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THE LONG-TERM IMPACT OF WAR ON MORTALITY: OLD-AGE MORTALITY OF THE FIRST WORLD WAR SURVIVORS IN THE FEDERAL REPUBLIC OF GERMANY

*Shiro Horiuchi**

SUMMARY

For an investigation of long-term impacts of the world wars on mortality of the survivors, vital statistics in the Federal Republic of Germany from 1959 to 1974 were analysed using the age-period-cohort binary-variable regression method and the period rate of mortality change with age, which is a measure very sensitive to cohort variations. The results have revealed that the cohort of males of the Federal Republic of Germany who were adolescents (about age 15) at the end of the First World War experienced high mortality in its old age, as compared to its preceding and succeeding cohorts. This pattern has not been observed for females. Similar cohort variations have been found, though to a lesser extent, among males in some other countries, such as France and Austria, that were deeply involved in the First World War, and have begun to appear in the middle-age mortality of the Second World War survivors in the Federal Republic of Germany and Japan. The mortality patterns seem to reflect long-term impacts of malnutrition under the hardship of life during war upon vascular structures of male adolescents. The present study highlights the significance of further research on the long-term influences of catastrophic events on the health of the survivors, to which little attention has been paid.

INTRODUCTION

Mortality tends to rise during war. A number of people are killed in combat, and the hardship of life during war may also increase the number of deaths. Furthermore, warfare usually has some impact on the health and mortality of the survivors who were injured in combat or exposed to poor hygiene and malnutrition. However, little attention has been given to the long-term effects of war on mortality.

Okubo has analysed age patterns of mortality in Japan after the Second World War and has shown by graphic presentation that the male cohort that was about age 15 at the end of the War, a generation that is slightly younger than the one that suffered heavy casualties in combat, experienced relatively high mortality in middle age.[1] Such a cohort variation has not been found for females. Okubo has speculated that the malnutrition in those days might have weakened the blood vessel structures of male adolescents. His findings lead us to the expectation that a similar cohort variation in mortality might be found among the First World War survivors, especially in the Federal Republic of Germany where people experienced significant hardships near the end of the War. The present investigation is undertaken in order to analyse cohort patterns of old-age mortality in the Federal Republic of Germany.

MATERIALS AND METHODS

Data on the mid-year population and the number of deaths by age, published by the Statistical Office of the Federal Republic of Germany, were obtained to compute the age-specific mortality rates shown in table 1. The years 1959, 1964, 1969 and 1974 were chosen in order to follow the five-year cohort born between 1899 and 1904, which seems to correspond approximately to the high-mortality male cohort in Japan, with respect to age at the end of the world wars.

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TABLE 1. AGE-SPECIFIC MORTALITY RATES FOR THE FEDERAL REPUBLIC OF GERMANY 1959, 1964, 1969 AND 1974
(Deaths per 1,000)

	Males				Females			
Age	*1959*	*1964*	*1969*	*1974*	*1959*	*1964*	*1969*	*1974*
35–39	2.44	2.39	2.46	2.36	1.68	1.53	1.50	1.23
40–44	3.35	3.54	3.68	3.61	2.34	2.32	2.30	2.07
45–49	5.40	5.49	5.88	5.66	3.53	3.47	3.66	3.32
50–54	9.29	9.17	9.62	9.21	5.33	5.12	5.42	5.12
55–59	15.95	15.97	15.82	14.64	8.20	7.90	8.03	7.20
60–64	25.19	26.75	27.84	23.94	13.61	13.09	13.37	11.56
65–69	38.84	41.07	46.35	40.72	23.98	22.22	23.56	20.09
70–74	60.08	61.43	71.13	65.19	43.16	39.23	41.49	36.35
75–79	95.85	94.08	104.42	99.78	78.64	69.49	72.80	65.28
80–84	154.67	144.35	154.83	149.92	133.67	119.05	123.79	114.92

Source: Federal Republic of Germany, *Statistisches Bundesamt*, 1961, 1965, 1972, 1976, *Bevoelkerung und Kultur, Reihe 7: Gesundheitswesen. 1959. 1964. 1969. 1974* (Stuttgart, *Statistisches Bundesamt*).

Two methods of data analysis were employed. First, the rate of mortality change with age, defined by

$$k(x) = \frac{d \log (\mu(x))}{dx}, \tag{1}$$

where $\mu(x)$ is the mortality rate at exact age x, was estimated for ages 40, 45, . . ., 80 in each study year. Coale and Horiuchi have shown that the measure is useful for analysing age and cohort variations of mortality that are not easily detected using more conventional measures.[2] With five-year age group data, $k(x)$ is approximated by

$$k(x) \doteq \frac{\log ({}_5M_x / {}_5M_{x-5})}{5}, \tag{2}$$

where ${}_5M_x$ is the number of deaths divided by person-years at risk in the age interval $(x, x+5)$.[3,4]

Second, in order to divide mortality variations into age, period and cohort components, a dummy variable regression analysis was conducted. The age-specific death rate M_{ij} is transformed logarithmically:

$$G_{ij} = \log M_{ij}, \tag{3}$$

where $i = 1, 2, \ldots, 10$ are the five-year age groups 35–39, 40–44, . . ., 80–84[5] and $j = 1, 2, 3, 4$ are the years 1959, 1964, 1969 and 1974, respectively.[6] Also, let $k = 1, 2, \ldots, 13$ be the cohorts born in the periods 1874–1879, 1879–1884, . . ., 1934–1939. Note $k = 10 + j - i$. The model proposed is

$$\hat{G}_{ij} = \alpha + \beta_i + \gamma_j + \delta_k, \tag{4}$$

where $\hat{G}_{ij}$ is an estimate of G_{ij}, α is a constant, and β, γ and δ are the age factor, period factor and cohort factor, respectively. This is the simplest kind of age-period-cohort model since the effects of age, period and cohort on G_{ij} are assumed to be additive.[7]

Model (4) has been used in several mortality studies. Sacher adopted the model for analysing the mortality from tuberculosis,[8] and Barrett also employed it for studying death rates due to cancers of the cervix, bladder, breast and prostate.[9]

The analysis was conducted with the conditions $\beta_{10} = 0$, $\gamma_4 = 0$ and $\delta_{12} = \delta_{13} = 0$.[10] Although the model was made estimable by setting factors for the two youngest (12th and 13th) cohorts equal to each other, strong interdependency among age, period and cohort may jeopardize the substantive interpretability of the estimated factors. In this sense, the use of the $k(x)$ analysis described above is especially helpful because the consistency of results can be examined between the $k(x)$ analysis and age-period-cohort regression.[11]

