Infant mortality and mortality from arteriosclerotic heart disease

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ABSTRACT

Objective: To estimate how much of the fall in mortality from arteriosclerotic heart disease from 1973 to 1993 that may be explained by the improved infant mortality experienced by these cohorts (Forsdahl effect).

Design: Longitudinal observational study on mortality rates.

Setting: Norway and Norwegian counties.

Main results: For females up to 50% of the observed reduction in cardiovascular mortality from 1973 to 1993 might be due to the Forsdahl effect. For males the corresponding figure is about 40%. *Conclusions:* If living conditions in childhood as indicated by infant mortality in a cohort is a valid causative factor for later cardiovascular mortality in the same cohorts, almost 1/2 of the reduction in cardiovascular mortality from 1973-93 in Norway could be explained by this factor.

INTRODUCTION

Arteriosclerotic heart disease is still the No. 1 killer in Norway as in most Western countries. Cardiovascular mortality in Norway is five times that of Japan, and three times that of France (1). However, cardiovascular mortality has dropped considerably during the past 20 years, by about 50% for men, and 40% for women. Forsdahl demonstrated 20 years ago that mortality from arteriosclerotic heart disease among the middleaged (40-69 years) was correlated to the infant mortality in the county when and where they were born (2,3). These findings were later duplicated by others (4). Criticism has been raised that these studies inadequately take account of the fact that areas which were severely deprived earlier this century remain the most deprived today (5,6). However, Arnesen and Forsdahl also showed that on an individual level, poverty during childhood followed by a high standard of living operates as a risk factor for coronary heart disease (7). The mortality from arteriosclerotic heart disease is a complex matter and the result of many different forces: the life-style, changes in the diagnostic and curative efficiency etc. Infant mortality is most often used as an indicator on living conditions of a cohort at childhood.

The question we raise in this paper is: how much of the fall in mortality from arteriosclerotic heart disease from 1973 to 1993 may be explained by the improved infant mortality experienced by these cohorts? We do not attempt to answer which specific causal mechanisms that would contribute to this effect.

MATERIAL AND METHODS

Two separate data sources were used. First, Forsdahl's data on regression of mortality from arteriosclerotic heart disease on the past infant mortality. Second, official Norwegian statistics on mortality from arteriosclerotic heart disease in 1973 and 1993, infant mortality in 1918 and 1938, and the average year of birth for the 40-69 years old cohorts in 1973 and 1993.

The Forsdahl regression lines were used to estimate the hypothetical reduction in mortality from arteriosclerotic heart disease from 1973 to 1993 on the basis of the infant mortality in 1918 and 1938. The Forsdahl regressions were calculated on the basis of Forsdahl's original data (Table 1) (Forsdahl, personal communication).

OBSERVATIONS/FINDINGS

Figure 1 is based upon data from Forsdahl (2,3; personal communication), and shows the correlation between mortality from arteriosclerotic heart disease and infant mortality 40-69 years earlier for 20 Norwegian counties. Figure 2 shows that infant mortality by the end of this century is less than one tenth of what it was in the beginning of the century. Figure 3 shows the fall in mortality from arteriosclerotic heart disease by different age groups over the last 20 years in Norway.

The reduction in infant mortality from approximately 60 in 1920 to 40 in 1940 corresponds to a reduction in mortality from arteriosclerotic heart disease

 Table 1. Regression of mortality from arteriosclerotic heart disease (per 1000) on infant mortality (per 1000)*.

	Males	Females
Intercept	138.509	36.513
Regression coefficient	3.627	1.036

* Mortality from arteriosclerotic heart disease = intercept + (regression coefficient • infant mortality)

among the same cohorts 40-69 years later of 20% for males and 21% for females. Figure 4 compares this observed trend with the hypothetical trend explained by the Forsdahl effect alone. The figure shows that for females up to 50% of the observed reduction might be due to the Forsdahl effect. For males the corresponding figure is approximately 40%.

DISCUSSION

This study has shown that almost 1/2 of the drop in cardiovascular mortality in Norway from 1973 to 1993 could be explained by the decrease in infant mortality among the same cohorts, if there is a causative link between the two. It is important to emphasise that the study does not provide new evidence in favour of the Forsdahl hypothesis.

We know that the observed reduction in mortality from arteriosclerotic heart disease in Norway from 1973 to 1993 is the result of many factors. Many lifestyle factors have changed, and the diagnostic and therapeutic quality in health care has improved. In addition to this, the Forsdahl hypothesis claims that cohorts with infancy in poverty are badly prepared for affluence later in life. These cohorts respond with enhanced mortality from arteriosclerotic heart disease.

The potential of this proposed effect for the future is extensive, but uncertain. The observed cohorts experienced infant mortality of 40 and 60 per 1000 births. Later, the infant mortality has been gradually reduced to 5! There is, however, little reason to expect a parallel reduction in mortality from arteriosclerotic heart disease. We know that the present mortality from arteriosclerotic heart disease in Japan and France is only a fraction (20% and 30%) of the Norwegian level in cohorts of the same age (1), but which experienced much higher infant mortality than the Norwegian cohorts. The Forsdahl effect might be valid for these countries as well, on top of other factors.

If poverty as indicated by the infant mortality makes people highly vulnerable for the kind of western, affluent society diets and drinking habits that we have in Norway, we may hypothesise dramatic increases in cardiovascular mortality in developing countries as their standard of living increase. We know, for instance that several Pacific countries are experiencing rapid increase in incidences of diabetes mellitus, an indicator of dramatic changes in lifestyle.

Our analysis is meant as a comment to the present trend on mortality from cardiovascular disease. It is not meant as a proof, but indicates a potential that deserves further study, and a warning to the fast developing countries.

One possible shortcoming of our study is that Forsdahl's calculations pertained to infant mortality rates in the range of 50 to 130 per thousand. It might not be applicable in the range of 40 to 60 as we have assumed. Even if the effect is present in this range, it might not be linear.

CONCLUSION

It is possible that as much as 1/2 of the decline in mortality from arteriosclerotic heart disease between 1973 and 1993 may be explained by improved living conditions for the same cohorts during childhood, as indicated by infant mortality rates.



Figure 1. Mortality from arteriosclerotic heart disease 1974/75 vs. infant mortality 40-69 years earlier in 20 Norwegian counties.



Figure 2. Decline in infant mortality 1910-1995.



Figure 3. Ratio between mortality from arteriosclerotic heart disease 1993 and 1973.



Figure 4. Observed trends in cardiovascular mortality 1973-1993 in % of mortality in 1973 compared with estimates based on the Forsdahl regressions.

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