International Seminar on

Pathways to Health:
How intermediary life conditions mediate or modify early life effects

Berkeley, CA (USA), 1-2 May 2012

Organized by and with support from:
IUSSP Scientific Panel on Pathways to Health
Berkeley Population Center, and
Institute for the Study of Societal Issues, University of California, Berkeley

REPORT

This report was written by Mikko Myrskylä (Panel Chair), Tommy Bengtsson and Alain Gagnon (Panel Members)

Short summary:
Adverse early life conditions have lasting effects on old-age health and mortality. Exposure to disease, nutritional deprivation, or adverse socioeconomic conditions early in life, as early as in the foetal stage, have all been linked to decreased adult health or increased mortality. Evidence suggests that the association between these and other adverse early life conditions with later health may be causal. Often, however, the mechanism through which early life conditions are linked with later life health is unclear. In particular, it is not well understood whether negative early life insults affect health directly through damage to cells and organs, indirectly through the ability to accumulate human capital, or through yet other pathways. However, a thorough understanding of the mechanism through which early life conditions influence adult health is critical for the development of policy interventions aiming to improve population health by attenuating the impact of adverse early life conditions and exposures.

The IUSSP Seminar "Pathways to Health: How intermediary life events and conditions mediate or modify the early life effects" focused on the mechanism through which adverse early life conditions influence adult and old-age health and mortality. The Seminar included 13 presentations and 22 participants mostly from the academia, with backgrounds in demography, economics, anthropology, epidemiology, sociology, and public health. The presentations addressed the pathways that link early life conditions and adult outcomes from various complementary perspectives. Several papers found support for the indirect mechanism that postulates that adverse early life conditions influence later health mainly through compromised adult socioeconomic attainment. These findings are important for the understanding of various pathways to health and for the planning of interventions, in particular because education, which is amenable by policy, often emerged as a strong mediator. However, several seminar papers also found that the association between early life conditions and adult health is robust to adult characteristics, thus supporting a direct rather than an indirect link. Further work is needed to better understand the conditions under which adverse early life conditions are likely to result in direct scarring of health, and the conditions under which the influence on adult health might run through modifiable, potentially socioeconomic, pathways.

Introduction

Adverse early life conditions have lasting effects on old-age health and mortality (Almond 2006; Bengtsson and Lindstrom 2003; Gagnon and Bohnert 2012; Mazumder et al. 2010; Myrskylä 2010; Roseboom, de Rooij and Painter 2006; Schulz 2010; van den Berg, Doblhammer and Christensen 2009), and some even consider reductions in early life disease exposure to be a primary driver of historical mortality declines (Finch and Crimmins 2004). Consequently, understanding the complex relationship between early life conditions and adult health and mortality, and in particular the mechanism through which these are linked, has become an area of concern in the development of strategies for population health and successful aging.

Prior research shows that exposure to disease, nutritional deprivation, or adverse economic conditions early in life, possibly as early as in the foetal stage, are all linked to decreased adult health or increased mortality. Evidence from natural experiments suggests that the association between these and other adverse early life conditions and later health may be causal. Although numerous pathways have been postulated including those relating to foetal under-nutrition, dysregulation of the immune function, and compromised socioeconomic attainment (Costa 2000; Gluckman et al. 2008; Schulz 2010), the mechanism remains unclear.

The aim of the IUSSP Seminar "Pathways to Health: How intermediary life events and conditions mediate or modify the early life effects " was to shed light on the mechanisms through which early life events and conditions influence adult and old-age health and mortality. From the policy perspective, the fundamental question is whether adverse early life conditions affect later health directly through damage to cells and organs, indirectly through the ability to accumulate human capital, or through yet other pathways. In particular, for the planning of effective interventions aiming to improve population health through attenuating the impact of adverse early life conditions and exposures, it is critical to understand the mechanisms.