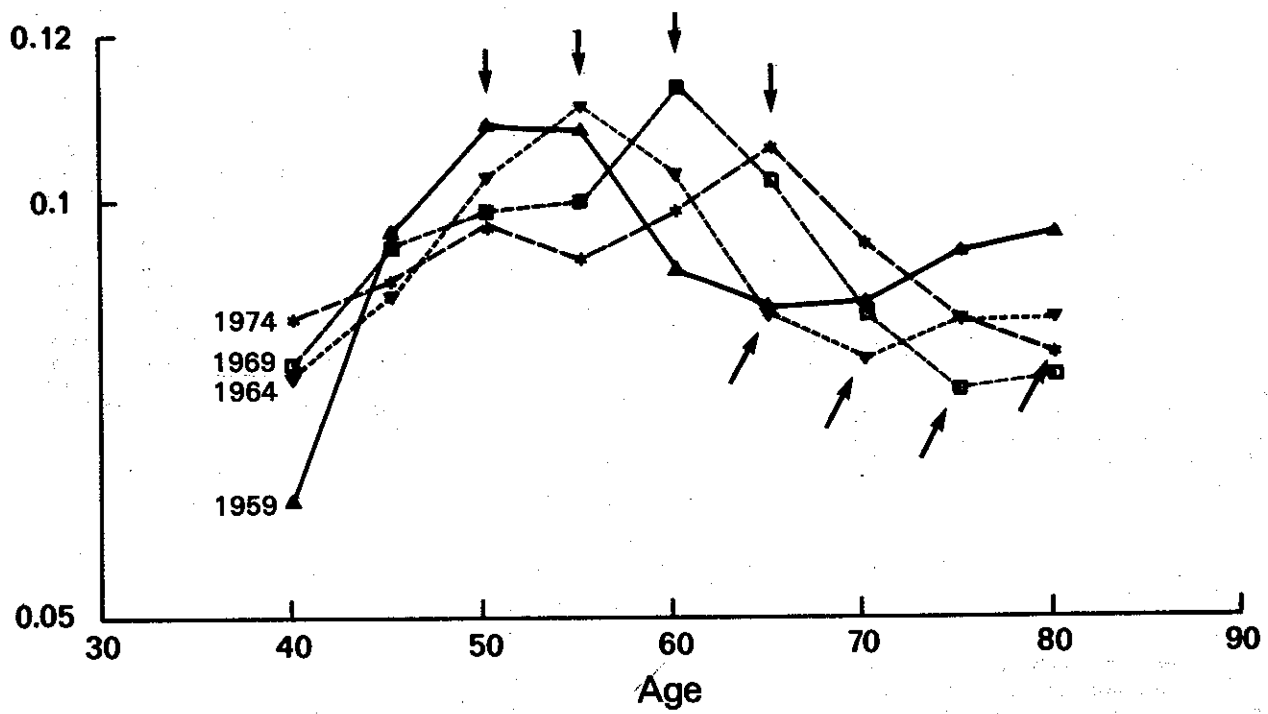
RESULTS

Figure I illustrates the age pattern of $k(x)$ for males. It is clearly seen that a dip and a peak, corresponding to births at the beginning of 1894 and 1909, respectively, shift to the right as cohorts get older. This cohort pattern suggests that a high-mortality cohort exists between the dip and the peak, for the following reason. If a low-mortality cohort is followed by a high-mortality cohort, mortality increases with age from the younger group to the older group at a relatively low rate, so that $k(x)$ tends to be small. If the order is reversed, $k(x)$ tends to be large. Therefore, if a cohort experiences a relatively higher mortality schedule than its adjacent ones, low values of $k(x)$ in older ages tend to be followed by high values of $k(x)$ in younger ages, thereby resulting in a sequence of a dip and a peak as seen in figure I. Note that the cohort located in the middle of the dip and the peak was about age 16 in 1918, the year when the War ended.[12]

Such a shift of dip and peak of $k(x)$ with cohorts is not found for females. As seen in figure II, age patterns of $k(x)$ for females are very similar for the four distinct periods, and no strong indication of cohort variation is observed.

For further investigation of cohort variations in male mortality, age-period-cohort regression analysis was conducted and R^2 was above 0.99. As presented in table 2 and illustrated in figure III, the highest cohort factor was obtained by the cohort that was born between 1899 and 1904, the one that was about age 16 in 1918. Its cohort factor is larger than those of the cohorts 10 years older and 10 years younger by 0.0832 and 0.0792, corresponding to 8.7 and 8.2 per cent higher mortality rates, respectively. As shown in figure IV, the high-mortality cohort is slightly younger than the generations of soldiers. In figure IV, the area bounded by the serrated line roughly approximates soldiers' lives lost in combat.

Figure I. Rate of mortality change with age, $k(x)$, for males, Federal Republic of Germany, 1959, 1964, 1969 and 1974



NOTE: The upward-pointing arrows and downward-pointing arrows indicate the positions of the cohorts born in 1894 and 1909, respectively.

Figure II. Rate of mortality change with age, $k(x)$, for females, Federal Republic of Germany, 1959, 1964, 1969 and 1974

