful way of interpreting research on lifetime implications of childhood poverty.

ACKNOWLEDGEMENTS

This is part of my doctorate titled 'Born poor, stay poor?' supervised by Michael Lipton and Robert Eastwood at Sussex University. My doctorate is funded by the UK's Economic and Social Research Council under award R00429734678. Marzia Fontana, Caroline Harper and Rachel Marcus kindly helped with ideas.

REFERENCES

- Aksoy T, Link CR. 2000. A panel analysis of student mathematics achievement in the US in the 1990s: does increasing the amount of time in learning activities affect math achievement? *Economics of Education Review* **19**(3): 261–277.
- Alderman H, Hoddinott J, Kinsey B. 2001. *Long Term Consequences of Early Childhood Malnutrition*. World Bank: Washington, DC.
- Barker DJP. 1994. *Mothers, Babies and Disease in Later Life*. British Medical Journal Publishing Group: London.
- Bartley M, Power C, Blane D, Davey Smith G, Shipley M. 1994. Birth weight and later socioeconomic disadvantage: evidence from the 1958 British cohort study. *British Medical Journal* **309**(6967): 1475–1478.
- Beaton G, Kelly A, Kevany J, Martorell R, Mason J. 1990. Appropriate uses of anthropometric indices in children. Administrative Committee on Coordination/Subcommittee on Nutrition ACC/SCN, Nutrition Policy Discussion Paper 7. United Nations: Geneva.
- Berk LE. 1996. *Infants, Children and Adolescents* (2nd edn). Allyn and Bacon: Boston.
- Berkman DS, Lescano AG, Gilman RH, Lopez SL, Black MM. 2002. Effects of stunting, diarrhoeal disease and parasitic infection during infancy on cognition in late childhood: a follow-up study. *Lancet* **359**(9306): 564–571.
- Björklund A. 1993. A comparison between actual distributions of annual and lifetime income: Sweden 1951–89. *Review of Income and Wealth* **39**(4): 377–386.
- Bornstein MH. 1989. Sensitive periods in development: structural characteristics and causal interpretations. *Psychological Bulletin* **105**(2): 179–197.
- Bruer JT. 1998. Brain science, brain fiction. *Educational Leadership* **56**(3): 14–19.
- Burgess SM, Propper C. 1998. Early health related behaviours and their impact on later life chances: evidence from the US. CASE Paper 6. London School of Economics: London.
- Burgess S, Propper C, Rees H, Shearer A. 1999. The class of 81: the effects of early career unemployment on subsequent unemployment experiences. CASE Paper 32. London School of Economics: London.

- Buvinic M. 1998. Costs of adolescent childbearing: a review of evidence. *Studies in Family Planning* **29**(2): 201–209.
- Ceci SJ. 1999. Schooling and intelligence. In *The Nature–Nurture Debate*, Ceci SJ, Williams WM (eds). Blackwell: Oxford; 168–175.
- Clarke A, Clarke A. 1999. Early experience and the life path. In *The Nature–Nurture Debate*, Ceci SJ, Williams WM (eds). Blackwell: Oxford; 136–146.
- Dawson G, Fischer KW. 1994. *Human Behavior and the Developing Brain*. Guildford Press: London.
- Degazon-Johnson R. 2001. A new door opened. A tracer study of the teenage mothers project, Jamaica. Practice and Reflections Paper 13. Bernard van Leer Foundation: Netherlands.
- Del Rosso JM, Marek T. 1996. *Class Action: Improving School Performance in the Developing World Through Better Health and Nutrition*. World Bank: Washington, DC.
- Deutsch R. 1998. *How Early Childhood Interventions Can Reduce Inequality: An Overview of Recent Findings*. Inter-American Development Bank: Washington, DC.
- Engle PL, Castle S, Menon P. 1996. Child development: vulnerability and resilience. FCND Discussion Paper 12. IFPRI: Washington, DC.
- Ezzati M, Kammen DM. 2002. Evaluating the health benefits of transitions in household energy technologies in Kenya. *Energy Policy* **30**(10): 815–826.
- Fagan JF, Holland CR. 2002. Equal opportunity and racial differences in IQ. *Intelligence* **30**(4): 361–387.
- Feinstein L. 2000. Pre-school educational inequality? British children in the, 1970 cohort. Economics Department Discussion Paper 56. University of Sussex: Brighton.
- Fischer KW, Rose SP. 1994. Dynamic development of coordination of components in brain and behaviour. In *Human Behavior and the Developing Brain*, Dawson G and Fischer KW (eds). Guildford Press: London; 3–66.
- Fuemmeler BF, Elkinb TD, Mullins LL. 2002. Survivors of childhood brain tumors: behavioral, emotional, and social adjustment. *Clinical Psychology Review* **22**(4): 547–585.
- Geweke J, Keane M. 2000. An empirical analysis of earnings dynamics among men in the PSID: 1968–1989. *Journal of Econometrics* **96**(2): 293–356.
- Glewwe P, Jacoby H, King E. 1999. Early childhood nutrition and academic achievement: a longitudinal analysis. FCND Working Paper 68. IFPRI: Washington, DC.
- Gomes-Neto JB, Hanushek EA, Leite RH, Frota-Bezzera RC. 1997. Health and schooling: evidence and policy implications for developing countries. *Economics of Education Review* **16**(3): 271–282.
- Gorman KS, Pollitt E. 1996. Does schooling buffer the effects of early risk? *Child Development* **67**(2): 314–326.
- Grantham-McGregor S, Powell C, Walker S, Chang S, Fletcher P. 1994. The long-term follow-up of severely malnourished children who participated in an intervention program. *Child Development* **65**(2): 428–439.
- Grotberg E. 1995. Guide to promoting resilience in children. Practice and Reflections Paper 8. Bernard van Leer Foundation: The Hague.
- Hauser RM, Warren JR, Huang M, Carter WY. 2000. Occupational status, education, and social mobility in the meritocracy. In *Meritocracy and Economic Inequality*, Arrow K, Bowles S, Durlauf S (eds). Princeton University Press: New Jersey; 179–229.
- Hertzman C. 2000. The case for an early childhood development strategy. *ISUMA* **1**(2): 11–18.
- Hobcraft J. 1998. Intergenerational and life-course transmission of social exclusion: influences of childhood poverty, family disruption, and contact with the police. CASE Paper 15. London School of Economics: London.

