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Valuing a Healthy Start to Life

How should governments make effective use of research that examines the costs of a ‘less than healthy’ start to life? Are there any ‘free efficiency lunches’ to be had by improving intervention processes in early childhood health and education? On the surface at least, health and education are prime areas for improved efficiency gains because of the large amounts of public money spent and the potential to create substantial public value.

In this article this is considered in the context of a society which struggles with how social choices are made.¹ One approach suggests that we allow society to pursue its goals whatever they may be. In modern debates this is about public value. Moore (1995) suggests that public value depends on the authorising environment and operational capacity.² The focus of this article is on operational capacity,³ and, in particular, on exploring new

techniques for assessing where further value might be created by investigating the impacts of a less than healthy start to life from epidemiological, economic and policy standpoints.

My wish is to integrate our understanding of the various approaches and emerging evidence in a manner appropriate for policy makers to use. Thus, I am looking from the ‘policy-making market’ back down the ‘research

pipeline’ to assess the salient factors that may guide child health investment decisions.

The epidemiological evidence of the implications of a less than healthy start to life is growing. A policy response is required. However, while we now have a deeper understanding of the physical consequences of a healthy start to life through the Developmental Origins of Health and Disease (DOHaD) programme of research, the growing economic evidence is not well connected to the science, and the policy debates are nascent. The notion that being healthy at birth maximises the chances of being a healthy adult is a simple idea with complex implications. This also makes it difficult to quantify.

The epidemiological complexity requires understanding the variable strength of the pathways which deprivation *in utero* prescribes, and how these might differ given different ethnicities, genetic profiles and other potentially relevant attributes. The rough proxy to indicate a constrained *in utero* environment is birth weight. According to the World Health Organization and UNICEF (2004), low birth weight is a birth weight

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of less than 2,500 grams. The economic and policy challenge is to construct a practical and logical connection with the epidemiological research. Further challenges include identifying and assessing the direction and strength of associations and how they might change; understanding how changes in income influence health and what the dynamics are of the interaction between the two (Thomas and Frankenberg, 2002); and identifying what other factors come into play in predisposition to illness.

McMichael (1999) sets out the major problems to be overcome: a preoccupation with risk factors that represent an immediate vulnerability for a particular condition, when rather

programming for later life health: the development deficit approach; the thrifty phenotype (Hales and Barker, 1992); and the predictive adaptive response (Gluckman and Hanson, 2004a, 2004b).

The Barker hypothesis has been modified into a more nuanced understanding of how a wide range of early life effects and illnesses may affect individuals (Gluckman and Hanson, 2004b). These explanations are not mutually exclusive, and focus on the importance of *in utero* conditions.

What has emerged is the DOHaD – Developmental Origins of Health and Disease – hypotheses (see www.soton.ac.uk). For example, researchers have linked cardiovascular disease, type

Animal studies

Conformation of the Barker and Hales fetal origins hypothesis focused on animal studies. These studies examined ‘western lifestyle’ risk factors, including high-energy, high-fat and low-fibre diets (Benyshek, 2007). They confirmed that processes that had an influence on health, particularly obesity, begin in the periconceptual and embryonic periods (Fleming, 2006) and are extended through postnatal growth (Eriksson et al., 2003). Animal models also show that maternal nutrition has a critical impact on the development of obesity and related diseases (Armitage et al., 2004).

Longitudinal studies

Links between a poor start to life and later life diseases were noticed by examining longitudinal studies (Barker et al., 1989). Researchers uncovered relationships (correlations) between variables over long time periods. Studies such as the Aberdeen Children of the 1950s cohort study (Batty et al., 2004) are typical of the type of ‘data warehouse’ which has been employed to examine life course influences. The Aberdeen study and others have illustrated the relationship between low birth weight and coronary heart disease (Leon et al., 1998), and low birth weight and non-insulin-dependent diabetes mellitus (Lithell et al., 1996). Low birth weight has also been linked to schizophrenia (Cannon, Jones and Murray, 2002), cognition (Breslau, 1995), behaviour in childhood (Kelly et al., 2001) and psychiatric disorders in adulthood (van Os et al., 1997).

