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Commentary

The successes and challenges of life course epidemiology: A commentary on Gibb, Fergusson and Horwood (2012)

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The successes of life course epidemiology

Smaller babies live shorter lives (Barker, Winter, Osmond, Margetts, & Simmonds, 1989). The quest to understand why has been a major factor contributing to the emergence of "life course epidemiology" as a sub-discipline in epidemiology. Life course epidemiology is concerned with trying to understand the extent to which long-term health and well-being are influenced by factors very early in the life course—including the prenatal period, the mechanisms accounting for these influences and whether or not pathways of risk can be avoided, and developmentally sensitive periods for health from childhood to adulthood (Barker, 2004; Ben-Shlomo & Kuh, 2002; Gluckman & Hanson, 2006; Glymour, Tzourio, & Dufouil, 2012; Kuh, Ben-Shlomo, Lynch, Hallqvist, & Power, 2003). Of course, developmentalists have been focusing for decades on the childhood environment as a source of risk and protection for adult health (Bronfenbrenner, 1975; Elder & Rockwell, 1979). Thus, it would be fair to say that epidemiologists "re-discovered" life course epidemiology.

The successes made to date in the field of life course epidemiology are owed almost entirely to the foresight of scientific pioneers who invested in long-term follow-up studies of children. One example is the Christchurch Health and Development Study, which has provided insights into health and development ranging from infancy to early childhood, adolescence, and young adulthood (Fergusson, Horwood, Shannon, & Taylor, 1978; Gibb, Fergusson, & Horwood, 2011). Others include the Dunedin Multidisciplinary Health and Development Study (Poulton et al., 2002), the British Birth Cohort Studies (Elliott & Shepherd, 2006; Power & Elliott, 2006), the Avon Longitudinal Study of Pregnancy and Childhood (Baker & Taylor, 1997), and the Collaborative Perinatal Project (Buka, Tsuang, & Lipsitt, 1993; Hardy et al., 1997). These studies and others like them lead to major advances in our understanding of risk and protective factors for disease across developmental stages. They have also evolved with time by incorporating advances in genetics (Fergusson, Horwood, Miller, & Kennedy, 2011), by using previously stored samples to test novel hypotheses (Buka, Cannon, Torrey, & Yolken, 2008), and by expanding to new generations of participants (Gilman et al., 2009), all of which have enhanced the value of these cohorts beyond what could have been conceived at the time of each study's inception. It is both important and timely to reflect on the enormous gains in knowledge these studies have produced, particularly in light of current struggles over the United States National Children's Study, which are testing our scientific and financial resolve to make continued large-scale investments in the research infrastructure for the next generation (Kaiser, 2012).

One of the major substantive advances made by long-term follow-up studies of children has been in the area of health inequalities (Barker & Osmond, 1987). This work has demonstrated that inequalities in physical and mental health originate in childhood and strengthen with subsequent exposure to socioeconomic

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disadvantage during adulthood (Gilman, Abrams, & Buka, 2003; Gilman, Kawachi, Fitzmaurice, & Buka, 2002; Loucks et al., 2012, 2010; Melchior, Moffitt, Milne, Poulton, & Caspi, 2007). The implications for public health policy are substantial: efforts to reduce health inequalities must halt the emergence of inequalities in the earliest years of life (Bartley, Blane, & Montgomery, 1997; Knudsen, Heckman, Cameron, & Shonkoff, 2006; Lynch, Kaplan, & Salonen, 1997). Gibb et al. provide new evidence from the Christchurch study that children's socioeconomic conditions have long-lasting influences on adult well-being, and leverage data collected over the study's 30-year follow-up period to address the question: *would changing family income during a child's first ten years of life lead to improvements in educational, economic, behavioral (e.g., crime, teen pregnancy), and mental health outcomes at age 30* (Gibb, Fergusson, & Horwood, 2012)? They conclude that it would, for educational and economic outcomes, but it might not, for behavioral and mental health outcomes, given potential confounding by social, familial, and contextual factors associated with income.