- Hobcraft J, Kiernan K. 1999. Childhood poverty, early motherhood and adult social exclusion. CASE Paper 28. London School of Economics: London.
- Hotz VJ, McElroy SW, Sanders SG. 1999. Teenage childbearing and its life cycle consequences: exploiting a natural experiment. Working Paper 7397. National Bureau of Economic Research: Cambridge, MA.
- Huttenlocher PR. 1994. Synaptogenesis in human cerebral cortex. In *Human Behavior and the Developing Brain*, Dawson G, Fischer KW (eds). Guildford Press: London; 137–152.
- IADB. 1999. *Breaking the Poverty Cycle*. Inter-American Development Bank: Washington, DC.
- Ivanovic DM, Leiva BP, Perez HT, Inzunza NB, Almagià AF, Toro TD, Urrutia MSC, Cervilla JO, Bosch EO. 2000. Long-term effects of severe undernutrition during the first year of life on brain development and learning in Chilean high-school graduates. *Nutrition* **16**(11/12): 1056–1063.
- Jacobson SW, Jacobson JL. 2000. Teratogenic insult and neurobehavioral function in infancy and childhood. In *Effects of Early Adversity on Neurobehavioral Development: Minnesota Symposia on Child Psychology 31*, Nelson CA (ed.). Lawrence Erlbaum: London; 61–112.
- Johnson DE. 2000. Medical and developmental sequelae of early childhood institutionalization in Eastern European adoptees. In *Effects of Early Adversity on Neurobehavioral Development: Minnesota Symposia on Child Psychology 31*, Nelson CA (ed.). Lawrence Erlbaum: London; 113–162.
- Kagan J. 2000. The brain may not be the answer. *Canadian Journal of Policy Research* **1**(2): 55–56.
- Levitsky DA, Strupp BJ. 1995. Malnutrition and the brain: changing concepts, changing concerns. *Journal of Nutrition* **125**(Suppl. 8): S2212–S2220.
- Lumey LH, Ravelli ACJ, Wiessing LG, Koppe JG, Treffers PE, Stein ZA. 1993. The Dutch famine birth cohort study: design, validation of exposure, and selected characteristics of subjects after 43 years of follow-up. *Paediatric and Perinatal Epidemiology* **7**(4): 354–367.
- Martorell R. 1996. Undernutrition in young children and its consequences on behavioral development, work capacity and reproductive health. *Conference on Early Child Development: Investing in the Future*, 8–9 April. Atlanta: Georgia.
- Messer E. 1986. The small but healthy hypothesis: historical, political and ecological influences on nutritional standards. *Human Ecology* **14**(1): 57–75.
- Moore SE, Cole TJ, Collinson AC, Poskitt EM, McGregor IA, Prentice AM. 1999. Prenatal or early postnatal events predict infectious deaths in young adulthood in rural Africa. *International Journal of Epidemiology* **28**(6): 1088–1095.
- Norton R. 1994. Maternal nutrition during pregnancy as it affects infant growth, development and health. *SCN News* Number 11, Administrative Committee on Coordination/Subcommittee on Nutrition ACC/SCN. United Nations: Geneva.
- O'Neill D, Sweetman O. 1995. The persistence of poverty in Britain: evidence from patterns of intergenerational mobility. *Economics Department Working Paper N61/10/95*. National University of Ireland: Maynooth.
- OECD. 1996. Earnings inequality, low-paid employment and earnings mobility. In *Employment Outlook 1996*. Organisation for Economic Cooperation and Development: Paris.
- OECD. 1997. Earnings mobility: taking a longer run view. In *Employment Outlook 1997*. Organisation for Economic Cooperation and Development: Paris.
- Osmani SR. 1992. On some controversies in the measurement of undernutrition. In *Nutrition and Poverty*, Osmani SR (ed.). WIDER Studies in Development Economics; University Press: Oxford; 49–96.
- Payne P. 1992. Assessing Undernutrition: the need for a reconceptualization. In *Nutrition and Poverty*, Osmani SR (ed.). WIDER Studies in Development Economics; University Press: Oxford; 121–164.