Two studies in New Zealand (Dunedin and Christchurch) have highlighted material on the impacts of disadvantage and family violence on later life criminality (Jaffee et al., 2002). A third New Zealand study, Growing Up in New Zealand, is now under way. Its main strengths are that it takes a much larger and more ethnically diverse cohort of parents and children than previous New Zealand studies, and that it asks a wider range of questions: medical records are set in the context of the social and cultural environment that surrounds the children, and the study is designed to elucidate the multiple determinants

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what we may face is a ‘web of causality’ (Wolfson, 2002); reconciling the difference between individual and population-level influences on health; understanding how risk factors change over time in a life-course approach; and how to gauge the impact of large-scale social change using scenario-modelling techniques.

The following section of this article sets out the research scene, looking at why epidemiologists became interested in this problem and their research agenda. The article then looks at how economists have responded to the epidemiological research. Finally, it examines how policy makers might approach the problem, given ongoing research in a new area.

Setting the scene

The impact of low birth weight on stunting and chronic disease in later life has been explicitly set out as a hypothesis only relatively recently (Barker et al., 1989). Barker, in collaboration with Hales, demonstrated that adult health could be strongly associated with size at birth (Hales et al., 1991). Three theories have been advanced by researchers to explain the importance of intra-uterine

2 (non-insulin-dependent) diabetes and osteoporosis in adults with low birth weight. DOHaD’s aim has been to provide a scientific basis for public health measures directed at preventing these diseases. DOHaD suggests that adult-related diseases ‘originate through adaptations which the fetus makes when it is undernourished, and which permanently change its structure, physiology and metabolism.’⁴

Epidemiology

For the DOHaD hypotheses, the complexity of biological systems means that it is difficult to say with absolute accuracy how even the simplest versions behave. Epidemiological research focuses on understanding why we see associations between temporally distinct events and the pathways between them. Understanding the strength of a causal inference requires the gathering of data and applying a broad range of biomedical and psychosocial theories in an iterative way to generate or expand theory, test hypotheses, and make educated, informed assertions about which relationships are causal and how they are causal.

that interact over time to determine why intra-uterine development is associated with later life health outcomes, beginning in early life with childhood growth and cognition. Importantly, and unusually, the study collected baseline pregnancy data to examine the influence of development and environments existing before and during pregnancy.

Longitudinal studies are prospective and observational; they examine the world without controlling or manipulating it. They have less power to detect causal relationships relative to other experiments. However, sufficiently large longitudinal studies with repeated observation at the individual level are valuable. Scale allows subgroups that can proxy 'with' and 'without' samples to be drawn. Other strengths lie in the ability to use analysis to exclude time-invariant unobserved individual differences, and to use the temporal order of events as evidence. They also provide an opportunity to understand the relative influence of distal and more proximal influences on developmental outcomes. Longitudinal studies have the great advantage of providing data that can be used to establish parameters for the population where interventions are planned.

Famines

While there have been many famines, in few cases has there been the data to test aspects of the DOHaD hypothesis. One example where data was available was the Dutch 'hunger winter' famine, in which a short and unexpected event caused 18,000 deaths. During the famine, which lasted from November 1944 to April 1945, average daily rations fell below 800 calories. The Dutch Famine Birth Cohort Study found that the children of pregnant women exposed to the famine were more susceptible to diabetes, obesity, cardiovascular disease, microalbuminuria and other health problems (see Hart, 1993; Roseboom et al., 2001; Neugebauer, Hoek and Susser, 1999). The children of the women who were pregnant during the famine were smaller than average. While stunting was expected, what was more surprising was that when these children grew up and had their children,

those children were also smaller than average. This data suggested that the famine experienced by the mothers may have caused epigenetic changes that were passed down to the next generation.