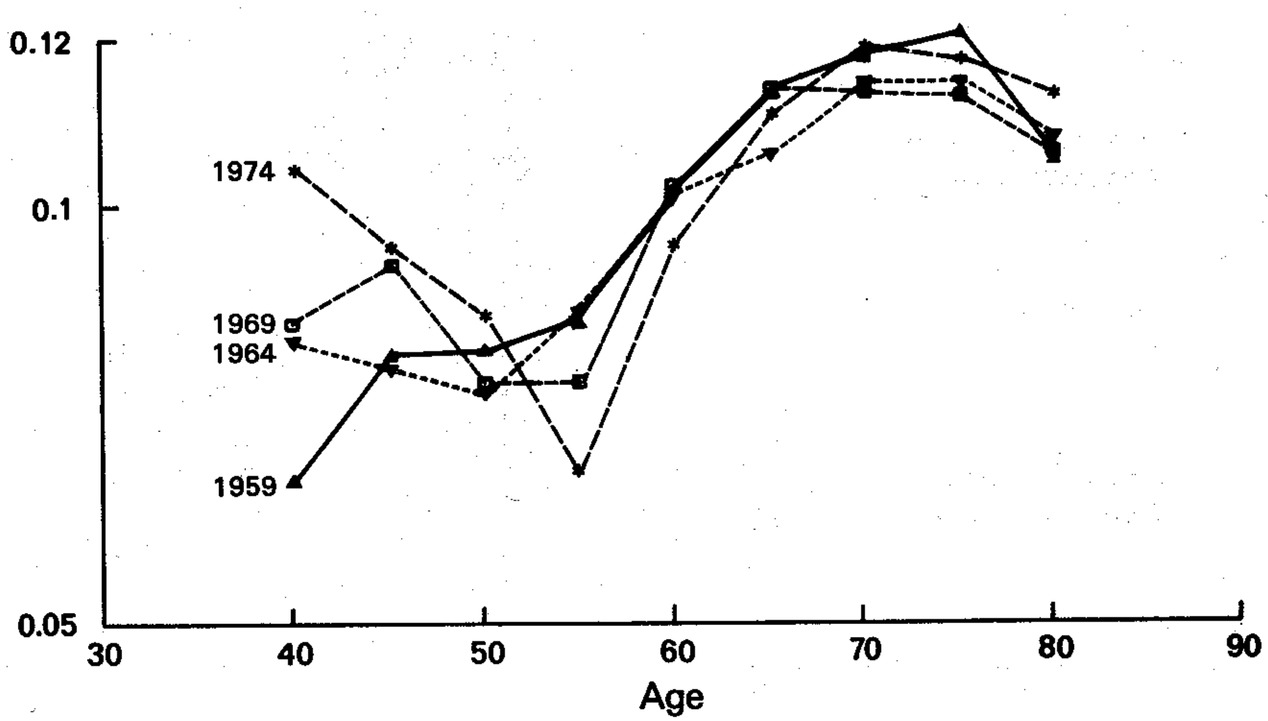


Figure III. Plot of some cohort factors (δ's) in model (4)

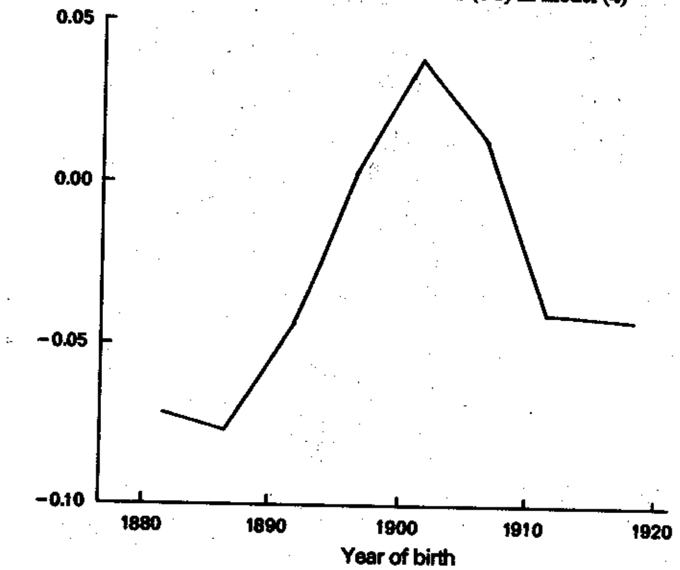
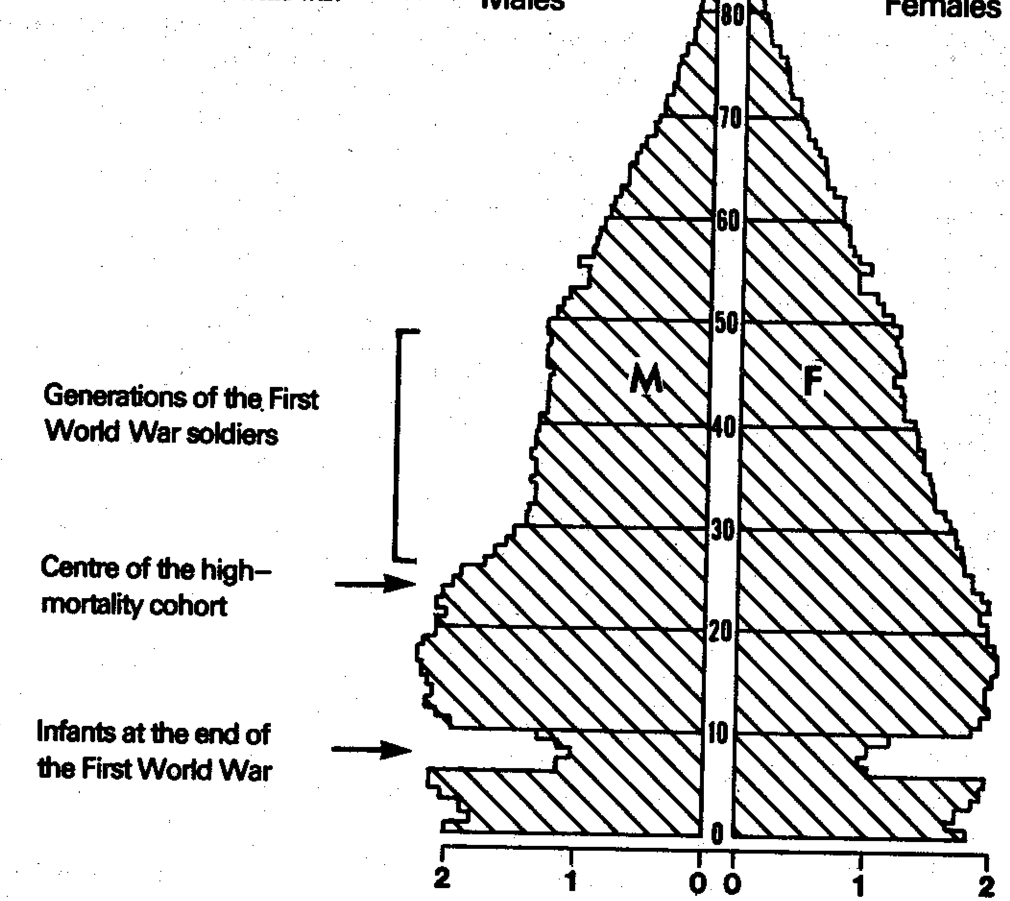


TABLE 2. ESTIMATED COEFFICIENTS OF REGRESSION ANALYSIS OF THE LOGARITHM OF AGE-SPECIFIC MORTALITY RATE ON AGE, PERIOD AND COHORT FOR MALES IN THE FEDERAL REPUBLIC OF GERMANY

Age (β)		*Period (γ)*		*Cohort (δ)*	
35–39	−4.1839	1959	0.0068	1874–1879	−0.0083
40–44	−3.7892	1964	0.0151	1879–1884	−0.0714
45–49	−3.3199	1969	0.0739	1884–1889	−0.0765
50–54	−2.8166	1974	0.0000	1889–1894	−0.0448
55–59	−2.3126			1894–1899	0.0025
60–64	−1.8161			1899–1904	0.0384
65–69	−1.3395			1904–1909	0.0127
70–74	−0.8828			1909–1914	−0.0407
75–79	−0.4296			1914–1919	−0.0420
80–84	0.0000			1919–1924	−0.0022
				1924–1929	−0.0062
Constant	−1.8650			1929–1934	0.0000
				1934–1939	0.0000

Figure IV. Age distribution of German population, 1925

NOTE: The area bounded by the serrated line roughly approximates the lives that were lost in the First World War.



