Purpose

1. This paper was prepared for the EAG to provide a summary of the findings from cohort studies about the negative lifecourse consequences of childhood poverty.

2. This paper has informed the direction and recommendations of the EAG’s Solutions to Child Poverty in New Zealand: Issues and Options Paper for Consultation. These are preliminary findings, and a final report will be published in December 2012. The findings in this paper do not necessarily represent the individual views of all EAG members.

3. The EAG wish to acknowledge Professor Richie Poulton and Dr Sandhya Ramrakha from the Dunedin Multidisciplinary Health and Development Research Unit, Department of Preventive and Social Medicine, Dunedin School of Medicine, University of Otago for their work on this paper.

Findings from cohort studies about the negative lifecourse consequences of childhood poverty

4. A vast literature exists linking socioeconomic disadvantage or poverty (hereafter simply referred to as poverty), with a broad range of negative outcomes. This pattern of association is observed regardless of the study design (cross-sectional vs. prospective-longitudinal), country of origin, or historic era in which data was obtained. Moreover, it exists at whatever point in the lifecourse that one chooses to focus - childhood, adolescence and adulthood. This almost ubiquitous finding has become known as the social gradient in which the poorest tend to have the worst outcomes, those in the middle of the socioeconomic spectrum have intermediate outcomes and those at the top, the best outcomes.

5. In what follows we: (i) report examples from different countries providing strong evidence for the basic association between poverty and poor outcomes; (ii) identify international studies that have been, by virtue of their design and the type of data collected, able to isolate independent effects of childhood poverty on lifecourse outcomes; (iii) discuss in detail New Zealand prospective-longitudinal data that has made important contributions to this international literature, due to its salience and relevance.
for New Zealand policy-makers; (iv) explore what might explain this robust poverty-poor life outcomes association i.e. possible pathways and mechanisms; and (v) consider the implications of the above for solutions to child poverty in New Zealand.

6. The take-home message from this brief overview is that circumstances in early life matter greatly for how life turns out as an adult. Thus, savvy investment for positive adult outcomes must begin in childhood.

Documenting the basic relationship between poverty and poor lifecourse outcomes

7. Research from cohort studies from around the world, conducted in different times in history, on samples of varying ethnicity and gender composition, document robust and significant correlations between childhood poverty and a range of deleterious outcomes in adulthood. The panoply of outcomes related to child poverty include elevated risk for: physical diseases (including cardiovascular disease and cancer), specific and all-cause mortality, as well as poor educational, cognitive, psychological and social functioning (e.g., Carson et al., 2007; Chen et al., 2007; Claussen et al., 2003; Davey Smith, & Hart, 2002; Galobardes et al., 2004; 2008; Glikson et al., 1995; Hallqvist et al., 2004; Hart et al., 2000; Kittleson et al., 2006; Kivimaki et al., 2004; Kuh et al., 2002; Lynch et al., 1997; McLaren, 2007; Power et al., 2003; Power, Graham, et al., 2005; Power, Hypönen, et al., 2005; Schreier & Chen, 2010; Singh-Manou et al., 2004; Sobal & Stunkard, 1989)

8. These studies illustrate the scientific ‘fact’ of the poverty-poor outcomes association. Not all studies, however, have shown conclusively that poverty in childhood exerts an independent (or direct) effect on adulthood outcomes. In other words the association between childhood poverty and poor adult health might arise simply because childhood poverty leads to adult poverty, which in turn determines poor adult health.

Childhood poverty is especially pernicious and has long-term effects

9. There is now sufficient evidence to support independent effects of childhood poverty on poor adult outcomes, above and beyond adult socioeconomic status (SES) and cumulative life exposure to disadvantage. Importantly, a number of studies have shown that the childhood effects persist even after taking into account upward social mobility (e.g., Hart et al., 2000; Kuh et al., 2002; Power et al., 2005; Ljung & Hallqvist, 2006; Pensola & Martikainen, 2003; Poulton et al., 2002).

10. The latter findings are taken to suggest that the experience of poverty during childhood can have particularly long-lasting, harmful effects. By way of illustration, we detail the findings of the highly cited New Zealand cohort study by Poulton and colleagues (2002). These researchers found that compared with children in the Dunedin Study from high socioeconomic status (SES) backgrounds, those who grew up in relative poverty had poorer cardiovascular health by age 26 years. Significant differences were also found on
dental health measures, with a threefold difference in adult periodontal disease (31.1 percent vs. 11.9 percent) and caries level (32.2 percent vs. 9.9 percent) in the poverty versus high SES groups. Substance abuse resulting in clinical dependence was also related in a similar way to childhood poverty (i.e. 21.5 percent vs. 12.1 percent for adult alcohol dependence). No childhood effects were observed for tobacco dependence or depression.