Estimating economic parameters

The literature on wider health, social and economic costs and benefits of a healthy start to life is growing. As Alderman (2009) points out, while awareness of the need for investment in nutrition is not new, the spotlight has now been firmly placed on the data and techniques to prove the case, which are new.

Economists have made passing references to the ongoing epidemiological research, with the possible exception of

later life, and intergenerational benefits. Also, improved verbal and cognitive ability can improve later life outcomes (Dustmann and van Soest, 2003) and reduce likelihood of partaking in risky activities or crime (Heckman, Stixrud and Urzua, 2006). There is a direct economic impact in that healthier people are better workers: a productivity dividend from improved physical capacity and increased cognitive ability. Other costs of poor mental and physical health (e.g., see Heckman, Stixrud and Urzua, 2006) potentially have a major impact: New Zealand health system expenditure projections, for example, forecast a rise from 6.9% of GDP in 2011 to 11.1% in 2060 (Treasury, 2012).

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Almond and Currie (2010). In most articles, Barker is cited and cursory attention is paid to how the epidemiological research is progressing. A possible reason for this is the lack of incentives to connect the epidemiological and economic research. A major challenge for economists and epidemiologists will be to work more closely together to further understand the impacts of a less than healthy start to life.

Why are economists interested in DOHaD?

The Barker hypothesis has encouraged a growing economic literature (Almond, 2006; Black, Devereux and Salvanes, 2007; Dustmann and van Soest, 2003; Heckman, Stixrud and Urzua, 2006). The evidence across different populations shows that poor health outcomes have large costs to society throughout the life course of individuals (Ruger, Jamison and Bloom, 2001). The generalised links suggest that there are social 'dividends' that can be gained.

The health dividend includes lower infant and child mortality, reduced health care costs for neonates, infants and children, reduction in chronic diseases in

Heckman's observation that 'skills beget skills' succinctly describes the path dependency associated with the education dividend. Learning today improves the ability to learn in the future. If further work can establish the mechanisms by which a healthy start to life can improve early-intervention educational outcomes further, then the impact will be much greater. This is of particular importance, because we know that the influence of early educational intervention can be felt not just today but in the future: it improves the rate of learning long after the child has left early education programmes. Relative to later life education, 'skills beget skills' is a reason for the higher rates of return from early life education.⁶

Moving from high to low rates of mortality and fertility and higher life expectancy can have a dramatic impact on a country's development (demographic dividend). Increasing numbers of young children can increase the working-age population – witness East Asian growth, for example (Bloom and Sachs, 1998). The opposite can also occur. In sub-Saharan Africa the mounting disease burden

means that resources are spread among large numbers of children, creating high fertility and high mortality (Bloom and Sachs, 1998).

These insights offer explanations of how health improvements can assist development goals. However, the challenge is to demonstrate the strength of each DOHaD mechanism. Estimating how much public value can be generated will be crucial in influencing policy makers. This is where the economic tools can be utilised to great effect to illustrate the magnitude of the various impacts.

Theoretical underpinnings

Arrow (1963) first set out the differences between good health and other goals using a production function approach. Phelps (2003) identifies these factors as heavy government involvement, asymmetric information, spillover effects (externalities), barriers to entry and

respond to further health investments and socio-economic issues and the possible interaction between biological and environmental impacts (ibid.).

Despite the appeal and persuasiveness of a production function approach by economists, the key limiting factor to further progress is the lack of availability of large-scale longitudinal data sets with which to evaluate the long-term impacts of a less than healthy start to life in detailed terms.

Animal and longitudinal studies

Questions remain over the strength of the DOHaD hypothesis pathways that link later life diseases with a constrained *in utero* environment. Retrospective epidemiology has not proven the hypothesis since we lack the clinical data of neonate development for large populations. It is also unethical to test the hypothesis using human populations. Women cannot be

so it is not known if one intervention on a specific link will be effective.

Natural experiments

Elements of understanding are vital, since, as a matter of effectiveness, proof is required that *a priori* effect is necessary and/or sufficient. A key matter is to establish sufficiency, since it is uncertain that all variables are required for testing cause are included. In many cases, the confounding variable is poverty. Poor regions have poor health outcomes and poverty is passed on through generations.

