Extended abstract for EPC 2008

Early Life Conditions and Mortality Later in Life in Scania, Sweden 1814-1894: New Ways of Measuring Conditions During Early Life.

Kent Johansson

&

Jan Beise

It has recently often been claimed that our longevity – at least partly – is determined already during intra-uterine life or during infancy.[1] The idea of early life conditions affecting later life health is not new, and earlier studies based on aggregated data have shown such a relationship during the general mortality decline.[2] However, data on contemporary populations for testing this hypothesis is hard to find, but recent studies of historical populations have established such links with family reconstituted data,[3] where the relationship is formally tested with Cox-regressions based on life-histories of real individuals. This is a huge improvement compared to the tabulations methods used in the early studies. Generally, the use of individual data has several advantages over using aggregated data. The nature of micro data makes it possible to test hypotheses using multivariate statistical tools that allow control for individual characteristics as for example sex, birth-place, family-specific characteristics, and socio-economic group simultaneously in an empirical model. However, even if the life-histories of individuals are known, the conditions in early life *per se* are not known. Nutrition intake and disease histories are usually unknown even in modern data compilations. Hence, in most cases early life conditions has to be measured by some proxy variable or variables, approximating nutrition and/or disease load. This paper discusses such measures, and their advantages/disadvantages, and how to improve them. A major issue dealt with here is that many studies have been able to establish a link between disease in infancy and later life mortality – what could be called the Fridlizius link from early life conditions to health in later life,[4] but have failed to establish the Barker link from nutrition in the foetal stage to health in later life.[5] In this paper, some new and improved measures for early life conditions are suggested and discussed relative to what has been commonly have been used. The suggested improved measures are both supposed to be theoretical and practical improvements compared to earlier measures. Further, one measure approximates foetal stage nutrition and disease effects on later life mortality; thus, the "true" Barker hypthesis.[6]

Some commonly used measures for early life conditions

One measure commonly used is birth season, and this is supposed to be a combined proxy measure of nutrition and disease load during early life. This method has been used as the sole measure of early life conditions in some studies,[7] but also controlling for birth season while measuring conditions in early life with other proxies.[8] A drawback with the birth season measure is that it is a summary measure – it does not measure any direct link from early life conditions to later life health or mortality, but is rather a summary of everything related to the season/month of birth connected to later life health, just as place of birth and antropomethric

[1] Barker (1998:5-41, 2001:69-88).
[2] Kermack, McKendrick & McKinlay (1934:702-703), Preston & van de Walle (1978:290-291), Fridlizius (1989:16-17).
[3] Bengtsson (1997:15-19), Bengtsson & Lindström (2000:273-275, 2001:10), Alter & Oris (2000:PAGES) Bengtsson, Broström & Lindström (2002:1-4, 20-24), Johansson (2004:207-212), Johansson, Beise & Desjardins (2006).
[4] Fridlizius (1989:PAGES), Barker (1998:Chapter 9).
[5] Barker (1998:PAGES).
[6] Measuring of early life condition effects was one of the topics that should be more thoroughly investigated in reaseach on early life conditions and later life mortality, as recognized by the commentator Gabriele Doblhammer at the "Early Life Experiences and Mortality" session at PAA 2006.
[7] Doblhammer (1999:4-7), Gavrilov & Gavrilova (1999:365-366), Doblhammer & Vaupel (2001:2938-2939), Gavrilova, Gavrilov, Evdokushkina, & Semyonova (2001:2, 10-13), Doblhammer (2002:17-20).
[8] Bengtsson & Lindström (2001:5, 24), Bengtsson, Broström & Lindström (2002:20-23), Johansson (2004:207-212).

measures as adult height.[9] To avoid the summary measure problem, a more direct method of measuring the link between early life conditions and later life health or mortality should be preferred.[10]

One frequently used method is based on a model that combines individual data with aggregated data on community food prices; [11] *i e*, what can be called a combined micro and macro analysis model. It uses data on community aggregates as fixed food prices during early life as a proxy for nutrition intake in early life.[12] A natural extension of this method is to use local mortality rate as a proxy for disease load. Several investigations have used this approach for measuring the connections between early life disease load conditions and mortality later in life.[13] The method uses individual life histories that could for example be based on church record family reconstitutions. In a survival regression framework, this means that this fixed covariate can be used to test if there is a significant relationship between the price of food during infancy and mortality later in life; thus, testing an extra-uterine conditions variant of the Barker hypothesis.[14] In the same way it is possible to test the infancy disease load early life condition hypothesis, as has for example been suggested by Fridlizius.[15] The standard way of approximating infancy disease load is by the use of local infant mortality rates.[16] It is reasonable to assume that local infancy mortality rates gives an indication of how strong the disease load is if we are considering the conditions before the age of sulpha/antibiotics; thus, before WWII. If infants were exposed to many infectious diseases and without any cure for these diseases (thus; antibiotics), many infants would die since no cure was available, making the infant mortality rate a good indicator of the general local disease load.

