A later version of this paper appeared in the Journal of International Development 14 (8), 2002

"Poor children grow into poor adults": harmful mechanisms or over-deterministic theory?

Aug 2002

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Abstract

This paper examines evidence on whether childhood poverty causes adult poverty. Childhood is recognised rightly as a sensitive period for developing cognition, physical vitality and personality. This is in part traceable to specific 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 reached biological maturity alterations to their developmental trajectories rely increasingly on alterations in behavioural relationships. Opportunities remain vital throughout life for sustained socioeconomic attainment. The subject of the paper is important in suggesting comparison of costs of *poverty reversals* through adult interventions versus *poverty avoidance* through child interventions.

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 p.4) 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, p.12). 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. Traditional policy tensions are eased apparently because child interventions can be claimed to powerfully avert poverty whilst fitting comfortably within the human capital framework of economic growth (e.g. IADB 1999; Deutsch 1998).

The paper probes empirical justifications for this role accorded to childhood. Section 2 presents supporting evidence. Cognition, physical vitality and personality have strong basis in childhood. 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, p.xiii). And yet, children resist and bounce back from harmful experiences, indicating 'resilience' and 'plasticity' in their functionings. Some people from disadvantaged backgrounds do succeed. Section 3 contextualises such ideas with evidence on socioeconomic opportunities and individual agency. These are easily under-emphasised by uncritical interpretation 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: 1/ different aspects of human development each have one or more 'sensitive periods' when development is most receptive to influence (Bornstein 1989); 2/ even during sensitive periods, certain characteristics of the individual (perhaps including genes) and his/her environment give 'resilience' against damage from poverty, at least partially – this implies that thresholds into developmental failure may differ between people to some extent (Engle et al. 1996; Grotberg 1995); 3/ even if damage occurs during sensitive periods, there may be 'plasticity' (or reversibility) in some areas, and permanence in others.

Neurons are brain cells that store and transmit information, and by the sixth month of prenatal gestation, all the neurons a person ever has are produced (Berk 1996). By age 3 years the brain is 70 percent of its adult size, and by 5 years nearly 90 percent (Berk 1996). Protein-energy malnutrition occurring prenatally, or in infancy for more than a month, retards physical brain development and correlates with later developmental deficits. In Chile, Ivanovic (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 behavior, not both" (Fischer and Rose 1994, p.55). Regions of the brain are functionally specialised, 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 especially in infancy, because then iron assists in permanent structural changes in the brain (Rao and Georgieff 2000).

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 to

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 (e.g. Sternberg et al. 2002; Fagan and Holland 2002). Intelligence quotients measured at different points in childhood do change (i.e. intrapersonally). 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 (Aksoy and Link 2000; Kagan 2000; Bruer 1998). Undernutrition and morbidity, especially in combination, retard attention spans, motivation, memory, and school attendance (Del Rosso and Marek 1996; Glewwe et al. 1999; Alderman et al. 2001; Grantham-McGregor et al. 1994; 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 (Berk 1996). 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, p.49). Maximum oxygen intake (VO_2max), 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 percent VO_2max, often involves movement of one's own body, and biological and ergonomic adaptations may extend stamina (Payne and Lipton 1994).

Prenatal adversity heightens morbidity *throughout life* – referred to as the 'foetal origins of disease' (Barker 1994). People of foetal age in the Dutch famine of 1944-5 were as adults more vulnerable to diabetes, high blood pressure and coronary heart disease (Lumey et al. 1993). Such diseases of affluence also are correlated to low birth weight and low infant weight, and are thought to have arisen because of intrauterine biological programming for an anticipated life of scarcity (Scrimshaw 1997). Similar effects are found for infectious diseases, suggesting foetal 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, and 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, p.40). 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: 1/ intergenerational, 2/ sibling, and 3/ intrapersonal correlations. In these, a person's attainments in incomes, class, education, health, and employment have been shown to correlate, *respectively*, to attainments of their parents, siblings, and themselves at a prior time (see Yaqub 2000 for a review). The strength of welfare correlations between people that shared similar socioeconomic backgrounds (i.e. family members) suggests that socioeconomic background strongly influence 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 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.[2] The point here, however, is that in these correlations a lot of

[2] Preliminary estimates for my DPhil. show stability in intergenerational earnings and

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. 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 economic success in life, up to around mid-30 years, after which intrapersonal economic mobility declines. Geweke and Keane (2000) found that in the USA, at age 30 years, low earnings strongly predicted low earnings persistence *throughout life*, controlling for race and education. Björklund (1993) found that in Sweden only after age 30 years did single year income inequality converge on lifetime income inequality. 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.

Table 1: Measures of earnings mobility by age-group

	Shorrocks for Theil0 after 6 yrs				Pearson correlation after 5 yrs			
	<25 yrs	25-34	35-49	50-64	<25 yrs	25-34	35-49	50-64
Denmark	0.75	0.85	0.91	0.94	0.23	0.56	0.72	0.78
Finland	Not reported				0.12	0.33	0.46	0.48
France	0.71	0.85	0.91	0.92	0.29	0.64	0.80	0.83
Germany	0.52	0.88	0.93	0.93	0.39	0.73	0.87	0.89
Italy	0.70	0.84	0.91	0.90	0.39	0.70	0.83	0.84
Sweden	Not reported				0.65	0.48	0.70	0.82
UK	0.81	0.85	0.91	0.91	0.48	0.65	0.75	0.76
USA	0.73	0.85	0.91	0.91	0.52	0.63	0.73	0.70

Source: OECD (1996), OECD (1997)

Note: The lower the Shorrocks or Pearson, the greater the mobility. The Shorrocks (1978) measure 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 over a six year accounting period. The Pearson used a five year

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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. Comparisons are interesting because 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. The intergenerational correlation in wealth at death in the USA was 0.59 around the mid-1700s and 0.71 around the mid-1800s, and in the UK was 0.53 around 1900 and 0.60 around 1950 (Behrman and Taubman 1985).

accounting period.

Such data could imply people experience a sorting process characterised by instability early in their careers, but subsequently, people follow more closely their 'true' lifetime income paths. Lifetime paths may represent some continuation of processes initiated 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. 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, p.209) 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, p.35). 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 possible 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, p.36). This could be an example of public safety-nets working. Similarly in Jamaica, Degazon-Johnson (2001) showed beneficial effects 10 years later (compared to a control group) from a programme targeted on teen-mothers to promote school completion, marketable skills and parenting knowledge.

5. 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* – of which may be permanent. The caveats indicate, respectively, resilience and plasticity in human functionings. Supposedly resilience and plasticity are related – in a complex and poorly understood way – to genetics, environment, and the interaction of the two.

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 not only moderates claims about child poverty determining lifetime achievements, but also defines possibilities for reversing poverty via interventions implemented amongst adult populations. The former recognises 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 recognises that beyond ages when physiological maturity is reached (which is in fact staggered in its various dimensions), adult antipoverty interventions rely for their success on the possibility of continued functional development, albeit perhaps at rates varying across people. As people age, alterations to their developmental trajectories therefore 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 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 (Yaqub 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 prioritised 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 way of interpreting research on lifetime implications of childhood poverty.

Acknowledgements

This work is part of my doctorate titled '*Born Poor, Stay Poor?*', supervised by Michael Lipton and Robert Eastwood, and funded by the UK's Economic and Social Research Council. Marzia Fontana, Caroline Harper and Rachel Marcus kindly helped with ideas.

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