Twin studies

Twin studies can control for omitted bias. Genetic and environmental pregnancy and maternal factors can be held constant to focus attention on the results of physical differences across populations. Studies by Royer (2009), Black, Devereux and Solvanes (2005) and Oreopoulos et al. (2006) for different countries found that twin differences in birth weight were positively associated with subsequent educational attainment, although Royer suggests these differences were small. Twin birth weights were found to be positively associated with adult labour market outcomes (Black, Devereux and Solvanes, 2005).

Royer points to a number of constraints. Parents and health care providers may give varying degrees of care (Becker and Tomes, 1976). Low birth weight may systematically alter the investments in each twin: i.e., the lighter twin might receive more care. Furthermore, there are concerns about survivor bias, where the lack of fetal nutrients may increase early-age mortality rates, and therefore those who survive are likely to be resilient. This is reinforced by Rosenzweig and Zhang (2006), who point to expenditures on schooling being positively correlated with weight differences at birth.

Unique data sets

To mitigate the impact of the omitted variable problem, Almond (2006) uses the 1918 influenza pandemic in the United States to test the DOHaD hypothesis. The 'Spanish flu' arrived unexpectedly in September 1918 and was largely over by January 1919. The sharpness and brevity of the event, coupled with its heavy

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third-party agents. Grossman (1972) built on Arrow's work in setting out a health demand function which portrayed health as a stock variable which varied over time with further investment and depreciation. Wagstaff (1986) demonstrated how early life investments can have a disproportionate impact on later life health.

Almond and Currie (2010) document further additions to the Grossman model. They include the use of a constant elasticity function to overcome the strong perfect substitutability assumption, i.e. that all health investments should be concentrated in one period (Heckman, 2007); the level of health investment to ensure that 'damage' can be shown as proportional to the total stock of health investment in a person (ibid.); the potential resilience when damage in the first period is particularly large (Almond and Currie, 2010); and demonstrating how children

randomly assigned deprived or enriched environments during pregnancy and their offspring's morbidity and mortality be tracked without intervention.

A disconnect exists between the debates being carried out among epidemiologists about proving the way the various associations and causal pathways can be understood as a biological mechanism, and economists scrambling for data to test different aspects of the general hypothesis. The disconnect can occur in a number of ways. The use of animal studies focuses in on proving one causal link. The problem is, how might you generalise this to a population level? If there is a web of causality, perhaps only one link within it has been uncovered. By using longitudinal studies, distal correlations are uncovered but causal pathways are not. While a general relationship may include all causes, they are not untangled

impact on pregnant women, make it ideal for examining the DOHaD predictions. Almond shows that those cohorts who were *in utero* during the short period that the pandemic was active suffered from reduced educational attainment, increased rates of physical disability, lower income and status and higher transfer payments relative to other cohorts.

Van den Berg, Lindeboom and Portrait (2006) use Dutch registers of birth between 1815 and 2000 combined with macro-economic data outcomes to examine the impact of recessions on individual mortality. They found that being born during a recession increases the mortality rate late in life for most of the population. Lower-income earners tend to be affected disproportionately from being born in recessions. Higher-income earners born during recessions have a much more muted mortality response.

Almond and Mazumder (2011) focus on the holy month of Ramadan to illustrate the linkages between fasting and fetal health. They find that Muslims in Uganda and Iraq are 20% more likely to be disabled as adults if early pregnancy overlapped with Ramadan. The impact is larger for learning disabilities.

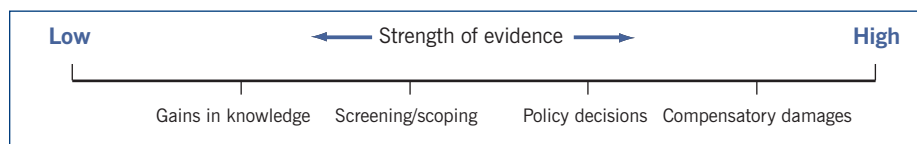
Implications for policy analysis

Much of the research debate has been about seeking stronger proof of the DOHaD hypothesis. Policy-making has a different logic; it is fundamentally practical and looks for results more than knowledge.