Source: Federal Republic of Germany, *Bundesministerium für Gesundheitswesen, Das Gesundheitswesen der Bundesrepublik Deutschland, Band 1* (Stuttgart/Mainz, Verlag W. Kohlhammer, 1963), p. 47.

Patterns of age factors and period factors in table 2 appear reasonable. The age factor increases with age at the rate of 8 to 10 per cent per year of age, thus agreeing quite well with the model of geometric increase of mortality that assumes a constant rate of mortality growth.[13] Note that the age pattern of observed death rates is less in agreement with the model, since the observed death rate increases with age at a more fluctuating rate, as shown in figure I, reflecting cohort variations.

Period factors have a high peak in 1969, suggesting that conditions in the year raised mortality by 6 to 8 per cent higher than the other years. Although the peak does not meet the expectation of declining mortality, the pattern simply mirrors periodical variations in observed death rates, since the geometric mean of age-specific death rates[14] from age 35 to 84 in 1969 is also 6 to 8 per cent higher than the others.

Discussion

It has been shown above that the male cohort centred at the birth years of 1901 and 1902 has experienced relatively high mortality in its old age. The evidence itself does not necessarily imply that the high mortality is related to the experience of the cohort during the First World War. However, similar patterns of mortality are observed, though to a lesser extent, among some other countries that were deeply involved in the First and Second World Wars.

Figure V(*a*) shows that a similar shift of dip and peak of *k(x)* is also seen for French males, although the mid-point of cohorts between the dip and the peak seems two or three years older than its counterpart in the Federal Republic of Germany. As revealed in figures V(*b*) and (*c*), shifts of *k(x)* patterns with cohorts are found for males in the German Democratic Republic and Austria, both of which were deeply involved in the First World War. On the other hand, similar cohort variations are difficult to find in such countries as Japan and Sweden, which did not play major roles in the First World War, as shown in figures V(*d*) and (*e*). However, it seems that the footprint of the Second World War began to appear in Japan, and the Federal Republic of Germany as well. In figure V(*f*), the peak-and-dip pattern is seen in *k(x)* sequences from recent mortality data in both countries, and the cohort between the peak and the dip was about 14 at the end of the Second World War. Given this evidence, it seems quite plausible that the cohort variations in old-age mortality of males of the Federal Republic of Germany that were analysed in the last section reflect some impacts of the First World War.

Our findings may indicate that male adolescents are especially vulnerable to malnutrition experienced under the hardship of life during war, with respect to its long-term influences. The under-consumption of food seems to have been substantial. Production statistics in 1913 and 1920, for example, reflect the impact of the War on food supply. Comparing data on the production of basic food shifts, such as bread-making grains, potatoes, meat and animal fats, butter and vegetable fats, milk and so on, in those two years, Grebler has shown "that in 1920 the German people had still to be content with about 50 per cent of the supply of the most necessary articles of diet as compared with 1913".[15] Rubner estimated changes in the amount of nutrition taken by the German people during the War and concluded "that nutrition in the towns, particularly in the large towns, was not sufficient to maintain the population, and that for many under-consumption in 1914 to 1918 resulted in starvation . . .".[16] The malnutrition immediately after the end of the War was also significant. The Allied food blockage of Germany continued for about five months after the Armistice of 11 November 1918. According to Bane and Lutz, "the suffering of the German children, women and men, with the exception of farmers and rich hoarders, was greater under the continued blockade than prior to the Armistice".[17]

Problems that remain unsolved are: (*a*) why the influences last a long time, (*b*) why adolescents tend to be affected, and (*c*) why males are more vulnerable than females. Some speculations about these issues are given below.

First, if malnutrition has some detrimental effects on the growth of the blood vessel structures, as suggested by Okubo,[18] the influences will appear in old age, when cardiovascular diseases become the major cause of death. Figure VI shows the rate of mortality increase with age for two major causes of death in old age, cardiovascular diseases and neoplasms, for males of the Federal Republic of Germany. A shift of *k(x)* with cohorts as seen in figure I for all causes combined can be found for cardiovascular diseases, except for data in 1964 that do not fit the shifting pattern very well. On the other hand, substantial variations of cohort origin do not appear at all for cancers.[19]

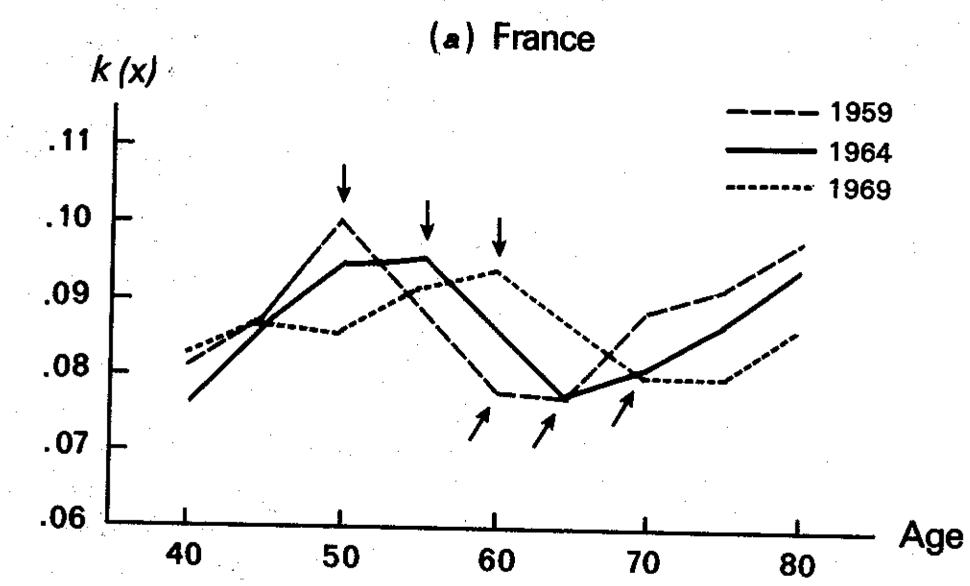
Secondly, the impact of nutritional deprivations in young childhood on the growth of the blood vessel structures may possibly be compensated to some extent by better nutrition in later years of physical growth. However, damages due to teenage undernourishment may tend to remain permanently since they are in the final stages of the major physiological development.

Thirdly, female adolescents may be less vulnerable to malnutrition because in general, females are capable of storing more fat in their bodies than males. These discussions are only tentative and further research on the cohort mortality patterns seems necessary from medical viewpoints.