Earlier literature identifies three key early life exposures that may be particularly important for later health, and two different frameworks that help conceptualize the mechanisms through which early life exposures may be related to later health. Of the various early life exposures the three chief factors influencing later health appear to be nutritional deprivation (Barker 1994), exposure to disease (Bengtsson and Lindstrom 2003; Bengtsson and Lindström 2000; Crimmins and Finch 2006), and socioeconomic adversity (Hayward and Gorman 2004; Smith 1997). For each of these exposures there is strong associational evidence and also evidence based on exogenous environmental shocks suggesting that they are causally linked to later health; however, much less is known about the ways these factors act. The IUSSP Pathways to Health Seminar papers covered each of these three key exposures, and addressed the mechanisms through which the exposures are linked to adult- and old-age health. In doing so, the papers take advantage of two complementary conceptual frameworks for understanding the early life exposure-later life health mechanisms. The first framework developed by Preston et al. (1998) considers direct physiological versus indirect associational mechanism which both may result in either positive or negative associations between early life conditions and later life health. The second framework introduced by Ben-Shlomo and Kuh (2002) discusses critical versus sensitive exposure periods and various ways in which health risks may accumulate over the life course. Collectively, the seminar papers shed light on most of the dimensions of these two frameworks.

Key exposures: Nutritional deprivation, disease exposure, and socioeconomic adversity

Among the various early life exposures, earlier literature has identified three that are among the chief factors influencing later health: nutritional deprivation, exposure to disease, and socioeconomic adversity. Inadequate nutrition *in utero* may result in physiological and metabolic restrictions that increase the risk of cardiovascular disease mortality in later life (Barker 2006; Cameron and Demerath 2002). Prenatal nutritional deprivation has also been connected to higher prevalence of death due to infectious diseases in adolescence in a series of contemporary African studies (McDade et al. 2001; Moore et al. 1999; Moore et al. 1997), and Fogel (Fogel 2004) and Costa and Lahey (Costa and Lahey 2005) attribute much of the secular declines in old age mortality to improved early life nutrition.

Exposure to infectious disease early in life may also cause damage that influences adult health and mortality. Two English studies demonstrated that exposure to airborne infectious diseases at a young age is associated with cough, phlegm, and impaired ventilatory function later in life (Barker et al. 1991; Shaheen et al. 1994). In 18^{th}-19^{th} century Sweden, an analysis based on longitudinal individual level data shows that individuals born during years of smallpox and whooping cough epidemics had an increased risk of death after age 50 (Bengtsson and Lindstrom 2003). Almond and Mazumder (2005) found that in utero exposure to the 1918 pandemic was associated with decreased self-reported health of adults over 50. Exposure to the 1918 Pandemic in late stages of foetal development also appears to influence educational and job-market outcomes and cardiovascular disease prevalence (Almond 2006; Mazumder et al. 2010). These results are consistent with the hypothesis that reductions in infectious diseases exposure (and thus inflammation) made a major contribution to the historical decline in old-age mortality (Crimmins and Finch 2006; Finch and Crimmins 2004).

Early life family characteristics and socioeconomic conditions have also been shown to be important predictors of later health. Several studies have documented strong associations between childhood SES, measured by father's occupation and/or parental education, housing characteristics and/or family income and various adult health outcomes and mortality (Hayward and Gorman 2004; Preston et al. 1998; Strand and Kunst 2007). Analyses exploiting exogenous macro variation in socioeconomic conditions, typically fluctuations in per capita gross domestic product, are consistent with the individual-level studies. For example, van den Berg and colleagues show that being born in a recession, compared to being born in a period of economic growth, increases all-cause and in particular cardiovascular disease mortality (van den Berg, Lindeboom and Portrait 2006; van den Berg et al. 2009).

Many of the aforementioned studies pay particular attention to identifying a causal association between early life conditions and later life health. According to Ben-Shlomo and Kuh (2002), however, the aim of life course epidemiology goes beyond correlating – possibly causally – intrauterine and childhood circumstances to later health by building and testing models that test for pathways linking later life health with exposures across the whole life course. The IUSSP Pathways Seminar papers considered each of the three key exposures – nutrition, disease exposure, and family characteristics and socioeconomic conditions – and analyzed how they are associated with later life health, with focus on whether the associations are mediated, modified or buffered by intermediate characteristics such as own socioeconomic attainment at young adult ages.

Pathways and mechanisms

The fundamental questions regarding the early life-later life mechanism are whether the mechanism is direct or indirect, and if indirect, whether the pathway runs through modifiable factors which could be influenced by policy interventions. The conceptual frameworks for understanding these processes were developed by Preston et al. (1998) and Ben-Shlomo and Kuh (2002).

Indirect physiological versus indirect associational mechanisms

Preston et al. (1998) discuss the difference between individual and population level associations, and set up a framework that allows distinguishing between direct physiological and indirect associational pathways by presenting a typology of four mechanisms that may relate early life conditions to adult and old-age health. These mechanisms are (1) scarring, which is positive and direct/physiological, (2) acquired immunity, which is inverse and direct/physiological, (3) correlated environments, which is positive and indirect/associational, and (4) selection, which is inverse and indirect/associational.