- Payne P, Lipton M. 1994. How Third World rural households adapt to dietary energy stress: the evidence and the issues. *Food Policy Review* 2. IFPRI: Washington, DC.
- Perez-Escamilla R, Pollitt E. 1992. Causes and consequences of intrauterine growth retardation in Latin America. *Bulletin of Pan American Health Organisation* 26(2): 128–147.
- Pilling D. 1992. Escaping from a bad start. In *Vulnerability and Resilience in Human Development*, Tizard B, Varma V (eds). Jessica Kingsley: London.
- Pollitt E, Mueller W. 1982. Relation of growth to cognition in a well-nourished preschool population. *Child Development* 53(5): 1157–1163.
- Pollitt E, Pollitt CS, Leibel RL, Viteri FE. 1986. Iron deficiency and behavioural development in infants and preschool children. *American Journal of Clinical Nutrition* 43(4): 555–565.
- Prentice AM. 1998. Early nutritional programming of human immunity. In *Annual Report 1998*. Nestle Foundation: Lausanne.
- Rao R, Georgieff MK. 2000. Early nutrition and brain development. In *Effects of Early Adversity on Neurobehavioral Development: Minnesota Symposia on Child Psychology 31*, Nelson CA (ed.). Lawrence Erlbaum: London; 1–30.
- Roseboom TJ, van der Meulen JHP, Ravelli ACJ, Osmond C, Barker DJP, Bleker OP. 2001. Effects of prenatal exposure to the Dutch famine on adult disease in later life: an overview. *Molecular and Cellular Endocrinology* 185(1–2): 93–98.
- Rutter M, et al. 1999. Developmental catch-up, and deficit, following adoption after severe global early privation. In *The Nature–Nurture Debate*, Ceci SJ, Williams WM (eds). Blackwell: Oxford; 108–134.
- Schaffer RH. 1992. Early experience and the parent–child relationship: genetic and environmental interactions as developmental determinants. In *Vulnerability and Resilience in Human Development*, Tizard B, Varma V (eds). Jessica Kingsley: London.
- Scrimshaw NS. 1997. The relation between fetal malnutrition and chronic disease in later life. *British Medical Journal* 315(7112): 825–826.
- Scrimshaw NS. 1998. Malnutrition, brain development, learning, and behaviour. *Nutrition Research* 18(2): 351–379.
- Seckler D. 1984. The ‘small but healthy?’ hypothesis: a reply to critics. *Economic and Political Weekly* 19(44) 3 November: 1886–1888.
- Sen AK. 1999. Investing in early childhood: its role in development. *Conference on Breaking the Poverty Cycle: Investing in Early Childhood*, 14 March 1999. Inter-American Development Bank: Washington, DC.
- Shorrocks AF. 1978. Income inequality and income mobility. *Journal of Economic Theory* 19(2): 376–393.
- Siegler RS, Richards DD. 1982. Development of intelligence. In *Handbook of Human Intelligence*, Sternberg RJ (ed.). University Press: Cambridge; 897–971.
- Smith KR. 2000. National burden of disease in India from indoor air pollution. *Proceedings of the National Academy of Sciences of USA* 97(24): 13286–13293.
- Solon G. 1989. Biases in the estimation of intergenerational earnings correlations. *Review of Economics and Statistics* 71(1): 172–174.
- Sternberg RJ, Grigorenko EL, Ngorosho D, Tantufuye E, Mbise A, Nokes C, Jukes M, Bundy DA. 2002. Assessing intellectual potential in rural Tanzanian school children. *Intelligence* 30(2): 141–162.
- Walker D, Greenwood C, Hart B, Carta J. 1994. Prediction of school outcomes based on early language production and socioeconomic factors. *Child Development* 65(2): 606–621.
- Weed K, Keogh D, Borkowski J. 2000. Predictors of resiliency in adolescent mothers. *Journal of Applied Developmental Psychology* 21(2): 207–231.

- Williams BG, Gouws E, Boschi-Pinto C, Bryce J, Dye C. 2002. Estimates of world-wide distribution of child deaths from acute respiratory infections. *Lancet Infectious Diseases* **2**(1): 25–32.
- Wohlwill JF. 1980. Cognitive development in childhood. In *Constancy and Change in Human Development*, Brim OG, Kagan J (eds). Harvard University Press: Massachusetts.
- Yaqub S. 2000. Intertemporal welfare dynamics: extent and causes. *Human Development Report Office Occasional Paper 70*. UNDP: New York [<http://hdr.undp.org>].
- Yaqub S. 2001. At what age does poverty damage most? Exploring a hypothesis about timetabling error in antipoverty. *Conference on Justice and Poverty: Examining Sen's Capability Approach*, 5–8 June, Von Hügel Institute, St Edmunds College, University of Cambridge [<http://www.st-edmunds.cam.ac.uk>].