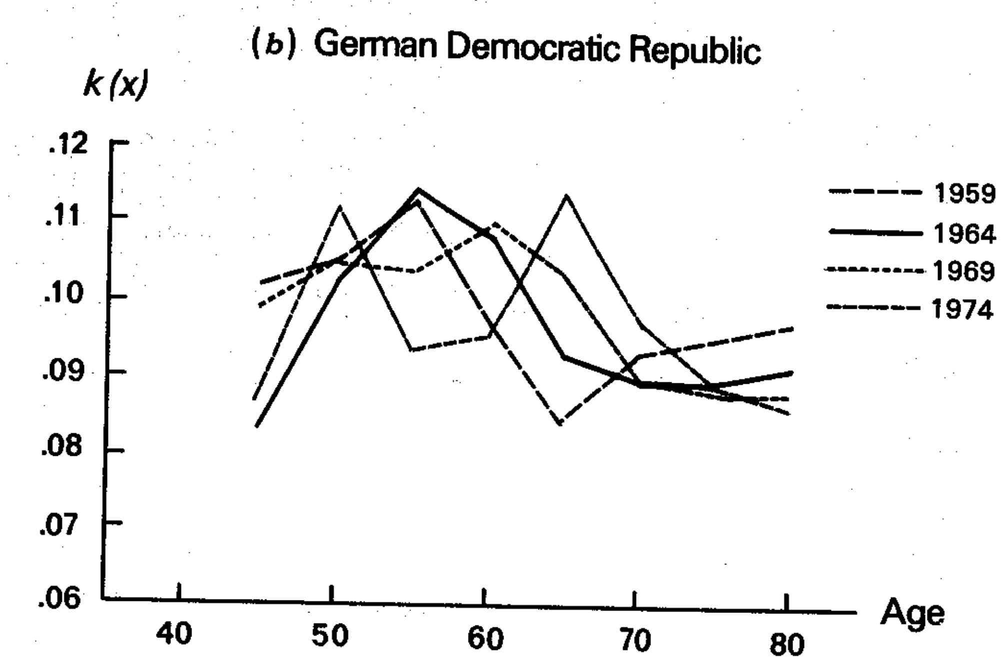
On the other hand, the cohort mortality patterns found in the present paper may be explained as an instance of age misstatement. Some young males in those days, perhaps with the help of their parents, might have succeeded in understating their ages in order to avoid or defer military service during the War, and remained in the younger cohort throughout their lives, thereby keeping the reported mortality of the cohort biased upward.

The age transfer must have kept the sex ratio (male/female) of the younger cohort higher than expected after the First World War until the draft of middle-aged males near the final stage of the Second World War and loss of their lives in battle lowered the sex ratio drastically. Therefore, the age-specific sex ratio at the 1933 German census is expected to show a trace of the transfer of males to the younger cohorts. As presented in column (4) of table 3, the sex ratio decreases with age gradually but very slightly from 20 to 33, as generally expected from the usual pattern of excess male mortality, then drops beginning with age 34, that is, age 19 at the end of the Second World War, reflecting the loss of young males during the War. The sequence of sex ratios at the 1933 census

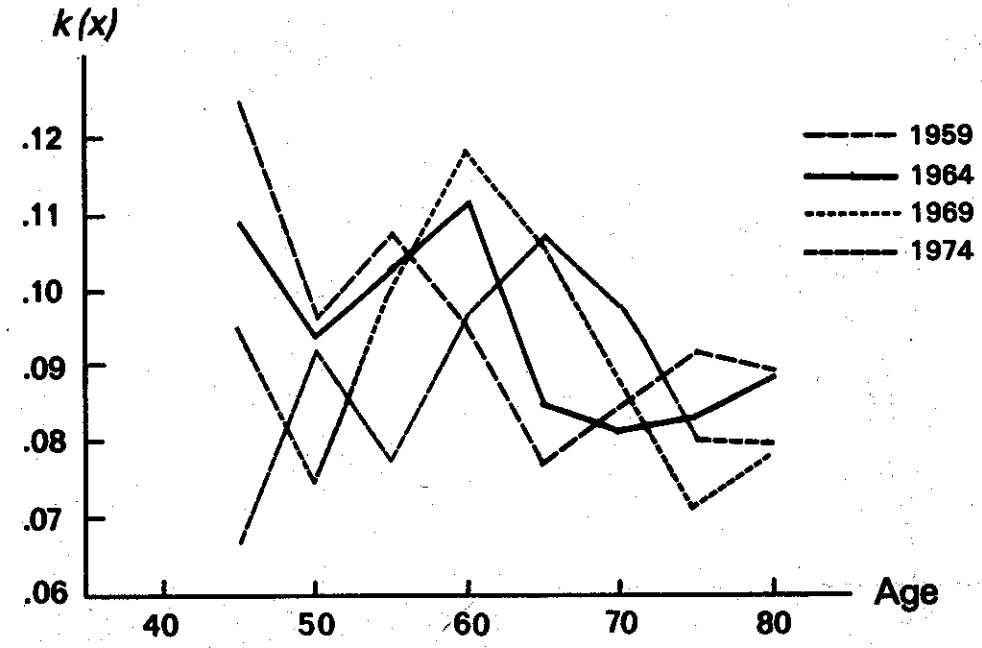
Figure V. Rate of mortality change with age, $k(x)$, for males, in selected countries



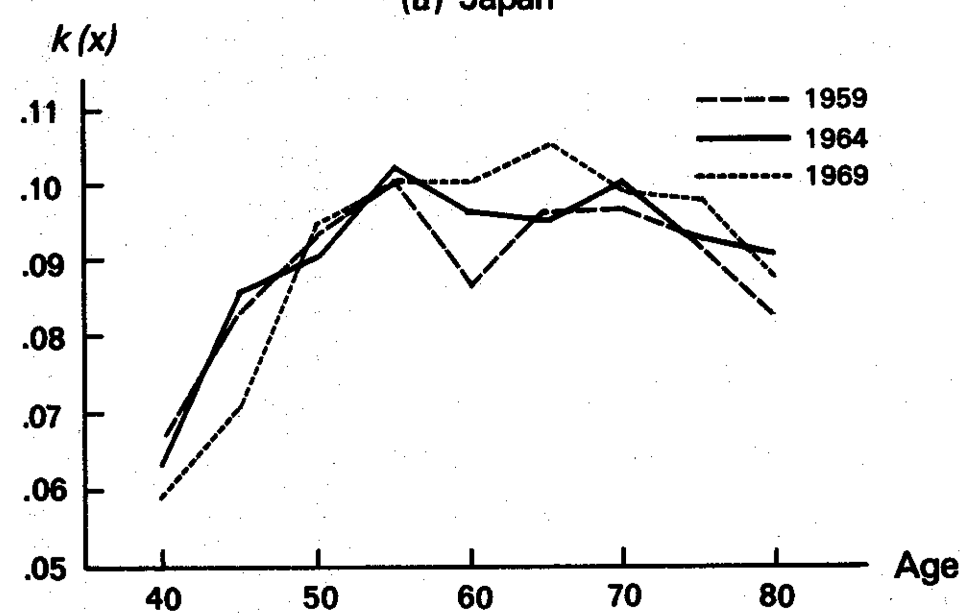
NOTE: The upward-pointing arrows and the downward-pointing arrows indicate the positions of the cohorts born in 1899 and 1909, respectively.



