Chapter 23

The Month of Birth: Evidence for Declining but Persistent Cohort Effects in Lifespan

Gabriele Doblhammer

23.1 Introduction

In the second half of the nineteenth century, mortality started to decline and this decline seemed to follow a cohort pattern. A cohort pattern is direct evidence for the effect of early-life circumstances on adult mortality: improvements in the living environment early in life lead to reduced mortality during the whole life course. Early studies for England, Wales and Scotland (Kermack et al. 1934, 2001) suggest that, as far as mortality up to the year 1925 is concerned, the year of birth had more predictive power than the year of death. Around 1925, the responsible factors for the mortality decline changed and the year of birth lost its predictive power. Period factors became more and more important. In a recent study, Davey Smith and Kuh (2001) updated the table of relative death rates for England and Wales from the Kermack paper. They showed that from 1925 onwards at younger ages, death rates fell faster than predicted based on birth cohort regularities, whereas at older ages, mortality declined at a much slower rate than predicted.

Elo and Preston (1992) point out that the early studies were probably so successful in demonstrating cohort effects because they were based on mortality data from a time period before the process of mortality decline on a period level had begun. Once both cohorts and periods began to show mortality improvements, it became more difficult to separate the two.

The cohort decline at the beginning of the epidemiological transition is not without contest either. In their study of the Swedish mortality decline, Kermack et al. (1934, 2001) admitted that the cohort effect is not as clear as it was for England and Wales. The authors argue that a rectangular block dating from 1855 onwards and

G. Doblhammer (✉)

University of Rostock, Rostock, Germany

Rostock Centre for the Study of Demographic Change, Rostock, Germany

e-mail: doblhammer@rostockerzentrum.de

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affecting the age groups 10 to 30 years was the primary cause of disturbances in the cohort decline. If the block is omitted, a cohort decline is observed. This result could not be replicated, however, in a study of Swedish mortality between 1778 and 1993 by Vaupel et al. (1997). The conclusion according to Vaupel et al. is that “the pattern is clearly more complex than a pure cohort-effect model would suggest” (Vaupel et al. 1997: 63).

As a result of the loss of the predictive power of cohort factors around 1925, the emphasis turned towards period factors such as advances in medical technology, life-style, smoking, physical activity and diet. In the 1970s, however, Forsdahl (1973, 1977, 1978) observed that regional differences in adult lung cancer and heart disease were not related to contemporary differences in lifestyle, smoking behaviour, or socioeconomic status but rather to differences in regional infant mortality during childhood and youth of the cohorts under study. His study is now considered the starting point of a large and productive area of research that tries to link early-life conditions to the manifestation of chronic disease later in life. The discussion about early-life effects on health at adult ages gained momentum with studies conducted by the Southampton group of Barker and colleagues (Barker 1994; Barker and Osmond 1986a, b, 1987). The group developed the fetal-origins hypothesis of adult disease (also known as the ‘Barker hypothesis’), which suggests that coronary heart disease at adult ages results from poor conditions in utero caused by inadequate nutrition on the part of the mother and infectious diseases she suffered during pregnancy. Since inadequate nutrition of the fetus is reflected in low birth weight, the Barker hypothesis claims that growth retardation in utero leads to low birth weight and to an increased risk of chronic disease later in life. It seems that the main connection between birth weight and heart disease later in life is systolic blood pressure – infants with a low birth weight experience increased systolic blood pressure at adult ages.

The fetal-origins hypothesis has led to a large amount of research that generally concludes that low birth weight is associated with an increased risk of heart disease at adult ages and that low-birth weight infants suffer from increased systolic blood pressure later in life. The interpretation of these outcomes has been repeatedly challenged, however. The main idea underlying the fetal-origins hypothesis is that a critical period exists early in life and that negative effects during this period cannot be reversed later in life. Critics of the hypothesis frequently bring forward the argument that birth weight is confounded with socioeconomic status. Negative social factors in the early-life environment may set people onto life trajectories that negatively affect their health over the whole life course. Therefore, the almost universally observed relationship between birth weight and the risk of chronic disease later in life may be an outcome of the whole life course rather than the result of a critical period early in life (Joseph and Kramer 1996; Kramer 2000).

