INVESTING IN EARLY CHILDHOOD EDUCATION AND CARE: THE HEALTH AND WELLBEING CASE


The dramatic social and demographic changes that have occurred in developed nations over the past several decades have lead to significant changes in the circumstances in which families are raising young children, and in the conditions that young children experience as they group up. In parallel with these social and demographic changes, there is evidence of worsening health and developmental outcomes for many children and young people (Perrin, Bloom & Gortmaker, 2007). These have associated social and economic costs that undermine the general productivity and wellbeing of countries, and the fact that they have occurred in those very countries that have benefited most from economic and technological advances has been termed ‘modernity’s paradox’ (Keating & Hertzman, 1999).

There has been extensive research into those biological and environmental factors that increase the risk of poor outcomes, in children and throughout the life course; similarly much is known about protective factors that foster resilience in children who are otherwise at risk. While the pathways that lead to health, developmental and social problems have been well described, the research about effective interventions is less than robust, both at an individual and especially at a population level.

FACTORS THAT INFLUENCE CHILD HEALTH AND DEVELOPMENT:

There are many factors, both biological and environmental, that impact on child health and development – genetic, familial, environmental, community and societal. It is rare that any one of these factors determine child health outcomes, or that they act in a simple causal fashion. While there are some genetic causes that act in a direct linear causal fashion to threaten health and wellbeing, multiple factors usually interact with each other in a more complex fashion to influence outcomes. This representation of interactions among multiple influences that affect children’s health has been called a ‘kaleidoscope model’ (National Research Council & Institute of Medicine, 2004). These interactions between factors may occur within single domains, such as behaviour or social environment, or they may extend beyond domains, for example the child’s social environment, behaviour, and local or national policies. Changes in any one factor may influence others, so giving rise to a complex interaction of factors that may all play a role in determining the child’s current and future health. Each influence in turn interacts with other influences to form a pattern, which in turn sets up the substrate for future patterns. In this way children’s health and wellbeing is determined by the prior state of the child’s health, and by the presence or absence of risk and protective factors and their interaction.

a) Genetic factors

Genes have their effect either in correlation with or in interaction with the environment. Individual differences in human development may to a large extent
be explained by gene-environmental interactions that result in differential susceptibility. Common childhood conditions such as obesity, asthma and ADHD have all been described as having a strong genetic foundation. Nonetheless, changes in gene pool cannot explain the recent dramatic growth of these conditions, suggesting that the strongest aetiological pathways are environmental (Perrin, Bloom, & Gortmaker, 2007).

b) Familial factors

**Poverty**: One of the most consistent associations in developmental science is the relationship between economic hardship and compromised outcomes in children’s health, wellbeing and development. Furthermore it is suggested that ‘the malleability of young children’s development and the overwhelming importance of the family ... context suggest that economic conditions in early childhood may be far more important for shaping children’s ability, behaviour and achievement than conditions later in childhood’ (Shonkoff & Phillips, 2000).

**Maternal employment**: There is some accumulating evidence that maternal employment in the child’s first year of life, especially if the mother works long hours, can be a negative factor for infant development. However, beyond the first year, there is evidence that children may actually benefit from maternal employment, especially those from disadvantaged families, and particularly with regard to cognitive outcomes. In this context, the out-of-home care that these children experience can be regarded as a form of early intervention. Overall, parental employment can be seen as being either a positive or a negative factor for young children’s development; this depends on the nature and structure of the job, the income it generates, and especially on the environments and relationships that children experience when they are not in the care of their parents.

**Parental education**: There is a strong consistent correlation between parental education level and their children’s achievement and behaviour. Parental education levels are strongly associated with the home literacy environment, parental teaching styles, and investment in a variety of resources that promote learning. Children of parents with limited education, especially maternal, are at an increased risk of behaviour problems, poor literacy and school performance, and developmental delay.

**Family structure**: There is little evidence that family structure in and of itself is a significant factor in determining child outcomes. While growing up in a single parent family may increase the risk of school difficulties and behaviour problems, these are related to the socio-economic realities of single parenthood – lower income, less parental time from both mothers and fathers – rather than from any direct effect of living with only one parent. There is no evidence that children growing up in non-traditional family forms are at any increased risk of poor outcomes.