What is the problem?

Policy analysis defines the problem and asks what intervention is appropriate. Interventions should be designed to maximise welfare over time; their context relates to the health issue identified and includes the potential costs and benefits. Ideally there would be no need to intervene, as markets would allow those affected to take care of the health risks. However, market failure can occur, such as lack of or poor parental information about the link between current maternal health and future economic outcomes of potential offspring. Possibly, regulatory or other intervention might pay off for society.

Figure 1: Continuum of decision settings



Source: Brookshire (1992)

Does the market resolve the problem?

Less regulation is preferred to more. Generally, people should be free to engage in activities unless they are prohibited for some good reason. Good regulatory design should signal the importance of innovation for economic growth and the maintenance and enhancement of standard of living. This is why many governments have been proactive on maternal smoking, because of the direct harm through lung cancer and the indirect health problems for children of smokers. The problem that the DOHaD hypothesis sets out is in a similar category. Many parents or caregivers require guidance to provide the early life-course environment appropriate to prevent risks of later life diseases. The market has not by itself been able to ameliorate the problem.

Is there a workable government intervention available?

No ready-made solution exists for intervention because the problem is new and there is debate over the strength of the impacts. However, a number of suggestions are made here as to how to think about a possible approach to intervention. While it is important that the evidence for an approach is as strong as possible, policy makers are willing to expose themselves to 'evidence error' to inform better policy-making advice (OECD, 2006).

Brookshire (1992) sets out an approach as shown in Figure 1. If the objective is to gain more information about a policy or to develop an initial assessment, then a relatively low level of data or evidence is required. Higher degrees of evidence are required if a national policy decision is being made. In such cases, a compelling case which supports any particular approach may be required.

However, this 'proportionate' approach depends on the assessment of the state of the evidence, which in itself can be subjective.

Different types of evidence need to be considered: evidence about the scale and workings of the problem; evidence about the different types of interventions and their technical effectiveness; and evidence from consultation to test the idea with the affected population and highlight responses likely responses which can be influential as to outcomes (NZIER, 2011). Policy needs to consider the evidence about the problem and the risks of doing nothing. The policy maker also has to be aware that those investigating the problem can give only a 'snapshot' of current thinking.

Developing effective interventions that are able to mitigate some of the impacts of a less than healthy start to life will be a major challenge for policy makers. It is also where the evidence from longitudinal research will be able to more clearly demonstrate the parameters of what can be achieved. This highlights the importance of Growing Up in New Zealand to inform policy options.

Policy framework

Wolfson (2002) sets out an approach to examining the impact of a less than healthy start and possible interventions. Using tax and climate change modelling, Wolfson illustrates how scenarios can be generated from an observed counterfactual. Tax authorities run detailed microsimulation models to estimate not only total revenue impacts but also changes in income for various groups within society. Similarly, global climate change models estimate temperature levels in different locations.

One concern is that the development of outcomes, particularly single-value outcomes, may confuse researchers' preferences with scientific facts. Wolfson suggests that this concern is misplaced if the 'what if' values are generated by richly-populated microsimulation models. These models are able to separate the amounts of time various groups (or units) spend in each health state over time. Thus an