11. Importantly, these effects were independent of the children’s initial infant health. In these types of studies it is important to account for any potential ‘selection’ effects because children from poor families are more likely to begin life in poor health because fetal and birth complications are more prevalent in this group (Conley & Bennett, 2000; Power et al., 1998). In what was a novel contribution at the time, the authors also tested whether changes in socioeconomic circumstances after childhood could counteract the effects of childhood poverty. According to the upward mobility hypothesis, a rise in SES from childhood to adulthood should have a protective effect and result in improved health. The downward mobility hypothesis predicts that a drop in SES should have the opposite effect. According to the social-origins hypothesis, growing up in poverty will have enduring negative consequences, regardless of adult SES. The data were most consistent with the social-origins hypothesis underlining just how hard it is to undo or mitigate the long-term effects of childhood poverty.

12. This lifecourse study, spanning the age period from birth through to the mid-twenties, provides some of the most rigorous evidence to date that relative poverty can have long-lasting negative influences on adult health, irrespective of what health cache one begins life with, or where one ends up in the socioeconomic hierarchy as an adult. Whereas most studies of the long-term health effects of poverty have narrowed their attention to specific diseases, such as cardiovascular diseases, this study documented that the child poverty-poor adult outcomes relation is far more ubiquitous and troubling. Poverty adversely affected multiple areas of people’s health, including their physical, dental and behavioural health. Notwithstanding, these findings do not mean that adult social class is inconsequential for health. For example, depression and tobacco dependence were more strongly linked to adult SES than to childhood poverty suggesting that proximal experiences in adulthood better account for the association between poverty and these mental health disorders (e.g. Miech et al., 1998).

13. This study is important because it provided a strong test of the impact of childhood socioeconomic experiences on adult health, while ruling out potential alternative explanations. It also noted that further work was required to identify the key mechanisms that bring about these longitudinal associations. The authors pointed out that although lack of resources and/or structural impediments (e.g., lack of community facilities for physical activities) may play a role, the breadth of adult-health variables affected (ranging across physical health, dental health, and substance abuse) suggests several other
candidate mechanisms including: class-biased health-care delivery during children’s formative years; differences in health-promoting parenting practices (e.g. Taylor & Repetti, 1997); and poverty related stressors in childhood may alter biological systems and exert long-lasting influences on adult health (e.g. Brunner, 1997).

14. Some of these possibilities were examined in a subsequent study on the Dunedin cohort that sought to understand what risk factors might mediate this association (Melchior et al., 2007). The risk factors investigated included familial liability to poor health, childhood/adolescent health characteristics, low childhood IQ, exposure to childhood maltreatment and adult SES. Adult outcomes examined at age 32 included major depression, anxiety disorders, tobacco dependence, alcohol or drug dependence and clustering of cardiovascular risk factors (i.e. overweight, high blood pressure, low cardiorespiratory fitness, high total cholesterol and risk of diabetes). Results showed that childhood poverty was associated with more than a doubling of risk for substance dependence and poor physical health as adults. Interestingly, the risk factors (i.e., potential mediators) studied accounted for between one half and two-thirds of the poor health outcome at age 32 among those growing up poor. Perhaps more importantly, no single risk factor emerged as the key player, indicating that the risk pathways by which childhood poverty translates into poor adult health are like to be multifactorial.

15. In a complementary study, Gibbs and colleagues (2012) used the Christchurch cohort to examine linkages between childhood socioeconomic circumstances and later educational achievement, a series of economic outcomes, crime, mental health and teenage pregnancy. In their analysis they adjusted for a range of childhood correlates of poverty including individual, family and social factors that could confound the relation between poverty (i.e. here indexed as income), and poor outcomes in adulthood. The aim was to see if solely monetary policy solutions would be likely to mitigate adult risks associated with child poverty. As expected, low family income during childhood was associated with all outcomes in unadjusted analyses. Following adjustment for the range of poverty correlates, the relation remained for educational achievement and adult economic circumstances but became non-significant for crime, mental health and teenage pregnancy. In other words, relative poverty, as indexed by low family income, directly impacted upon educational achievement by age 30 years (attainment of high school qualifications, university attendance and degree attainment) and to economic circumstances (measured in four ways including gross personal annual income, welfare dependence, economic hardship and working in paid employment). There was no direct ‘causal’ association with mental health, crime or teenage pregnancy suggesting that the associations were either (i) indirect, being mediated by either individual childhood and/or family psychosocial circumstances, or (ii) were not real, resulting instead from confounding.
16. It should be emphasised that this study focused on family income, and other indicators that might reflect poverty were adjusted for at birth like parental education, family socioeconomic status and family type (single parent/two parent family).