This criticism leads to the question whether one can find an indicator for the prenatal and early postnatal environment that is not related to the life-course. Birth weight certainly does not fulfil this criterion and, in addition, it is not widely available. Studies that use birth weight or other direct indicators of early-life circumstances are usually based on hospital data, which are invariable subject to selection bias. Moreover, their sample sizes tend to be modest.

The fetal-origins hypothesis suggests that nutrition and infectious diseases during the pregnancy of the mother are responsible for growth retardation in the infant, which leads to an increased risk of heart disease at adult ages. Both nutrition and infectious diseases are highly seasonal: respiratory infections peak in the autumn and winter, and gastrointestinal infections peak during warm periods of the summer months. The availability of fresh fruits and vegetables – and thus of micronutrients – tends to change according to the seasons of the year. An indicator that reflects the seasonally changing environment during the prenatal and early postnatal period is month of birth.

Epidemiological research on the underlying factors of schizophrenia has long used month of birth as an indicator for early-life circumstances that affect the risk of schizophrenia later in life. This line of research dates back to Ellsworth Huntington, who in 1938 published his book about seasonality (Huntington 1938), in which he describes the relationship between the seasons of the year and social, psychological, and demographic phenomena. By 1997, more than 250 studies about the month-of-birth effect in schizophrenia had appeared and many more are still being published (Torrey et al. 1997). Most of the research on the relationship between month of birth and the incidence of diseases has been conducted for mental disorders, in particular schizophrenia and bipolar disorders. The season-of-birth effect has also been studied for autistic disorder, Alzheimer patients, anorexia nervosa patients, and for diseases of the nervous system such as Parkinson's disease, multiple sclerosis and epilepsy. Recently much attention was for example given to the month-of-birth effect in insulin dependent childhood diabetes. For a review of studies about the relationship between month of birth and certain diseases, see Doblhammer (2004). Many of these studies suggest that virus infections in utero or in the first few months of life are responsible for the increased risk of developing a certain disease. None of these studies, however, provides concrete evidence for a specific causal mechanism.

Although widespread evidence exists concerning the month-of-birth effect for certain diseases, little attention has been given to the question whether there is a correlation between the month of birth and lifespan and whether this relationship has changed over cohorts. If the month-of-birth effect decreases in younger cohorts then this suggests that the influence of the very first period of life on adult lifespan has reduced over time. Two recently published studies point in this direction. A study of young adults in rural Gambia found a significant difference in survival to age 45 by month of birth (Moore et al. 1997). When the authors repeated their study on a rural Bangladeshi population, they could not detect an effect of season of birth on survival (Moore et al. 2004). Furthermore, a study of early adult death in rural Senegal also failed to find any influence between month of birth and survival at young adult ages (Simondon et al. 2004). Both Moore and colleagues and Simondon and colleagues suggest that the negative findings in the Bangladeshi and Senegal population are due to cohort effects. While the data for Gambia are based on births during the 1950s and 1960s, the Bangladeshi and Senegal data include individuals born between 1974 and 2000 (Bangladesh), and 1962 and 2000 (Senegal).

This article takes up the question whether the season-of-birth effect in life-span has changed over time and whether it is less important for more recent than for older

cohorts. To answer this question the article first describes the month-of-birth pattern in lifespan for selected countries of the Northern (Austria, Denmark, and United States) and Southern Hemisphere (Australia). Second, a cohort analysis is performed based on the Danish register data and of consecutive US census rounds that include information about the season of birth. Third, the underlying causal mechanisms behind the month-of-birth effect are reviewed and finally, outcomes are discussed in the light of mortality forecasting.

23.2 Data

The optimal data to test for differences in lifespan by season of birth are longitudinal data. Birth cohorts born in a specific season are followed from birth to death and life expectancy can be calculated using simple life-table methods. Such data rarely exist however. The data that are closest to this requirement are register data from the Scandinavian countries. The Danish data used in this study consist of a mortality follow-up of all Danes who were at least 50 years old on 1 April 1968. This totals 1,371,003 people, who were followed up to week 32 of 1998. The study excludes 1994 people who were lost to the registry during the observation period. Among those who are included in the study, 86% (1,176,383 individuals) died before week 32 of 1998; 14% (192,626 individuals) were still alive at the end of the follow-up.