Figure 2: Frame of reference

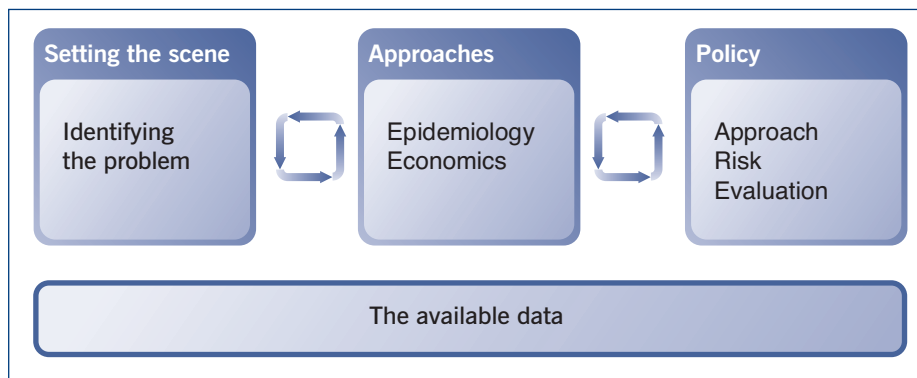
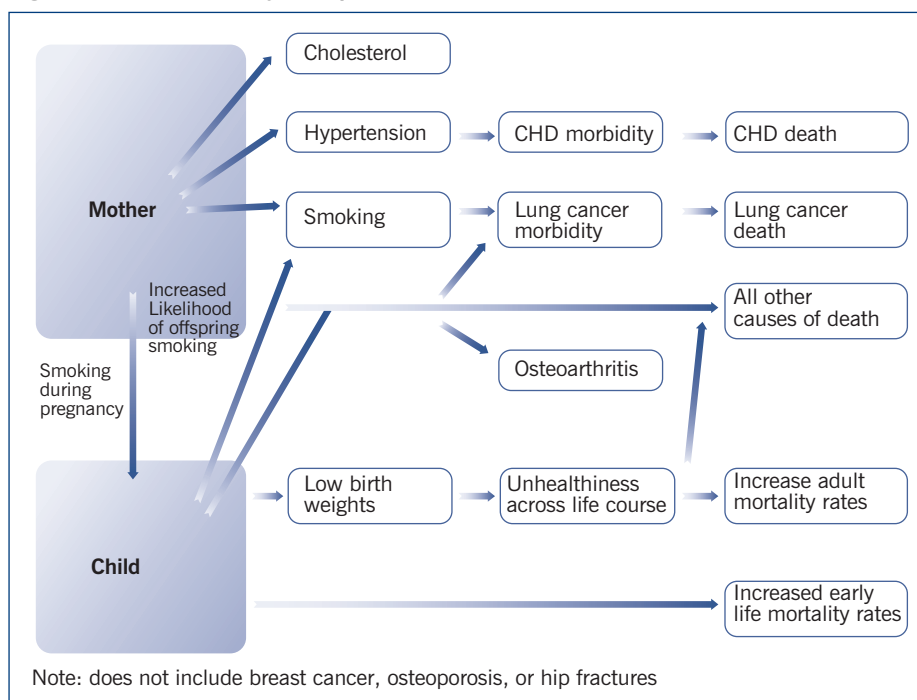


Figure 3: Web of causality example



Source: adapted from Wolfson (2002)

individual's life course can be modelled to analyse the impact of specific lifetime events. These models are able to test various assumptions associated with the parameters to assess their importance on the overall result. Also, the 'cause' impact can be disaggregated by age, sex and other population characteristics.

All scientific work is incomplete and further developments of methodology are expected, but the microsimulation approach represents the 'best of our current state of knowledge' (Wolfson, 2002).

Evaluation

If we are uncertain about the outcome of an intervention (investment), then an evaluation is required. This is not a trivial

exercise, since such an evaluation should not only pinpoint areas of weakness in the selected policy but set out the direction of future work.

Unfortunately, there are real issues with how programmes are evaluated across government. Hallsworth, Parker and Rutter (2011), commenting on the English regulatory framework, point to programme evaluation as being problematic: evaluations are often commissioned but are often ignored; central government is culturally not very interested in learning from the past; timescales for evaluation and policy-making are out of sync; departments have the incentives and opportunity to tone down unfavourable findings; evaluations are often not built into

policy design and are sometimes poorly executed; and evaluation findings are often not managed well and can inhibit organisational learning. For good policy-making, the design of the evaluation is just as important as the initial research, as it can act as a signpost and maximise chances of longer-term 'policy success'.