A further extension of both the food price proxy for nutrition intake and the infant mortality rate proxy for disease load is to use these for approximating foetal stage conditions. This can be made by assuming that foetal nutrition intake can be approximated by the price of food during pregnancy, since maternal nutritional intake should be affected by food prices; at least in a historical context.[17] This would then be a test of the Barker foetal origin of later life disease hypothesis.[18] In the same way, it is possible to use local mortality rates for foetal stage exposure to disease. Regarding such exposure, any foetal disease load must be argued to stem from the mother.[19] Thus, it seems quite straightforward to use local female reproductive age mortality rate as a proxy for foetal stage disease load. It can however be hard to use the sex-specific mortality rates since mortality in – say – the age span 15 to 45 is rather low even in

[9] Forsdahl (2002: 304-307, reprint; originally published in 1977), Barker & Osmond (1986a, 1986b, 1987), Barker, Osmond, Golding, Kuh & Wadsworth (1989), Osmond, Barker & Slattery (1990, Nyström-Peck (1994:PAGES), Alter & Oris (2000b:PAGES), Edvinsson (2001:252-265).

[10] Johansson (2004:PAGES).

[11] Bengtsson (1993:239-258).

[12] Bengtsson & Lindström (2000:270-276), Bengtsson & Lindström (2001:24-27), Bengtsson, Broström & Lindström (2002:14-15, 20), Johansson (2004:130-134).

[13] Bengtsson (1997:16-19), Bengtsson & Lindström (2000:274-275), Bengtsson & Lindström (2001:9-13), Bengtsson, Broström & Lindström (2002:11, 20-23), Johansson (2004:134-139), Johansson, Beise & Desjardins (2006).

[14] Often used as deviation from a "normal" price estimated by the Hodrick-Prescott short-term-trend – to get realtive prices comparable over time; see Johansson (2004:PAGES) for a discussion.

[15] Fridlizius (1989:PAGES), Barker (1998:Chapter 9)

[16] Bengtsson (1997:16-19), Bengtsson & Lindström (2000:274-275), Bengtsson & Lindström (2001:9-13), Bengtsson, Broström & Lindström (2002:11, 20-23), Johansson (2004:134-139).

[17] Bengtsson & Lindström (2000:270-276), Bengtsson & Lindström (2001:24-27), Bengtsson, Broström & Lindström (2002:14-15, 20), Johansson (2004:130-134).

[18] Barker (1998:PAGES)

[19] Johansson (2004:134-136).

historical populations, which can induce problems with too few cases when calculating sex-specific mortatlity rates, and mortality rates for both sexes have often been used.[20] Also, it is not unlikely that mortality in adult ages seldom is related to infectious disease but rather to accidents, work- and alcohol-related mortality, etc.[21] Hence, these measures are not unproblematic and improvements will therefore be suggested in the following section.

New ways of using infant mortality rates to approximate early life conditions

Infant mortality includes all deaths from 0 to 365 days of age, which means that also the deaths occurring in the first month(s) of life are also included in this measure. Mortality during first month is usually considered to be endogenous, which means that the first-month deaths should not have any exogenous causation.[22] These deaths should rather be considered as endogenous mortality, most likely caused by congenital defects or disadvantageous conditions during the foetal stage. Hence, the first-month fraction of the infant mortality rate should not reflect any exogenous conditions, and should thus not be related to any infancy infectious diseases. Does this mean that the local mortality rate is not a good approximation of local disease load since at least a fraction of it consists of endogenous mortality, not dependent on exogenous conditions? Since empirical evidence shows that first-month mortality accounts for quite a large fraction of infant mortality, this may be a more important issue than what one might initially think. Hence, to be a good proxy for disease load, the infant mortality rate should be reduced by a substantial fraction of deaths. A useful name for this mortality measure could be the exogenous infant mortality rate. An implication is that a reduction in the order of – say – 20 to 40 % of the 0-365 day infant mortality cases can affect the measure in a negative way (thus, more random variation) since many cases will be lost. In larger samples, this would not be any major issue, but in small samples, there is reason to be cautious with the endogenous infant mortality measure.