An example of the direct and physiological scarring effect is the hypothesized effect of low birth weight and growth retardation in childhood on cardiovascular diseases and diabetes (Barker 1992). A direct, but inverse physiological effect may arise through acquired immunity. For example, exposure to infectious diseases early in life may give protection against similar diseases in old age. Both direct mechanisms operate at the individual level. Among the indirect mechanisms, the "correlated environments" refers to a process in which those who are born into (dis)advantaged socioeconomic circumstances retain some of those (dis)advantages throughout life into adult ages (Mare 1990). These adult characteristics are often important for later life health, therefore a positive association between early life conditions and later health arises. Another indirect mechanism linking early life conditions to later life health is selection in which only the more robust individuals survive to older ages. Such a population level process may result in an inverse association between early life conditions and later mortality, or attenuate the otherwise positive association (Gagnon and Bohnert 2012).

Critical and sensitive periods, and risk accumulation

A complementary framework which distinguishes between critical and sensitive exposure periods as well as between different kinds of risk accumulation processes was developed by Ben-Shlomo and Kuh (Ben-Shlomo and Kuh 2002). For critical periods, exposure must take place during a specific window in order to have a lasting effect; for exposures outside the critical window there are no long-term consequences. An example of the critical period model is the fetal origins of adult disease hypothesis (Barker 1992). Exposure outside what is called a sensitive period may have a long-term impact, although weaker; the studies focusing on inflammation in the first years of life (Finch and Crimmins 2004) fall into this category.

In the framework of Ben-Shlomo and Kuh (2002), the pathway of an exposure during a critical or sensitive period that has a direct effect corresponds to scarring or selection in the Preston et al. (1998) conceptualization. The aspects that extend the framework of Ben-Shlomo and Kuh beyond the Preston et al. framework are the consideration of later-life modifiers and different kinds of accumulation of risks. These are critical for understanding the pathways linking early and later life. For example, a scarring effect that is direct and physiological could potentially be modified by later life environment. An example is the impact of adverse

early life conditions on later cardiovascular disease mortality that may be mitigated or even eliminated by medical treatment. Within the accumulation of risks framework, which corresponds closely to the Preston et al. correlated environments pathway, Ben-Shlomo and Kuh consider both correlated and independent risks. An important special case of correlated risks is the "chains of risk" model with a "trigger effect". This model postulates that the initial exposure leads to a sequence of linked exposures in which only the final link in the chain has a marked effect on disease risk. An example of such a process is the effect of adverse early life socioeconomic conditions, which may be related to adult mortality mostly through a chain that connects the early life circumstances to the trigger adult socioeconomic status.

The Berkeley Seminar and the presented papers

To explore the pathways through which early life conditions influence later life health and mortality we organized an international meeting entitled "Pathways to Health: How intermediary life events and conditions mediate or modify the early life effects" in May 1-2, 2012 at the University of California, Berkeley. The meeting, jointly organized by the International Union for the Scientific Study of Population, the Berkeley Population Center, and the Institute for the Study of Societal Issues at Berkeley, brought together a multi-disciplinary group of investigators with interests in demography, epidemiology, and public health.

The studies focused on the pathway that links early-life conditions to health, mortality and other outcomes in adult- and old-ages. The 13 Seminar papers highlighted several dimensions of the early life-later health association and the mechanism. Instead of discussing each paper individually, this report summarizes some key themes of the papers. Most studies used high-quality individual-level micro data that includes either a longitudinal follow-up from early life to old-age, or include macro-level characteristics of the early life conditions as instruments for the early life conditions.

The papers cover the three key early life exposures – nutrition, disease, and family/socioeconomic circumstances – and address the mechanisms through which they are linked to adult- and old-age outcomes by taking advantage of the frameworks developed by Preston et al. (1998) and Ben-Shlomo and Kuh (2002). Of the direct pathways, the vast majority of the papers address the scarring mechanism, and some provided evidence that also the selection mechanism is at work. Of the indirect pathways, a particularly important model is the indirect correlated environments model (Preston et al. 1998) and its special case "chains of risk" with a "trigger effect" (Ben-Shlomo and Kuh 2002). A canonical example of this model is a situation in which adverse early life conditions increase adult mortality through lower adult socioeconomic attainment. Several of the Seminar papers considered whether the effect of the early life conditions on later life outcomes is mediated by adult education or other dimensions of socioeconomic attainment. The papers find support for the indirect mechanism that postulates that adverse early life conditions influence later health mainly through compromised adult socioeconomic attainment. The papers also test alternative mediating mechanisms, such as adult height and cognitive ability, and timing of parental death. These findings highlighting the importance of later-life mediating factors are important for the understanding of the mechanism and for the planning of interventions, in particular because education, which is amenable by policy, often emerged as a strong mediator. However, several seminar papers also found that the association between early life conditions and adult health is robust to adult characteristics, thus supporting a direct rather than an indirect mechanism.