Population registers do not exist for Austria, Australia and the US, where only individual death records are available. Exact dates of birth and death are known for in all 681,677 Austrians who died between 1988 and 1996 and for 219,820 native-born Australians who died between 1993 and 1997 at ages 50+. Two data sources are used for the United States. First, US death records for the years 1989 to 1997, which include place of birth, are the basis for the analysis of the month-of-birth pattern in lifespan. Second, the three US census rounds 1960, 1970, and 1980 are used to study cohort patterns. These three census rounds are the only rounds that include information about the quarter of birth. Data are extracted from the “Public Use Microdata Samples”, which are accessible under <http://usa.ipums.org/usa/>. The extract is restricted to the native-born white US population aged 0 to 100. For whites, this gives a sample size of 1,490,444 in 1960, 1,672,107 in 1970, and 1,812,839 in 1980.

23.3 Methods

For Denmark, both the risk population and the number of deaths are known which means that it is possible to estimate remaining life expectancy at age 50 based on life tables that were corrected for left truncation. This was achieved by calculating occurrence and exposure matrices that take into account an individual's age on 1 April 1968. For example, a person who was 70 at the beginning of the study and who died at age 80 enters the exposures for ages 70 to 80 but is not included in the

exposures for ages 50 to 69. The central age-specific death rate is based on the occurrence-exposure matrix. The corresponding life-table death rate is derived by means of the Greville Method (Greville 1943).

For Austria, Australia and the United States, the population at risk is unknown, which means that lifespan by month of birth cannot be estimated based on simple life-table techniques. For these three countries, remaining lifespan at age 50 was therefore estimated by calculating the average of the exact ages at death. It has been pointed out that using mean age at death as an approximation for life expectancy may lead to serious bias in the observed month-of-birth pattern (Gavrilov and Gavrilova 2003). It is well known that mean age at death does not correctly estimate life expectancy in non-stationary and non-extinct populations. The emphasis of this study, however, is not life expectancy per se but the month-of-birth pattern in life expectancy, which generally should not be affected. There is one exception, however. If the seasonal distribution of births has changed over time then not only life expectancy but also the month-of-birth pattern is biased when estimated based on mean ages at death. If more people proportionally are born in spring in younger cohorts than in older cohorts, then for a given time period the mean age at death will be biased downward for those born in spring. In the case of Austria and Denmark, the changes in the seasonal distribution of births over time are minor, however, and the effect on the month-of-birth pattern is negligible (Doblhammer 2004).

The Danish register data consist of sufficiently large numbers of exposures and deaths to distinguish between age and cohort effects. Two ten-year birth cohorts are followed over an age-span of 20 years. The birth cohort April 1908 to March 1918 enters the study period between the ages of 50 and 59 and 11 months. They are followed from age 60 to age 79 in order, theoretically, to allow each member of the cohort to reach each age. The second cohort is aged 60 to 69 and 11 months at the 1968-baseline and is followed from age 70 to age 89. This specification allows the study of age-specific death rates at ages 70 to 79 for both cohorts. Conditional on surviving to age 70, all individuals are followed from age 70 to age 79. Those who survived age 79 are treated as censored. Mortality of the two cohorts between ages 70 and 79 is modelled by a proportional hazard model with the baseline hazard following a Gompertz function,

$$\mu(x|y_i) = ae^{b_0x}e^{b_1y_1+b_2y_2+b_3y_3} \quad (23.1)$$

where $\mu(x|y_i)$ is the force of mortality at age x conditional on covariates y_i , a is the age-independent level of mortality and b_0 the increase in mortality over age. The three indicator-variables, y_1 , y_2 and y_3 , denote the quarter of birth and take the value one if a person is born in a specific quarter, and zero otherwise; the first quarter is defined as reference group. The parameters a , b_0 , b_1 , b_2 , and b_3 are estimated by maximising the likelihood function. For each of the two cohorts, a separate model is estimated.