**Parenting**: An increasing body of research has documented the relationship between parenting and the development of a wide range of health, developmental and behaviour problems both in childhood and through to adulthood (Richter, 2004). For example, the quality of relationships that parents have with their
children predict healthy eating, and the only programs which have an (albeit modest) success in treating childhood obesity are those which focus on parenting skills as well as lifestyle advice. Adverse parenting is also a risk factor for the adoption of smoking and alcohol use, teenage pregnancy, and poor mental health in children and adolescents. These relationships appear to be independent of socio-economic status. It has been suggested that poor parent-child relationships have an adverse impact on the areas of the brain that deal with emotional and social functioning and with the physiological response to stress. Healthy and unhealthy patterns of relationships in the early years seem to be hardwired into the brain early in life, dictating subsequent resilience of vulnerability to stress and proving difficult if not impossible to moderate later in the life cycle.

c) Community factors

Evidence on the impacts of community and neighbourhood environments on child development and health is complex. Evidence suggests that dramatic changes - eg. moving from high poverty to low poverty neighbourhoods – can enhance the physical and psychological health of children. Whether smaller, more easily achieved changes in neighbourhood conditions produce improvements in children’s health, wellbeing and development is less clear.

Social Support: Numerous studies of children and families have shown that social support has a direct influence on the wellbeing of children and families. Social support has been found to be linked to a number of child and family outcomes, including low birth weight, child abuse, child neglect, maternal adjustment, mental health, and physical health (Cooper, Arber, Fee & Ginn, 1999). Those families who are most in need of social support and who would benefit from it are often those that are the most isolated

e) Societal Effects

Health is affected by environmental and social processes as well as by sociological factors, and the society and community in which a child lives are major determinants of health. Therefore, it has been argued that, because the primary determinants of disease in the twenty first century are mainly economic and social, the remedies must also include economic and social interventions (Rose, 1992). At a population level, the contribution of traditional medical care is modest, and healthcare needs to focus more on prevention. ‘This involves community approaches as well as individual health care, and must take into account the physical and mental health of the adults who interact with children and young people’ (Hall & Elliman, 2003).

Adverse health outcomes have been shown to be associated with environments that threaten personal safety, that limit the ability to develop strong social ties, or that are characterised by conflictual, violent or abusive interpersonal relationships. These effects occur across the lifespan. Positive health outcomes are associated with environments that provide safety, opportunities for social integration, and the ability to predict and/or control aspects of that environment.
Social capital, defined as the networks of social relations which are characterised by norms of trust and reciprocity, has been shown to be related to health outcomes. In communities that are high in social capital, there are strong connections between members of the community based on mutual trust and reciprocal exchanges. Like social support, social capital has been linked to a number of factors, including improved health, greater wellbeing, better care for children, and lower crime rate (Kroll, 2008).

Social gradients. There is clear evidence of the relationship between social gradients and health outcomes: where we stand in the social hierarchy is intimately related to our chances of getting ill and to how long we live. The Commission on Social Determinants of Health (2008) took a holistic view of social determinants of health:

‘The poor health of the poor, the social gradient in health within countries, and the marked health inequities between countries are caused by the unequal distribution of power, income, goods, and services, globally and nationally, the consequent unfairness in the immediate, visible circumstances of peoples lives – their access to health care, schools, and education, their conditions of work and leisure, their homes, communities, towns, or cities – and their chances of leading a flourishing life. This unequal distribution of health-damaging experiences is not in any sense a ’natural’ phenomenon but is the result of a toxic combination of poor social policies and programs, unfair economic arrangements, and bad politics. Together, the structural determinants and conditions of daily life constitute the social determinants of health and are responsible for a major part of health inequities between and within countries.’

How do social gradients affect health? Wilkinson (2005) argues that inequality is socially corrosive and affects health because the quality of social relations is crucial to well-being. In wealthy countries, health is not simply a matter of how material circumstances determine quality of life and access to health care; it is how social standing makes a person feel. Low social status — being devalued and looked down on — is stressful and can have devastating effects on people’s lives and communities. More unequal societies have poorer communal environments, which in turn is related to a range of social issues from higher levels of violence to more widespread depression.

f) Environmental Factors

Child health and development may be affected directly or indirectly by a number of environmental factors.

Climate change: Climate change poses direct and indirect risks to health, impacting on exposure to sun and extremes of climate, food safety, water supplies, and a higher risk of natural disasters such as fires and floods. There is almost no component of health and wellbeing that will be untouched by climate change. However, it is suggested that the risk distributed unequally across society, as a vulnerability to the effects of climate change depends on the degree of exposure, sensitivity, and adaptive capacity.
**Environmental toxins:** It is suggested that over the past few decades environmental exposures are contributing to children’s declining health status. Environmental agents that we know cause health and developmental problems in humans include, alcohol, nicotine, lead, mercury, arsenic, solvents, pesticides, and many others. Many of these are organic and inescapable; they enter the food chain from sources such as pesticides, chemical manufacturing and incinerated waste, and accumulate in animals higher up the chain. However, there are many more chemicals in common use whose effects on children, either singly or in combination, are unknown because they have never been tested (Collaborative on Health and the Environment, 2007).