The majority of the papers use analytical designs which allow conditional causal inference regarding the effect of the early life exposure. Approximately half of the papers exploited exogenous variation in the macro environment to first establish a causal link between early life conditions and later outcomes, and then proceeded to further analyze the pathway through which the causal effect runs. A few papers use individual-level techniques that allow causal inference via the elimination of unobserved confounders, and most other papers use extensive controls for early life characteristics to reduce the risk of unobserved confounding.

Several of the Seminar papers considered effect heterogeneity. It is possible that in favorable contexts, for example in a high-SES versus low-SES family, the impact of adverse early life conditions such as disease exposure is less harmful. The seminar papers that considered effect heterogeneity generally find evidence for such buffering, highlighting the importance of the local context in pathway linking early life conditions to later life outcomes.

The papers also shed light on the critical and sensitive periods framework. The important question regarding critical versus sensitive periods is not whether such periods exist, but rather what are these periods for any given exposure. Several seminar papers provided new insights into this discussion by challenging the "fetal origins of adult disease paradigm" by showing that at least for certain exposures, the first year(s) of life may be more important than the in utero period.

Conclusions

The papers presented in this seminar illustrate the pathways through which adverse early life conditions influence later life health and well-being. The issue is of great importance as a thorough understanding of the mechanism is critical for the development of policy interventions aiming to improve population health and successful aging by attenuating the impact of adverse early life conditions and exposures. Using both historical and contemporary micro-level longitudinal data sets which contain information on early life characteristics, on later-life mediating factors, and on older-age outcomes, the papers cover various early life exposures such as nutritional deprivation, exposure to disease, and socioeconomic adversity, and analyze the mechanisms that link these exposures to later outcomes from various complementary perspectives. The overall picture is rich and complex. While the studies confirm the importance of the life-course perspective by adding to the evidence showing that adverse early life events and conditions have long-lasting negative impact on adult health and well-being, the evidence regarding the pathways varied. Several papers find support for the indirect mechanism that postulates that adverse early life conditions influence later health mainly through compromised adult socioeconomic attainment. While these findings are important for the understanding of the mechanism and for the planning of interventions, still several seminar papers also found that the association between early life conditions and adult health is robust to a range of adult characteristics including socioeconomic attainment, thus supporting a direct rather than an indirect mechanism.

The findings of the papers highlight the importance of education and socioeconomic status in two complementary ways. First, the papers documenting that the impact of adverse early life conditions runs through adult socioeconomic attainment are important for the understanding of the mechanism and for the planning of interventions, in particular because education, which is amenable by policy, often emerged as a strong mediator. Second, many of the papers that documented heterogeneity in the effect of adverse early life events on later life outcomes

found that the magnitude of the adverse effect was mitigated if the parents were of higher socioeconomic status. This finding further stresses the importance of education and resources in health over the life course. Although education is clearly important, it appears not to be a universal fix, as several of the papers presented in the Pathways Seminar also found that the impact of adverse early life conditions on later life outcomes was robust to adult socioeconomic attainment.

We consider it important that future work continues to move beyond documenting the associations between early life exposures and later life outcomes by directly addressing the potential mechanisms. In particular, more work is needed to better understand the conditions under which adverse early life conditions are likely to results in direct scarring of health, and the conditions under which the influence on adult health might run through modifiable, potentially socioeconomic, pathways. Analyzing the mechanisms and testing the hypothesis separating direct versus indirect pathways puts heavy constraints on the data as the analysis of the mechanism often requires measurements at least in three points in time. However, longitudinal data sets that meet these requirements are becoming increasingly more available, extending the possibilities of inquiry.