While there is considerable evidence to support the effectiveness of educational interventions to improve the outcomes of low-income children (Heckman, Moon, Pinto, Savelyev, & Yavitz, 2010; Leventhal & Brooks-Gunn, 2004), there is limited experimental evidence on the causal effects of raising family income itself. Therefore, Gibb et al.'s study is important because of the wealth of data that is available in the Christchurch cohort to adjust for potential confounding factors, and thus generate an approximate estimate (to the extent possible with observational data) of the effect of an income intervention. Their findings are partly consistent with those of Costello et al.'s study that used a quasi-experimental design to estimate the effects on children's mental health of an intervention that raised family incomes (Costello, Compton, Keeler, & Angold, 2003). Costello et al. found that exposure to an income intervention lead to improvements in children's symptoms of oppositional defiant disorder and conduct disorder (in contrast to Gibb et al.'s findings regarding offending a teenage pregnancy), but had no effects on children's symptoms of anxiety or depression (similar to Gibb et al.'s findings). Gibb et al.'s results are also similar to those of Duncan et al., who found that interventions that raised family income lead to improvements in children's academic performance (Duncan, Morris, & Rodrigues, 2011). Combined with data from many observational studies linking family income to children's health and development, the available evidence suggests that raising family income will have long-term benefits for children; however, the evidence is inconsistent regarding the specific types of benefits that would be expected from family income interventions. Gibb et al.'s study highlights important challenges in identifying the effects of early childhood interventions from observational data and, more generally, in applying the conceptual frameworks of life course epidemiology.

The challenges of life course epidemiology

The conceptual basis of life course epidemiology derives from generic risk factor models—latency, pathway, accumulation—that serve as heuristics for the ways in which exposures at different point in time influence subsequent health (Ben-Shlomo & Kuh, 2002; Kuh et al., 2003). These heuristics have been influential in motivating and framing investigations into the timing, duration, and accumulation of risks (e.g., Caspi, Harrington, Moffitt, Milne, & Poulton, 2006; Fergusson, Horwood, Boden, & Jenkin, 2007; Gilman, Abrams, et al., 2003; Gilman, Kawachi, Fitzmaurice, & Buka, 2003a). Admittedly, these generic models of life course epidemiology tend to over-simplify complex processes which are often contingent upon one another and as a result unfold in tandem over time, making it challenging to disentangle discrete pathways of risk. Nonetheless these models remain useful, as their application to life course data should enable researchers to identify modifiable risk factors, and determine when during development interventions are most effective.

Gibb et al. analyzed quintiles of average family income over a period of 10 years and found that it significantly predicted outcomes at age 30. What is the hypothetical intervention that corresponds to the (assume for the sake of argument, causal) effects they identified (Hernan, 2005)? Most intuitively, it would be an intervention that leads to a sustained increase in family income throughout infancy and early childhood. If Gibb et al.'s findings are correct, the implication is that family income interventions need to be long-term interventions. Though Gibb et al. do not report how much variation existed in incomes across the study's annual assessments during the first 10 years of life, I assume there was substantial correlation between participants' serial reports of family income, as well as the relative ranking of family incomes over time. Still, a range of different patterns of income over time could have given rise to the same *average* level of income during the 10-year exposure period (e.g., participants would fall in the middle quintile if their incomes were in the middle of the distribution over the entire period, as well as if their incomes were at the lowest and highest ends of the distribution for half of the time). Therefore, Gibb et al.'s results are consistent with a range of hypothetical interventions. Sensitive period and accumulation models could also be evaluated with the Christchurch data to determine whether the long-term effects of family income are strongest at any specific age, and whether they depend on children's cumulative exposure to disadvantage (e.g., number of years exposed to low income); it may also be important to determine the long-term effects of *changes* in family income during the first 10 years of life (consistent with the evidence of adverse consequences associated with frequent changes in residence during childhood (Gilman et al., 2003a; Qin, Mortensen, & Pedersen, 2009; Wood, Halfon, Scarlata, Newacheck, & Nessim, 1993)).

The wealth of available information in the Christchurch cohort made it possible for Gibb et al. to examine the association between family income during childhood and adult outcomes controlling for numerous individual and familial risk factors associated with income. Gibb et al. considered two broad classes of covariates: background covariates that were assessed at the time of each participant's birth, and covariates assessed concurrently with the assessment of income from birth to age 10 (detailed in their Appendix, and depicted here in Figs. 1 and 2). Recall that the causal question of interest is the effect of a hypothetical intervention on family income during childhood on the change in offspring's outcomes at age 30. Do the statistical models used by Gibb et al. allow us to identify this causal effect?

Fig. 1 depicts a causal diagram for the effect of family income during childhood on offspring outcomes considering only background covariates. The solid arrows linking the background covariates with family income and outcomes at age 30 represent that the background covariates are prior common causes of both income and offspring outcomes, are therefore potential confounders, and should therefore be controlled for in a statistical analysis. A statistical model corresponding to Fig. 1 would be a regression of age 30 outcomes on family income and background covariates. Assuming the model is correctly specified (e.g., the correct functional forms of covariates are used), it would provide an unbiased estimate of the causal effect of income if there were no unmeasured confounders (the effects of which are depicted by the dashed lines). Of course with observational data one can never guarantee the absence of unmeasured confounders; however, the specific covariates adjusted for likely include the most important potential confounders of the effects of income, and decades of publications