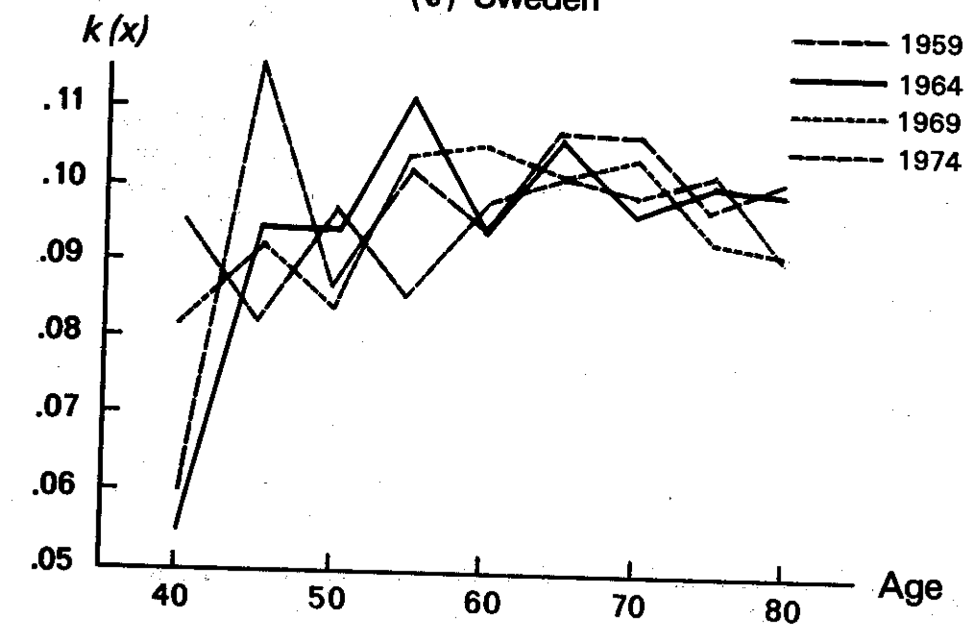
(*c*) Austria



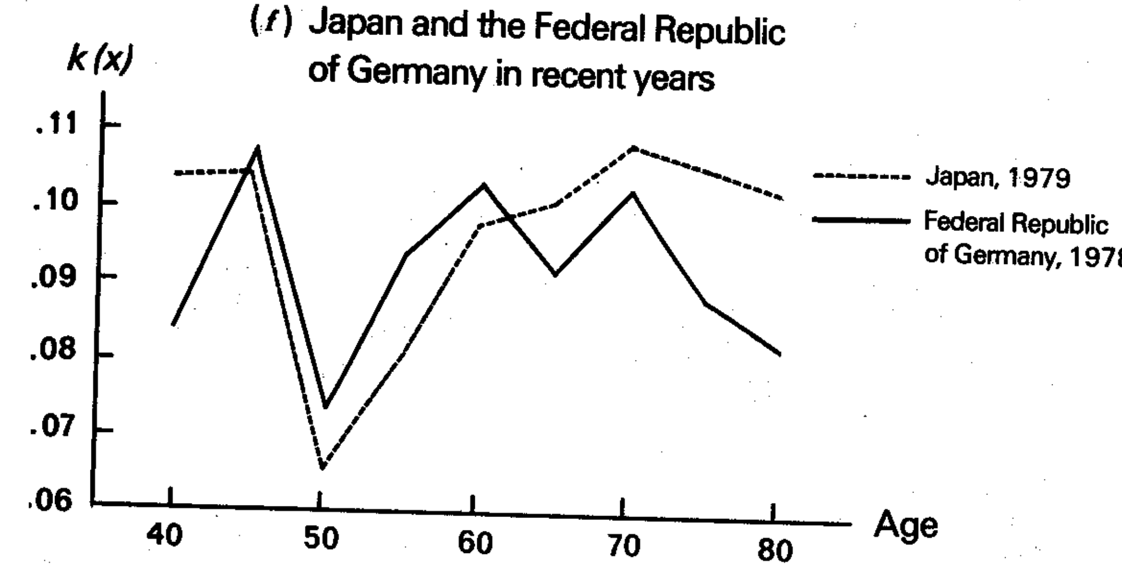
(*d*) Japan



(*e*) Sweden



(*f*) Japan and the Federal Republic of Germany in recent years



NOTE: Computed from the following sources: *Demographic Yearbook 1966* (United Nations publication, Sales No. 67.XIII.1); *Demographic Yearbook 1974* (United Nations publication, Sales No. E/F.75.XIII.1); *Demographic Yearbook, Special Issue: Historical Supplement* (United Nations publication, Sales No. E/F.79.XIII.8); Japan. Statistics and Information Department, Ministry of Health and Welfare, *Vital Statistics 1979, Japan* (Tokyo, Ministry of Health and Welfare, 1981); Federal Republic of Germany, *Statistisches Bundesamt, Statistisches Jahrbuch 1980 für die Bundesrepublik Deutschland* (Herausgeber, *Statistisches Bundesamt*, 1980).

TABLE 3. SEX RATIO AT BIRTH BY YEAR OF BIRTH AND SEX RATIO BY AGE AS OF 31 DECEMBER 1933, FOR THE FEDERAL REPUBLIC OF GERMANY

Year of birth (1)	*Sex ratio at birth* (2)	*Age on 31 December 1933* (3)	*Sex ratio on 31 December 1933* (4)
1912	1.0594	21	1.0107
1911	1.0554	22	1.0018
1910	1.0528	23	1.0031
1909	1.0531	24	0.9989
1908	1.0543	25	0.9988
1907	1.0565	26	0.9997
1906	1.0542	27	0.9947
1905	1.0565	28	0.9947
1904	1.0548	29	0.9922
1903	1.0516	30	0.9890
1902	1.0521	31	0.9944
1901	1.0547	32	0.9985
1900	1.0554	33	0.9863
1899	1.0530	34	0.9268
1898	1.0517	35	0.8766
1897	1.0528	36	0.8430
1896	1.0520	37	0.8071
1895	1.0524	38	0.7903
1894	1.0513	39	0.7757
1893	1.0539	40	0.7822

Source: Germany, *Statistischen Reichsamt*, 1936, *Volkszählung: Die Bevölkerung des Deutschen Reichs nach den Ergebnissen der Vokszählung 1933, Heft 2: Geschlecht, Alter und Familienstand der Bevölkerung des Deutschen Reichs* (Berlin, Paul Schmidt).

seems to reveal no strong indication of age transfer of males to cohorts about age 16 at the end of the War.[20]

However, the birth cohorts of 1902 and 1901 that were aged 31 and 32 at the end of 1933 and thus 16 and 17 at the end of the War perturb, though only slightly, the tendency of gradual slow decline of the sex ratio with age. This may be an indication of age transfer, although the perturbation seems partly attributable to variations in sex ratio at birth. As shown in column (2) of table 3, those cohorts have slightly higher sex ratios at birth than the cohort of 1903.