If the standard 0-365 day infant mortality rate is reduced by mortality during the first months and used as proxy for infancy disease load, this would – as discussed above – be the exogenous infant mortality rate. Thus, what could be called the endogenous infant mortality rate would be left out from the infant mortality rate. Since it is seldom a good idea to waste information, it might be a good thing not to just throw this endogenous infant mortality rate away before at least considering what it actually contains. Again, according to the discussion above, mortality during the first month(s) after birth is usually considered to be due to congenital defects or maternal conditions during the foetal stage, for example low nutrition. But the conditions for the foetus during pregnancy is exactly what the "true" Barker hypothesis is about, and if endogenous mortality stems from foetal conditions, this measure could then be used as an approximate measure of foetal conditions during the foetal stage. Since a quite large fraction of the deaths actually – as stated above – occur in the first month(s) after birth, the number of cases should be enough to make this a good proxy measure for foetal stage conditions.

[20] Bengtsson (1997:16-19), Bengtsson & Lindström (2000:274-275), Bengtsson & Lindström (2001:9-13), Bengtsson, Broström & Lindström (2002:11, 20-23), Johansson (2004:134-139).

[21] Imhof & Lindskog (1973:PAGES), Widén (1975:PAGES), Fridlizius (1983, 1985:PAGES), Fridlizius & Ohlsson (1983:PAGES).

[22] It can be debated if one should settle for one month or consider this period of "extended foetal stage" to two or three months; some scholars even suggest that as long as up to 6 months should be regarded as an extension of the foetal stage. Thus, what is stated as first month in the following text could be stated as first months of life instead, so one month should not be taken literally but rather as "the first months".

This new measure of early life conditions should be a better proxy for foetal stage conditions than the standard measure of foetal stage conditions, the maternal disease load, approximated by the local adult-age mortality, and nutritional status, approximated by foetal stage food prices. It is not only that it more generally should measure the conditions during the foetal stage, but also that since it is common that mortality in ages 15 to 45 is quite low, so mortality for both sexes has to be used and not female mortality only. Since most male mortality in this age group is due to accidents and drinking, and not due to infectious diseases, at least the male part of the mortality rate in this age group does not reflect any general disease load.[23] To a certain extent, it is likely to be the same with women. Most deaths were at least not due to infectious diseases as in the case with infants,[24] which means that this measure most likely is not a good proxy for foetal disease load, or maybe even of general foetal conditions. These doubts about the usefulness of the adult mortality rate during foetal stage have been raised before, and the discussion above could explain the meagre outcome when the foetal stage disease load hypothesis has been tested empirically with the local adult mortality rate.[25] The same is of course true for the nutritional link approximated by foetal stage food prices, where the link is rather far-fetched.[26]

To summarise, this new foetal stage condition measure would be theoretically correct, and also more accurate than the adult age mortality proxy (for example, local male and female mortality in age 15 to 45). It should also be empirically applicable without any practical problems if the deaths in the data sample are reasonable numerous. The measures could be a solution to some of the problems with approximating foetal stage early life conditions and testing the "true" Barker hypothesis: the investigations that have tested this hypothsis using the local adult mortality rate as a proxy for foetal stage disease load have not been able to show such a relationship. Hence, the reasons to use these new measures are that

1) the exogenous infant mortality rate is a better measure of early life conditions since it is more pure disease load approximation than with the full 0-365 day infant mortality rate, and

2) the endogenous infant mortality rate provides a measure of nutritional conditions in utero, and can replace the indirect measuring of nutrition and disease load in the foetal stage via food prices and adult mortality, and test the "true" Barker hypothesis.

Data: the data used are family reconstitued data from Scania, Sweden, from the Scanian Demographic Database at the Department of Economic History, Lund University.

Method: the suggested new and improved measures will be used in a Cox survival regression model for adult mortality with shared frailty at the family level for Scania, Sweden 1814-1894.

Results: are yet not available since data is still in preparation.

[23] Imhof & Lindskog (1973:PAGES), Widén (1975:PAGES), Fridlizius (1983, 1985:PAGES), Fridlizius & Ohlsson (1983:PAGES).
[24] Ibid.
[25] Johansson (2004:PAGES).
[26] See discussion on this in Johansson (2004:PAGES).