When to act?

Rasmussen (2001) argued that the DOHaD paradigm had not been proven to the point where causality has been shown and that it was too early to use research as a basis of intervention. Ten years on, there has been an increasing number of animal experience studies and numerous journal articles building the evidence base to support the DOHaD paradigm. Not all of these articles have been supportive of DOHaD conclusions, but most add to the evidence base supporting the DOHaD hypothesis.

If the DOHaD hypothesis is correct, every day some children are born who might be saved from a poor start to life and its consequences if worthwhile intervention programmes were commenced. The logic of the decision does not revolve around the level of proof of the hypothesis alone. It hinges on taking uncertainty into account in assessing the expected returns on possible interventions to address the groups who are potentially at risk. Such an assessment is not a simple task, as there are a range of factors that have to be estimated and this introduces more uncertainty.

Narrowing the uncertainty, multidisciplinary style

To further our understanding, a more coherent approach is required to tackling the issues. While a multidisciplinary approach has always been held up as best practice, in reality such approaches are rare. Ideally, a policy framework which focuses on the interaction between the problem definition, various approaches being taken and outcomes is required (see Figure 2). Current approaches, while extremely helpful in elucidating various epidemiological and economic aspects of a poor start to life, have, nevertheless, been disconnected and of passing use to policy makers.

Complexity of the interconnections

A compelling reason for closer cooperation between different disciplines is the complexity of the issues. A one cause–one effect outcome is highly unlikely (Rothman and Greenland, 1998). Figure 3 illustrates the web of causality for maternal smoking, for example.

With the DOHaD hypothesis there is still debate about the various pathways that cause later life diseases and their relative impact. This means that if an intervention acts by affecting a specific pathway, then its overall impact will be hazy. Therefore, a richer understanding of the linkages between competing/overlapping approaches is required by economists and policy makers to better appraise the suite of possible interventions.

Further work is required to identify the missing data to assist our understanding of the various linkages. We need to identify what we already know, relate what we know to the policy problem, establish the gaps relative to the policy issue, make judgements about the importance of the gaps and how amendable they are to research, and enlarge the knowledge base in a manner relevant for policy.

Conclusion

This article has set out the approaches being taken by researchers investigating the likely impacts of a healthy start to life from epidemiological, economic and policy standpoints. For the ‘free efficiency lunches’ to be grasped, closer cooperation is required between epidemiologists, economists and policy makers to demonstrate the public value generated by a healthy start to life. This will require asking the right policy questions, developing the theory in part with the use of microsimulation tools, and using New Zealand-specific data, partly from the new longitudinal survey Growing Up in New Zealand. It will only be when the evidence emerges from this process that policy makers will be able give wholehearted support to re-prioritising and ranking health interventions and diverting social welfare expenditure.

Challenges include the significant time lag between policy interventions and the benefits of those interventions, identification of the most appropriate interventions and how they will be delivered, and evaluation of those interventions and over what timeframes. Both the practical

and research requirements to successfully implement DOHaD principles will require changes to current approaches. While these challenges are not insurmountable, they will require concerted efforts from researchers and policy makers over a long period of time.

- 1 Arrow (1963) first identified this problem through the impossibility theorem.
- 2 The authorising environment can potentially come from the legitimacy bestowed on governments through the election process, or stakeholders within the wider community who can demonstrate a commitment to a particular area or issue. By operational capacity Moore means how institutions can harness and mobilise resources which support policy implementation that creates or increases public value.
- 3 See Prebble (2012) for an in-depth discussion and debate on the practical application of public value theory.
- 4 <http://www.som.soton.ac.uk/research/olddohad/centre/whatis/default.htm>.
- 5 See, for example, <http://www.child-encyclopedia.com/documents/HeckmanANGxp.pdf>.
- 6 However, as the ECE taskforce (2011) suggests, the impact of early childhood intervention is highly confounded with the child's socio-economic background.

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