Even if the relatively high sex ratios of these cohorts reflect some age transfer that really occurred, they seem too small to explain the observed size of cohort mortality variations. For instance, it can be shown by simple calculations that, if about one quarter of the cohort is in fact three years older than the age they report, then the observed death rate of the cohort is about 8 per cent higher than what it really is.[21] Note that an 8 per cent difference in death rates is slightly smaller than what is implied by the differences in the estimated cohort factors between the high-mortality cohort of 1899-1904 and the cohorts that are 10 years younger and older than the cohort. However, no trace of such a large size of transfer of males among age groups is found in table 3.

In addition, the fact that similar mortality patterns are observed among the First World War survivors in France and the Second World War survivors in Japan and the Federal Republic of Germany seems to make the hypothesis of age misstatement even less plausible, since the difficulty in avoiding military service by understating ages may change over time and vary among countries.

Besides the above-mentioned interpretations, that is, nutritional deprivation and age transfer, at least four other hypotheses seem to provide partial accounts for the cohort mortality patterns and thus are worth considering. First, the mortality of a certain cohort appears relatively high if the mortality of the preceding and succeeding cohorts is kept low for some reasons. It should be noted that the high-mortality cohort found in the present study is located between the generation of World War soldiers and those who were young children during the War, both of which may have experienced low mortality after the War for the following reasons. Namely, some soldiers died in combat and some young children, in particular, infants, died due to malnutrition and poor hygiene during the War, thereby making the survivors of the two generations a group of relatively strong persons who managed to survive under very difficult conditions. Cohorts between the two generations, on the other hand, may include a higher proportion of unhealthy persons, who push up the death rate for the cohorts.

This interpretation, however, has a few limitations. Although the hypothesis can differentiate the mortality of the cohort that was adolescent at the end of the War from that of the soldier generations, it does not fully explain the difference between the high-mortality cohort and the succeeding generations. If this "selection" explanation were valid, we might expect substantial cohort variations in female mortality, since malnutrition and poor hygiene are considered to strike both male and female children, as far as their short-term impacts on mortality are concerned. This expectation is not met by our data analysis results as previously shown. In addition, this interpretation does not provide a full explanation of the exact timing of the cohort variations. The cohorts between soldiers and young children spread over more than 10 years of age, as shown in figure IV, and thus the fact that the centre of the high-mortality cohorts was about age 15 and 16 at the end of the War remains unsolved.

Secondly, it may be speculated that the variations in cohort mortality are related to the post-War variations in cohort size. After many soldiers had lost their lives in the War, the survivors could enjoy the advantages of the reduced cohort size when they returned to civilian life. The cohorts several years younger, on the other hand, were significantly larger, so that they had to experience greater competition and more stress throughout their lives, resulting in higher mortality than the cohort of combat survivors. This explanation, however, does not agree very well with the fact that the larger cohorts experienced high mortality at old age even in their less competitive post-retirement life styles.

Thirdly, suppose that a particular type of weapon that has a long-term impact on health were introduced near the end of the First World War. If so, the last and youngest group of recruits who joined the armed forces after the older cohorts had lost many soldiers might have been the primary target of the weapon. The fact that poison gas was used for the first time in 1915, however, produces difficulties in finding weapons that satisfy the above condition. Moreover, this hypothesis does not seem very successful in accounting for the similarities in cohort mortality between survivors of the First World War and those of the Second World War.

Finally, it can also be speculated that cohort variations in cigarette smoking are related to the cohort mortality patterns. The habit of cigarette smoking, which, in the old days, was restricted only to small segments of societies, is considered to have spread widely early in this century. The armed

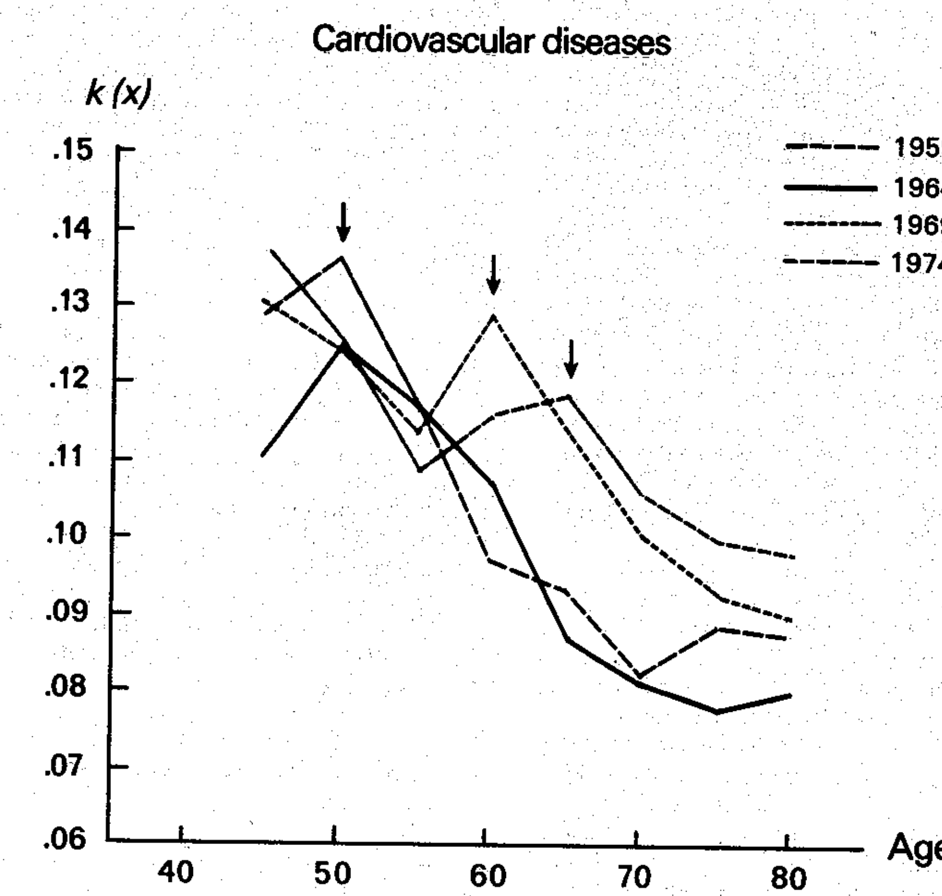
services in the First World War might have spurred the diffusion of the habit. Cigarette smoking is known to be related to various types of cancers and cardiovascular diseases[22] and it often generates strong cohort variations in mortality.[23] Thus, the question may be raised whether the cohort variations in agreement with the timing of the First World War reflect some impacts of cigarette smoking.