17. Importantly the findings reported in these two New Zealand cohort studies show (i) that the longitudinal associations were not attributable to lifecourse continuity of low SES, and upward mobility did not mitigate or reverse the adverse effects of childhood poverty of adult health (Poulton et al., 2002); and that (ii) child poverty directly predicted levels on educational attainment and a range of important economic outcomes including employment (Gibbs et al., 2011).

How does poverty lead to poor outcomes: possible pathways and thus policy targets

18. The challenge is to explain the reasons for these associations, ideally by mapping the developmental pathways leading from childhood poverty to poor adult outcomes. Using adult physical health as the exemplar, is it fair to say that far less is known about mediating pathways than might be assumed, perhaps because most datasets documenting health inequalities have limited data to explore possible explanations for these inequalities (Adler & Snibbe, 2003). The most significant limitation has been the lack of longitudinal birth cohort studies that contain detailed life history data from birth to adulthood and direct physiological assessments of health. Up to this point, most empirical work has been limited by several weaknesses: (i) most research on the psychosocial antecedents of poor physical health has been cross-sectional, whereas prospective-longitudinal approaches and within-subject comparisons offer a much stronger strategy for understanding developmental influences. Among longitudinal studies that do exist, many have high attrition rates, selectively losing those in worse health, potentially biasing results. Third, associations between psychosocial risk factors (e.g. psychiatric symptoms) and health outcomes (e.g. obesity) often neglect to control for important confounding factors (e.g. medical illness). Fourth, and more generally, epidemiological studies with good health data tend to have weak psychosocial data, and vice versa. Gold standard measurement of psychosocial variables is paramount, as are direct measures of physical health uncontaminated by self-report biases.

19. The importance of the psychosocial stress pathway has been hotly contested (Lynch et al., 2000; Marmot & Wilkinson, 2001). Poulton & Caspi (2005) suggested three classes of candidate ‘stress’ mediators to illustrate the rationale, data, and methods required to strengthen tests of psychosocial mediation of the relation between childhood poverty and adult health – all of which have significant implications for policy and intervention targets.

20. First, psychologically stressful family environments can compromise homeostatic processes resulting in health problems in children (Evans, 2003). At least three areas are
important. The first involves poor mental health of parents which can interfere with their ability to care and provide for the child. The second area is particularly germane as it includes family circumstances associated with poverty in the household that promote stress (e.g. parental loss, multiple residential and/or caregiver changes). The third area involves family functioning and concerns the quality of parenting, especially the emotional aspects of the parent-child relationship, conflict, and abuse in the home. Data on all three domains are required to properly test whether stressful family environments mediate the influence of childhood poverty on adult health.

21. Second, early-onset psychiatric disorders and the personality vulnerability of high Negative Affectivity could mediate the effect of childhood poverty on adult health (Gallo & Matthews, 2003). Depression and hostility are two key psychological contributors to physical illness among adults. Research has shown that childhood poverty is related to early-onset and persistent cases of depression and conduct disorder (of which hostility is a core feature). Likewise, childhood poverty can impact on developing personality traits like Negative Affectivity (e.g. stress reactivity, hostility), and these traits can increase youth involvement in high-risk health behaviours (Caspi et al., 2005). Longitudinal data collected multiple times from childhood about psychiatric problems and personality traits are required to test whether these psychological vulnerabilities can explain, at least in part, the effect of childhood poverty on adult health.

22. Third, low social capital, including tenuous social support, could help explain the association between childhood SES and adult health. It is well recognised that high levels of social connectedness are linked to physical health (Berkman & Kawachi, 2000). Further, there is evidence that social support (both of the emotional and instrumental type) is patterned by social class, with those in poverty having the lowest levels. Lifecourse data are needed to test whether the lack of social capital and poor social integration during childhood mediates the effect of childhood poverty on adult health.

23. It is important to control for selection effects (i.e., differences in newborn health contributing to greater stress exposure) in these types of mediational analyses, as well as for other psychological factors associated with both SES and adult health; for example, differences in childhood IQ might account for some of variance in the social/health inequalities (Gottfredson, 2004).

**Pathways and policy implications**

24. The anticipated health burden associated with the aging population due to increases in age-related diseases like cardiovascular disease, cancer, type 2 diabetes and dementia highlights the importance of new and effective strategies to prevent age-related diseases, beginning much earlier in life. As will be clear by now, there are indeed preventable risk exposures in early life that contribute to, or exacerbate, pathological processes leading to
age-related disease (e.g. see Miller et al., 2011 for a contemporary model of how this might occur).