To estimate differences in survival according to the quarter of birth based on the US censuses for the year 1960, 1970, and 1980, a method called Survival-Attributes

Assay (Christensen et al. 2001) is applied. This method uses cross-sectional data on “fixed-attributes” to estimate the effect of a fixed trait on survival.

Let N_x be the number of people at age x . Let p_x be the proportion of x -year-olds who have some fixed attribute such as the season of birth. Let p_{x+n} be the proportion at age $x+n$. Let s be the conditional survival probability from age x to age $x+n$ for the individuals who have the fixed attribute. Let S be the conditional survival probability from age x to $x+n$ for the entire cohort.

Then, because

$$p_x N_x s = N_x S p_{x+n} \quad (23.2)$$

it follows that

$$s = S \frac{p_{x+n}}{p_x} \quad (23.3)$$

Thus, the relative risk of surviving from age x to age $x+n$ for people born in a specific quarter is the ratio of their observed proportions in the two cross-sections. The proportion of the population within 10-year age groups that is born in a certain quarter of the year is followed over the three census rounds.

This method relies on the assumption that the $x+20$ -year-olds in the third cross-section were similar to the x -year-olds in the first cross-section 20 years earlier; e.g. the 70-year-olds in 1980 are the survivors of the 60-year-olds in 1970 and the 50-year-olds in 1960. In other words, the change in the proportion of the fixed attribute over an age range of 20 years is solely due to age effects and not affected by cohort effects. Differences in the survival probability of ages that are further apart than 20 years can be both due to cohort and age effects. The method of Survival-Attributes Assays therefore, does not permit a clear distinction between age and cohort effects. The main advantages of the method are, however, that it does not require the calculation of death rates in order to verify the month-of-birth effect and that it can be used to study differences in survival at ages where death rates are low and therefore subject to random fluctuations. Thus, it can be used to study whether differences in survival by season of birth not only exists among today's elderly but also among more recent cohorts.

23.4 Results

23.4.1 Differences in Lifespan in the United States, Austria, Denmark and Australia

A similar relationship between month of birth and lifespan exists in all of the Northern Hemisphere countries (Fig. 23.1). Adults born in the autumn (October–

Fig. 23.1 Deviation from average remaining lifespan at age 50 for people born in a specific month in Austria, Australia, Denmark and the United States

December) live longer than do adults born in the spring (April–June). The difference in lifespan between the spring- and autumn-born is twice as large in Austria (0.6 years) as in Denmark (0.3 years).

In Denmark for those born in the second quarter, lifespans are 0.19 ± 0.05 years shorter than average; for those born in the fourth quarter they are 0.12 ± 0.04 years longer than average. This difference is statistically significant (Cox-Mantel statistic: $p < 0.001$). Also in Austria the deviation in mean age at death is highly significant (Bonferroni test: $p < 0.001$) for those born in the second and the fourth quarters. The lifespans of people born between weeks 14 and 26 are 0.28 ± 0.03 years below average; lifespans of those born between weeks 40 and 52 are 0.32 ± 0.03 years above average. A highly significant difference in mean age at death by month of birth exists for US decedents who died between 1989 and 1997. Those born in June and July die about 0.44 years earlier than the October-born. The pattern in the Northern Hemisphere is mirrored in the Southern Hemisphere. The mean age at death of people born in Australia in the second quarter of the year is 78.0; those born in the fourth quarter die at a mean age of 77.65. The difference of 0.35 years is statistically significant (Bonferroni test: $p < 0.001$).

Table 23.1 Parameter estimates and 95% confidence intervals of the Gompertz models estimated for Danes born in a specific quarter for birth cohorts 1898–1909 and 1909–1918 at ages 70–79

	Parameter estimate	95% LCI	95% UCI
Cohort 1898–1909			
1st quarter	1.00		
2nd quarter	1.03	1.02	1.04
3rd quarter	1.00	0.99	1.02
4th quarter	0.98	0.96	0.99
Ln(a)	−3.44	−3.45	−3.43
$b_0 \times 102$	8.59	8.42	8.76
Cohort 1909–1918			
1st quarter	1.00		
2nd quarter	1.08	0.96	1.21
3rd quarter	1.05	0.94	1.18
4th quarter	1.06	0.95	1.19
Ln(a)	−3.51	−3.62	−3.39
$b_0 \times 102$	8.08	7.49	8.68