Children are often more susceptible than adults to the effects of exposure to environmental agents. This is a particular issue during pregnancy, where the foetus is exposed to larger doses relative to bodyweight (International Scientific Committee of the International Conference on Foetal Programming & Development Toxicity, 2007). Exposure during fetal development can adversely affect health and wellbeing and can lead to lifelong functional deficits and increased disease risks.

Exposure to environmental pollution, though a major source of health risk throughout the world, is particularly problematic in developing countries where unsafe water, poor sanitation and poor hygiene, along with indoor air pollution, are major sources of exposure.

Associations between environmental pollution and health outcomes are complex; individual pollutants may be implicated in a wide range of health effects, whereas few diseases are directly attributable to single pollutants. However, most of our exposures to these chemicals are not from sources traditionally regulated, such as remote waste sites and factories. Rather, the primary sources are close to us: within our indoor environments, and the personal activities, products, and materials inside those environments. The sources of these pollutants are largely unregulated – meaning that our environmental regulations, designed to protect and promote human health, are missing major sources of health risks.

**Changes in urban environments:** There is growing recognition that the built environment – the man-made physical structures and infrastructure of communities – has an impact on health. A good example of this is the opportunities children have for physical activity. Increased urban density, and reduced access to parks and safe places for children to play, accompanied by parental concern about children’s safety, is said to have contributed to the increase in obesity (Perrin, Bloom & Gortmaker, 2007). Physical environments and community recreation facilities have an impact on how children use their time, and particularly their likelihood of physical activity.

**Changes in home living environments:** Many allergies and immune system diseases have significantly increased in prevalence in the past few decades; asthma, hay fever and food allergies have all increased significantly (Stein, 2008). While the exact cause is debatable, it has been suggested that all may have a common explanation rooted in aspects of modern living. One theory, termed ‘the
hygiene hypothesis’, suggests that these increases are due to children growing up in an increasingly sterile environment; other causes postulated include changes in diet, air pollution, and increasingly sedentary lifestyles. Evidence for the hygiene hypothesis comes from studies demonstrating lower rates of allergies in children who live on farms and whose mothers lived on a farm during pregnancy.

**Changes in food consumption:** There have been considerable changes in children’s eating habits, including increases in high energy foods, meals and snacks eaten outside the home, and increased portion size. So-called fast foods tend to have low quality carbohydrates and fats, little fibre, few essential nutrients and high energy density. Fast food outlets are more highly concentrated in lower socioeconomic areas, contributing to the higher rates of obesity in disadvantaged communities. Changes in food production and food consumption have seen increases in the use of food additives, reduction in fruit and vegetable intake, and an increase in the consumption of sugar sweetened beverages.

**EVIDENCE OF LONG TERM IMPACTS OF EARLY EXPERIENCES:**

Developmental pathways originate in the complex interplay between biology and experience; there is accumulating evidence of the child’s immediate environment in the early years as having a major impact (Hertzman, 2004). The evidence suggests that early behaviour and functioning are predictive of later behaviour and functioning to the extent that children’s social and physical environments remain unchanged. In other words it is difficult for children and families to extricate themselves from adverse circumstances, especially when there is multiple risk or adverse factors in their lives.

However, there is also evidence of developmental plasticity over the life span. Plasticity is the potential for change in intrinsic characteristics in response to environmental stimuli. Children’s development continues to be shaped by experiences throughout the course of childhood. Moreover, there is emerging evidence that suggests that the brain can change itself or can be changed by experience to a much greater degree than was previously recognised. For instance, stress neurobiology is highly responsive to changes in the environment: although very sensitive to early social contexts, it is not a fixed or inflexible system, but reflects both the organism’s epigenetic history and its new circumstances. Improved living conditions, enriched environments, and corrective emotional experiences can reverse the adverse consequences of early adversity.

Hertzman and Power (2003) describe three mechanisms through which exposure to both beneficial and adverse circumstances over the life course impact on health and development.

**Latency or sleeper effects:** This hypothesises that there is a relationship between exposure at one point in the life course and its impact on health many years later. In recent years there has been an emerging body of research suggesting that the roots of adult disease lie in foetal and neonatal development (Barker, 1992).

It is suggested that the mechanism for this to occur is that nutrition or exposure to environmental toxins in utero in the neonatal period may affect the programming
of tissue function that occurs during development. This concept is called the developmental basis of health and disease, and the process by which this occurs has been named ‘biological embedding’ (Keating & Hertzman, 1999).