25. In trying to understand the policy implications of the child poverty-poor adult health relation, it is critical to integrate into a broader framework of childhood risk. This is because policy makers need to know how to evaluate sometimes competing claims about the importance of specific risk factors, all of which are “marketed: as being of high importance. Further, if risk factor redundancy exists, then intervening with only certain key risk factors will be most cost-effective, rather than having to target multiple risk factors.

26. In this regard, the Dunedin Study researchers recently reported findings showing that children experiencing poverty and/or maltreatment, and/or social isolation are more likely to have age-related-disease risks in adult life such as depression, inflammation, and clustering of metabolic risk factors (Danese et al., 2009). Importantly, the enduring consequences of adverse childhood experiences were not explained by established childhood, or concurrent adult risk factors.

27. These findings have several implications for policy makers: First, the data showed that the groups of children exposed to different adverse experiences do not necessarily overlap; for example, the majority of the children experiencing maltreatment or social isolation did not experience poverty. Put simply, different adverse childhood experiences exerted independent effects on age-related-disease risks. This suggests that different interventions might be needed to address specific adverse childhood experiences. Relieving childhood poverty alone may be insufficient, although it should remain a key part of a multi-pronged strategy.

28. Second, the results indicated that children exposed to more adverse experiences had more age-related-disease risks in adult life. The cumulative effect of adverse childhood experiences highlights new opportunities for disease prevention. Whereas social, political, and economic changes may take time to improve children’s socioeconomic conditions (Adler et al., 1993; Duncan and Brooks-Gunn, 2000), currently available interventions targeting childhood maltreatment (Olds et al., 1997) and social isolation (Bierman, 2004) can be rolled out more quickly. The corollary of the above is that if poverty is not properly addressed, the overall effectiveness of efforts to reduce the significant burden of age-related diseases will be lessened.

29. Third, the findings show that children who are exposed to adverse experiences appear to have enduring abnormalities across multiple biological systems. There was some specificity, consistent with earlier research showing that childhood poverty does not predict adult depression (e.g. Poulton et al., 2002; Gibb et al., 2012), and suggesting that childhood maltreatment does not predict metabolic risk marker clustering. Nonetheless, the general picture was that adverse childhood experiences may affect brain, immune,
and endocrine/metabolic functioning in adulthood. This evidence links early experimental findings in animals to humans (Levine et al., 1957, Soloman et al., 1968, Coe et al., 1989, Suomi, 1997; Miller et al., 2011) and supports the notion that adverse psychosocial experiences in childhood can disrupt a person’s physiological response to stress (Heim et al., 2000; Gunnar & Quevedo, 2007) and that chronic overactivation of the stress response system can result in damage in multiple stress-sensitive systems, namely, the nervous, immune, and endocrine systems, otherwise known as allostatic load (McEwen, 1998). The resulting cumulative stress burden could increase risk for age-related disease (Seeman et al., 2001).

30. Fourth, the findings suggest that children experiencing adversity are at risk of age-related-disease risks in adult life, independent of their familial liability for disease, birth weight, childhood weight, and adult socioeconomic status. This suggests that modifying these established risk factors is unlikely to wholly mitigate the economic health burden associated with adverse childhood experiences (Walker et al., 1999). Promoting healthy psychosocial experiences in childhood, with poverty reduction prime among them, will be necessary to improve quality of life and reduce health-care costs across the life course.

Conclusion

31. Incontrovertible evidence now exists showing that child poverty has long-lasting negative effects across multiple life domains. Newer research from cohort studies is beginning to map pathways between child poverty and poor adult outcomes. This is a boon to policymakers as this data can point to multiple ‘windows of opportunity’ for interventionists (e.g. see Shonkoff et al., 2012 for an example of ‘science to policy to practice’). Successful intervention will likely result in hugely favourable benefit-cost ratios when assessed across the life course, as well as conferring dual benefits to both this generation of children living in poverty, as well as to future generations by helping break cross-generational poverty cycles.

32. Very recent work from the New Zealand SoFIE cohort by Carter and Gunasekara (2012) provides a more nuanced picture of the nature and correlates of persistent versus short-term poverty experiences and how the timing of poverty exposure might moderate ultimate risks (see Rutter, 2012 for a discussion of individual differences and resilience in the face of adversity). Clearly any attempt to find workable, evidence-based solutions to child poverty is a challenging task, but one that has never been more tractable, thanks in part to important contributions from lifecourse studies such as those reviewed here.
References