Source: The Office for National Statistics

23.4.2 *Changes in the Month-of-Birth Pattern over Cohorts in Denmark*

Table 23.1 contains the parameter estimates $\ln(a)$ and b_0 and the odds ratios of the Gompertz models for the two cohorts 1889 to 1908 and 1909 to 1918. In the older cohort (1889 to 1909), there exists a significant excess mortality of those born in the second quarter (+3% as compared to those born in the first quarter). People born in the fourth quarter experience the lowest mortality risk (−2% compared to those born in the first quarter). In the younger cohort (1909 to 1918), the differences in mortality by quarter of birth become statistically not significant.

23.4.3 *Changes in the 20-Year Survival Probability by Quarter of Birth in the United States*

Figure 23.2 shows the relative risks of the 20-year survival probabilities conditional on age for people born in a specific quarter compared to the average population.

At younger ages, the mortality advantage of the autumn-born and the disadvantage of the spring-born is minor. For males, it is a maximum of 1% over an age range of 20 years. In other words, up to the age of 40 the conditional survival probability of surviving the next 20 years is about 1% higher for the autumn-born than for the average population; it is 1% lower for the spring-born. Differences in the 20-year survival start to accelerate from the age group 40–49 onwards, when those born in the fourth quarter have a higher chance of 2.4% to survive the next 20 years; this

Fig. 23.2 Relative risk of 20-year survival by age in the 1960 US census by sex (males = white-dotted bars, females = grey bars) and quarter of birth

advantage increases to 7.8% for ages 60–69. The disadvantage in the 20-year survival of people born in the second quarter starts at ages 50–59 and is about minus 2.8%. It increases to minus 6.6 per cent for the age group 60–69. Similar trends emerge for women.

23.5 Discussion

In all four populations, Austria, Australia, Denmark and United States, significant differences in lifespan exist by month of birth. Those born in spring generally face a lower life expectancy than those born in autumn. This finding is independent of the Hemisphere as is shown by the Australian result.

The US death data contain detailed information about the state of birth, education, marital status, and race of the deceased (Doblhammer 2004). Among the white US population, the age-standardised peak-to-trough difference in the month-of-birth pattern increases from the North to the South while the basic pattern remains unchanged. The difference is smallest in New England, with 0.31 years and largest in the East South Central Region, with 0.86 years. The differences in the West are intermediate. The age-standardised differences in lifespan by month of birth vary significantly according to education levels. The difference between the spring trough and the winter peak is 0.62 years for those with a low education and 0.38 years for the highly educated. There exists a highly significant difference in the month-of-birth pattern by marital status. The difference between the peak and the trough is largest for the never-married (0.62 of a year), and smallest for the married (0.40 of a year); the widowed (0.45 of a year) and the divorced (0.44 of a year) are intermediate. The month-of-birth pattern of the 1.7 million US African Americans differs significantly from that of the white population. It differs not only with respect to the number of years between the trough and the peak (0.57 years) but also with respect to the shape of the curve. The mean age at death is highest for those born between January and March. As in the case of the white population, the mean age at death for African Americans is lowest for decedents born in July.

In a multivariate analysis of ages at death in the United States (Doblhammer 2004), the main effects of sex, month of birth, education, race, region of birth, marital status and all the two-way interactions of the variables are highly significant. This implies that the regional differences in the month-of-birth pattern are neither due to differences in education nor to differences in race but they exist independently of them. Overall, above factors explain 25% of the variation in ages at death. About 86% of the model explanation is due to the effect of marital status and only 0.4% to the effect of month of birth. The large majority (70%) of this 0.4% result from the interactions of month of birth with region of birth and race.

What are the causal mechanisms behind the month-of-birth effect on life-span? One frequently raised concern is that the month-of-birth effect reflects the seasonal distribution of deaths rather than the seasonal changes in the early-life environment. More specifically, the concern is that the interaction between the seasonal distribution of deaths and the monthly increase in adult mortality causes a month-of-birth pattern. This hypothesis has been already widely discussed in the research about the month-of-birth effect in schizophrenia, whose incidence is seasonal and whose risk increases with age. Two studies (Doblhammer and Vaupel 2001; Doblhammer 2004) have shown that, although month of birth, age, and month of death influence mortality simultaneously, they are independent of each other.