It has been shown that low birth weight (small for gestational age) strongly predicts the subsequence of hypertension, hyperlipidemia, insulin resistance, type 2 diabetes, and ischemic heart disease (Heindel, 2007). Low birth weight is taken to be an indicator of poor nutrition during pregnancy, and it is hypothesised that the foetus permanently changes its structure and metabolism as an adaptation to a limited supply of nutrients. The metabolic demands of the growing brain and heart arefavoured at the expense of other tissues.

Cumulative effects: In this model, there are either multiple exposures to a single recurrent factor (such as poverty) or a series of exposures to different factors; these exposures to risk factors (and protective factors) may accumulate over the life course. While in some cases exposure occurs in a dose response manner – eg. the health effects of exposure to a toxic substance usually increase with the duration and intensity of exposure – the biological mechanisms by which disadvantages and inequities carried over a life course of differential exposures leading to health disparities are not well understood. One mechanism might be the body’s biological response to stress, involving the autonomic nervous system, the hypothalamic-pituitary-adrenal (HPA) axis, the cardiovascular, metabolic and immune systems.

Many studies have shown the cumulative impact of risk and protective factors on young children’s development. These studies show that the risk of core developmental outcomes in children and adolescence increases in a linear fashion as the number of environmental risks increases. The corollary is that the incidence of positive developmental outcomes increase as the number of protective factors in children’s lives increases. There are well established correlations between a range of adverse childhood experiences, including abuse, neglect and household dysfunction, and later health problems such as ischemic heart disease. This effect is cumulative – the more adverse childhood experiences, the more likely to develop ischemic heart disease later in life. These adverse early experiences are more strongly predictive than traditional risk factors such as smoking, obesity, diabetes and hypertension. Adverse events in childhood have also been shown to be directly correlated with other problems in adulthood, including alcoholism, illicit drug use, and mental health problems such as depression.

Pathway effects: In this model, exposure to risk factors at one stage of the life course influences the probability of other exposures later in the life course. Early events may influence the life course trajectory, and once a child is established on that trajectory he or she is more likely to experience other exposures which strengthen the likelihood of poor outcomes. For example, children from disadvantaged backgrounds have a greater risk of poor school readiness, as measured by cognitive and social-emotional competencies at school entry. Because of this, they are more likely to experience problems at school, with a subsequent increased risk of unemployment, poor self esteem, and mental health problems. It is hypothesised therefore that early adverse experiences disrupt
neurological development, resulting in social, emotional and cognitive impairments, which in turn lead to increased risk to health behaviours that result in disease, disability, and social problems. Mechanisms for this are uncertain, though one suggestion is that a cascade of risk is created early in life that exacerbates certain genetically based vulnerabilities. This may lead to deficits in children’s control of and expression of emotions and social competence, and also lead to disturbance of physiological and neuro-endocrine system regulation that can have cumulative, long term adverse effects.

The three models described are not mutually exclusive, but are present simultaneously in any individual’s life course. Environmental lead exposure can be used to illustrate this. This can have a cumulative impact, because lead is retained in the body, and there is a dose response curve so that the more retained the greater the health risk. At the same time, lead exposure in utero may also have a latent effect on subsequent health and wellbeing through inhibiting the production of brain cells. Finally, there may also be a pathway effect, since the socio-economic position will affect the probability of exposure to lead in utero and in childhood, which may impact negatively on a range of outcomes throughout the life course.

Each of the three sets of influence on health described by Hertzman and Power (2003) - latency, cumulative, and pathway - carries with it a strategic message for policies and interventions to improve population health. The message of latency is ‘the earlier the better’; the message of the cumulative model is ‘intervene wherever there is an effective intervention’; and the message of the pathways model is ‘intervene at strategic points in time’. All three of these forms of intervention will be needed if broad-ranging improvements in health and developmental outcomes for children are to be achieved.

Summary and Conclusions

Humans are unique in the degree to which they can adapt to their environments. Young children’s neurology and biology are particularly adaptable, being designed to learn from their prenatal and early post-natal environments. They are highly sensitive to both positive and negative experiences, which lay down biological, neurological and behavioural patterns which become increasingly difficult to change, and which can have life-long consequences for health and well-being.

Given the evidence of the links between early experience and later health outcomes, there is a strong argument for greater investment in the early years to reduce the long-term burden of poor health in adulthood. And given the evidence that the primary determinants of poor health outcomes in the twenty first century are mainly social and economic, these investments should focus not so much on traditional forms of medical care, but on addressing the social and economic conditions under which families are raising young children.

Bibliography


**Further Reading**


Kuh, D. & Ben-Shlomo, Y. (Eds.)(2004). *A life course approach to chronic disease epidemiology; tracing the origins of ill-health from early to adult life (2nd Ed.).* Oxford: Oxford University Press.