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الاتحاد الدولي للدراسات العلمية للسكان

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Programme

Monday, April 30, 2012

Welcome dinner / Angeline's Louisiana Kitchen	6 PM

Tuesday, May 1, 2012

Welcome and Introduction (Myrskylä, Gagnon, Bengtsson)	9:00-9:15AM
1. Alain Gagnon, Université de Montréal, Canada *"Fertility mediates the impact of birth season on longevity in a natural fertility population"*	9:15-10:00AM
2. Irma T. Elo, University of Pennsylvania, Philadelphia, USA, Pekka Martikainen, University of Helsinki, Finland, Mikko Myrskylä, MPIDR Rostock, Germany *"Early- and Later Life Family and Socioeconomic Conditions and Cause-Specific Mortality in Finland"*	10:00-10:45AM
Coffee Break	10:45-11:15AM
3. Thomas Fritze, University of Rostock, Germany, Doblhammer-Reiter Gabriele, University of Rostock, Germany Gerard van den Berg, University of Mannheim, Germany *"Effects of Earliest Life and Childhood Circumstances on Cognitive Functioning at Old Age: A Life-Course Perspective Based on the First Three Waves of SHARE"*	11:15-12:00PM
Lunch Break	12:00-1:15PM
4. Ken Robert Smith, University of Utah, Salt Lake City, USA *"Perishing Parents and Persevering Progeny: Effects of the Timing of Parental Death and Remarriage on Adult Offspring Mortality"*	1:15-2:00PM
5. Jennifer Karas Montes, Harvard University, Cambridge, USA Mark D. Hayward, University of Texas, Austin, USA Chi-Tsun Chiu, University of Texas, Austin, USA *"Cumulative Childhood Adversity and Active Life Expectancy among U.S. Adults"*	2:00-2:45PM
Coffee Break	2:45-3.15PM

6. Lambert H. Lumey, Columbia University, New York, USA Peter Ekamper, NIDI, The Hague, The Netherlands Aryeh D. Stein, Emory University, Atlanta, USA Frans van Poppel, NIDI, The Hague, The Netherlands *" Social class and education at age 18 as possible mediators of the relation between prenatal famine and adult mortality"*	3:15-4:00PM
7. Bruna Galobardes, University of Bristol, UK S. Patel, University of Bristol, UK R. Granell, University of Bristol, UK G. Davey Smith, University of Bristol, UK J. Sterne, University of Bristol, UK J. Henderson, University of Bristol, UK *"Understanding the life course socioeconomic patterning of asthma provides clues about the exposures and pathways leading to the disease"*	4:00-4:45PM
Dinner	6 PM

Wednesday, May 2, 2012

Presentations	
8. Mikko Myrskylä, MPIDR, Rostock, Germany *"Adverse early life conditions and decreased cardiovascular disease mortality: evidence for selection among Finnish 20th century cohorts"*	9:15-10:00AM
9. Tommy Bengtsson, Lund University, Sweden Jonas Helgertz, Lund University, Sweden *"Effects of early life exposure to infectious diseases on occupational skills - the case of the Spanish flu"*	10:00-10:45AM
Coffee Break	10:45-11:15AM
10. Christopher W. Kuzawa, Northwestern University, Evanston, USA *"Epigenetics, Intergenerational Inertia, and Human Adaptation: Hypotheses and Policy Implications"*	11:15-12:00PM
Lunch Break	12:00-1:15PM
11. George Ploubidis, London School of Hygiene and Tropical Medicine, UK Emily Grundy, London School of Hygiene and Tropical Medicine, UK Mike Kenward, London School of Hygiene and Tropical Medicine, UK Rhian Daniel, London School of Hygiene and Tropical Medicine, UK Bianca de Stavola, London School of Hygiene and Tropical Medicine, UK *"Lifelong Socio Economic Position and biomarkers of later life health: A formal comparison of the critical period, accumulation and chains of risk hypotheses"*	1:15-2:00PM
Coffee Break	2:00-2:45PM
12. Jonas Helgertz, Lund University, Sweden Mats Persson, Lund University, Sweden *"Early Life Conditions and Sickness Absence During Adulthood – A Longitudinal Study of 15,000 Siblings in Sweden"*	2:45-3:15PM
13. Joseph P. Ferrie, Northwestern University, Evanston, USA Karen Rolf, University of Nebraska at Omaha, USA *"The Role of Mid-Life Conditions in Mediating the Link Between Early-Life Circumstances & Late-Life Outcomes: Linked Birth, Census, Military, and Death Records for the U.S., 1915-2005"*	3:15-4:00PM
End of the Seminar (Myrskylä, Gagnon, Bengtsson)	4:00PM

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