A second, frequently raised concern is that the month-of-birth effect is caused by socioeconomic differences in the seasonal distribution of births. The number of births is distributed seasonally over the year with the exception of only a few populations. If the seasonality in births is partly driven by the preference of couples for giving birth in certain seasons of the year, then this preference may differ between social groups. In schizophrenia research, this explanation is generally

known as the procreational habits theory. Individuals with schizophrenia may have a procreational pattern that differs from those of the non-schizophrenic population (Torrey et al. 1997). On basis of the 1981 census for Austria, it was possible to refute this hypothesis (Doblhammer 2004). This is also true for the deadline hypothesis. Starting school is usually tied to reaching a certain age before a certain deadline. Children who are born shortly after the deadline have to wait an additional year before they can start school and will therefore be among the oldest of their classmates. This may pose a special advantage compared to those who are born shortly before the deadline, who will thus always be among the youngest. However, since the mean age at death of the autumn-born is higher than that of the spring-born, the deadline hypothesis cannot explain the month-of-birth effect on the lifespan.

Public health experts at the beginning of the twentieth century felt that the health status of mothers and whether mothers breastfed their babies were the two most important factors determining the survival of an infant, followed by housing, sanitation and general poverty (Preston and Haines 1991). The health status of pregnant women depended largely on their diet and on the general disease load. Breastfeeding the infant is related primarily to a lower incidence of infectious diseases of the gastrointestinal tract, which historically is the major cause of infant mortality. Danish data on historical infant mortality between the years 1911 and 1915 show that it is the spring-born who experience higher mortality in their first year of life (Doblhammer 2004). The standardised death rate of the June-born infants is 30 per cent higher than the death rate of the December-born. This finding implies that those factors that contributed to the high infant mortality of the past are also the factors that cause the differences in lifespan by month of birth.

Nutrition is highly seasonal. Diet at the beginning of the twentieth century did not much resemble contemporary dietary patterns. People ate less meat, fruits, and vegetables and more starchy staple food. The first vitamins were not discovered until 1911, and in the early 1900s, nutritionists were even opposed to greens, which were considered to require more bodily energy for digestion than they provided. Although severe malnourishment was not widespread, people had inadequate nutrition – particularly during the winter and early spring. Peak growth of the fetus in utero occurs during the third trimester. For infants born in spring, the third trimester coincides with a period of largely inadequate nutrition; for those born in the autumn it coincides with a period of plenty.

The effect of nutrition early in life on adult health is highly contested. Studies that looked at the old-age mortality of cohorts born shortly after periods of famine, which were thus presumably marked by severe malnutrition of the mother during the gestational period of their unborn, did not find any differences (Kannisto et al. 1997). Two studies about the long-term effects of severe starvation during the siege of Leningrad come to contrary results (Stanner et al. 1997; Sparén et al. 2003). The effect of the Dutch famine in 1944–1945 on later life disease and mortality is explored in a series of studies (Rosenboom et al. 2001, 2000a, b). The authors find that mortality up to age 18 was higher for those born before the famine and those exposed to the famine in the third trimester. Between the ages of

18 and 50, however, no effect of prenatal exposure to the famine could be demonstrated. Thus, the evidence is weak concerning the effect of nutrition during gestational age on mortality later in life.

The incidence of infectious diseases depends on the climate and on the seasons of the year. The incidence of waterborne infectious diseases, which affect mainly the gastrointestinal tract, is correlated with warmer temperatures and flooding. Peak climatological temperatures coincide with the incidence of foodborne diseases. Many childhood diseases are highly seasonal; airborne diseases affecting the respiratory tract usually peak in autumn and winter. Historically, people born in years with extremely high infant mortality caused primarily by whooping cough and smallpox tend to have higher mortality later in life (Bengtsson and Lindström 2003).

Infant mortality at the turn of the twentieth century was mainly caused by exogenous factors, in particular infectious disease. Infants born in spring had an increased risk to die from infectious disease during their first year of life. The month-of-birth pattern in adult lifespan suggests that those who survived were debilitated and suffered from higher mortality during adult ages.

Explanations other than nutrition and infectious disease have also been brought forward to explain the month-of-birth effect. One of the first to study the influence of the month of birth on the lifespan was Elsworth Huntington, who formulated the hypothesis in 1936 that high temperatures at the time of conception weaken the “germ plasma” of the parents, with negative effects on the development of the foetus. Recent research has shown that the sperm quality of men who work outdoors does indeed decrease during periods of high temperatures (Centola and Eberly 1999). A related hypothesis is that hot summers are the cause of protein deficiencies at the time of conception (Pasamanick 1986). This hypothesis is clearly ruled out on basis of the US death data. The United States consists of six major climatic zones with very different climatic conditions. Since the US death data contain the state of birth, it is possible to correlate the peak-to-trough difference in lifespan by month of birth for people born in a specific state with maximum and minimum temperature and with the maximum difference in temperature. It appears that no correlation exists between the peak-to-trough difference and the temperature variables, neither for total mortality nor for major causes of death (Doblhammer 2004).

Another explanation is that seasonal changes in the hours of daylight influence the human endocrine functions and that the month-of-birth effect might be caused by variations in the internal chemistry or neural development brought about by the seasonal variations in light (Wehr 1998; Turnquist 1993; Qusted 1991; Morgan 1978; Jongbloet 1975; Pallast et al. 1994).

23.6 Conclusion

The analysis of the Danish register data and the consecutive US census rounds shows that the differences in lifespan by month of birth have become smaller over time. This is consistent with the explanation of nutrition and infectious disease since both

have considerably improved over time. Although diet still differs between spring and fall, the difference in the nutritional value is much smaller than at the beginning of the twentieth century. In addition, the epidemiological transition has reduced infectious disease to a minor cause of death.

Ample evidence exists that the health of today's elderly is scarred by negative events that they experienced during their pre-natal or early post-natal life. This study shows that already among the elderly, those born in more recent years are less affected by seasonal early life factors and that the month-of-birth effect has become smaller. This finding suggests that in more recent cohorts period factors as opposed to cohort factors have gained increased importance and that period factors may therefore predominantly determine future gains in life expectancy.

On the other hand, the US census rounds indicate that differences in the 20-year survival still exist at young ages between 1960 and 1980. In addition, a study of twins born in the 1970s in Minnesota (Doblhammer 2004) shows that the seasonal pattern in birth weight – a widely used indicator for growth retardation in utero – is positively correlated with the month-of-birth pattern in the mean age at death of decedents aged 50+ who were born in Minnesota. Thus, there exists evidence that the seasonal fluctuations in the early life environment of recent cohorts have still an effect on life expectancy.

Huge gains have been made in the health environment during the very first period of life during the last century. Infant mortality has fallen drastically during the last century. Since infant mortality in the early twentieth century was primarily due to exogenous factors such as infectious disease, the decline in infant mortality points to a largely improved health environment of infants and children. However, in the 1980s and 1990s researchers have repeatedly pointed out that decreasing poverty among the elderly has led to increasing poverty in childhood (Preston 1984), particularly in the United States. A recent article by Komlos and Baur (2004) finds that in the most recent decades the height of Americans has been lagging behind that of Europeans while at the beginning of the twentieth century they were the tallest of the world. The authors even present some evidence that heights have been stagnating among US men and might actually be decreasing among females born in the 1960s. Height primarily reflects the socioeconomic and epidemiological environment during childhood and adolescence. It is significantly correlated with health and longevity at adult ages. Height and life expectancy rises together.

In an ageing society with too few children and an ever increasing proportion of the old and very old the danger exists that resources in general and social transfers in particular are channelled towards the elderly which may lead to increasing poverty among children. In his 1984 article, Preston wrote, “that the transfers from the working age population to the elderly are also transfers away from children [...]”. Thus, childhood poverty may become an ever more widespread phenomenon leading to a deterioration of the social and epidemiological environment early in life. In this case, cohort factors may gain importance again in mortality forecasting.

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