The diffusion of cigarette smoking, however, would rather cause an upturn of the cohort mortality factor than boost the death rate of a particular cohort relative to that of the preceding and succeeding generations. There seems to be no strong reason to expect only teenagers at the end of the wars to become the heaviest smokers. In addition, this interpretation, as well as the "special weapon" interpretation, seems less successful in explaining the cohort variations of the Second World War survivors than those of the First World War survivors.

Therefore, although the four interpretations described above may agree with certain elements of the results, none of them seem to provide a full account of all the important aspects of the mortality patterns. Although it is too early to discard these different interpretations, the "malnutrition" hypothesis seems to stand more plausible than the "age-transfer" account and the other four explanations, at least for the time being.

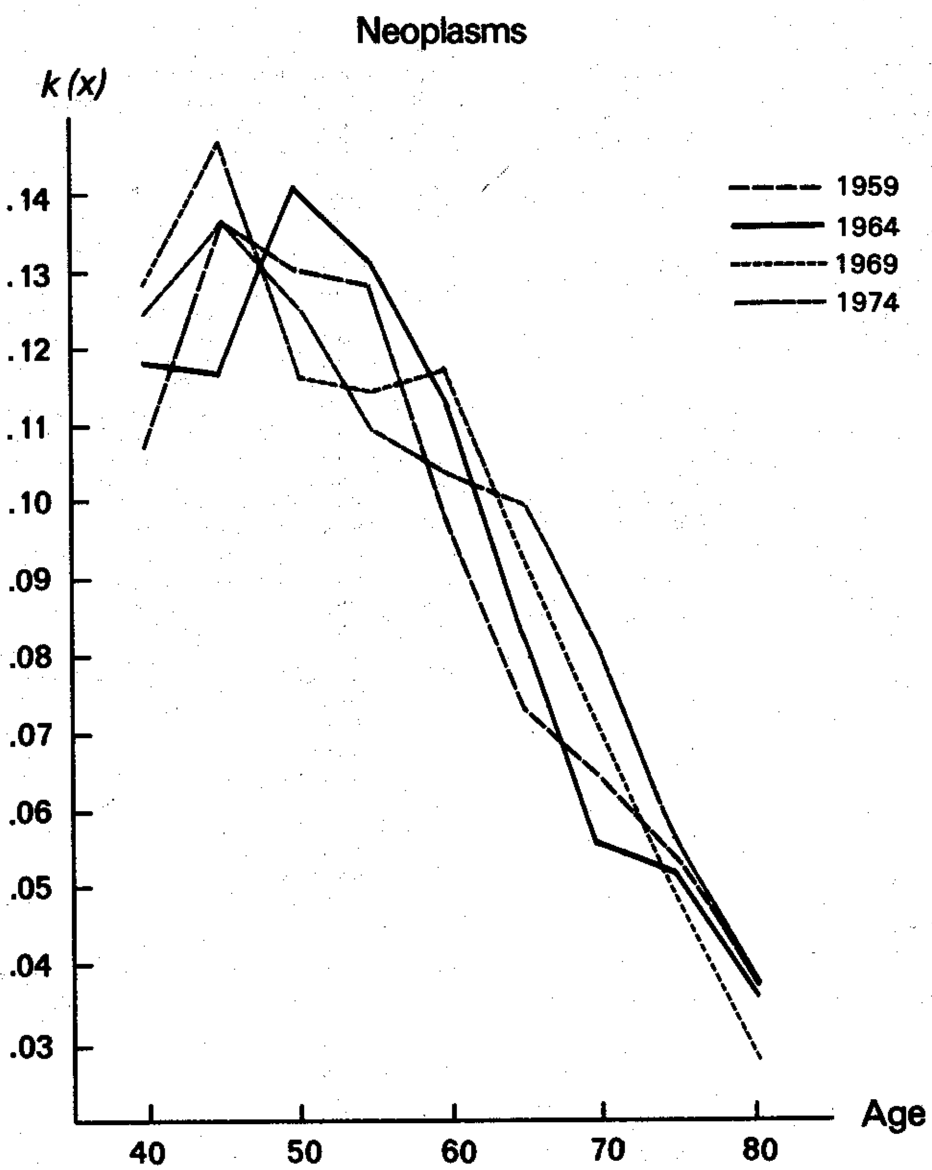
In summary, cohort mortality variations that perhaps reflect long-term impacts of the First World War on health have been found among older males in the Federal Republic of Germany. The findings seem to shed light on the importance of a research topic to which relatively little attention been given, that is, the long-term influences of catastrophes, including famine, drought and epidemic, as well as warfare, upon the mortality of the survivors, and in particular, the effects of nutritional deprivations experienced under those conditions. The results of the present study suggest that further research on this subject should be conducted.

Figure VI. Rate of mortality change with age, *k (x)*, for males, Federal Republic of Germany, 1959, 1964, 1969 and 1974: cardiovascular diseases and neoplasms



NOTE: The downward-pointing arrows indicate the positions in 1959, 1969 and 1974 of the cohort born in 1909.

Figure VI. *(continued)*



[1] Masakazu Okubo, *Increase in Mortality of Middle-Aged Males in Japan* (Tokyo, Nihon University Population Research Institute, 1981).

[2] Shiro Horiuchi and Ansley Coale, "Age patterns of mortality for older women: an analysis using the age-specific rate of mortality change with age", paper presented at the 1983 Annual Meeting of the Population Association of America at Pittsburgh, Pennsylvania.

[3] Researchers in population studies may find that $k(x)$ is computed in the same way as the population growth rate (r) by replacing population increase over time with mortality increase with age.

[4] Five-year age group data are quite useful for the $k(x)$ analysis, since sequences of $k(x)$'s obtained from single-year age group data tend to be more or less erratic so that some smoothing techniques need to be applied.

[5] Death rates for ages under 35 were not included in the analysis for four reasons. First, those who were under age 35 in 1959 were born after the First World War. Secondly, the main focus of the present research is on mortality in old age. Thirdly, the number of deaths for the five-year age groups 15-19, 20-24, 25-29 and 30-34 tend to be small because of very low mortality in these ages and thus susceptible to stochastic variations. Finally, although mortality tends to increase with age above age 35, it is not necessarily the case for ages under 35. Mortality is likely to decrease with age in childhood and, in some populations, there is a local peak of death rate in young adulthood ages. Thus, in order to avoid unnecessary complexities that may result from the inclusion of these different age patterns of mortality in the analysis, the present research was confined to the age groups in which age variations of mortality appear to be dominated mainly by the physiological deterioration of the human body that proceeds with age.

[6] There are four other alternative ways to choose a set of calendar years in five-year intervals. These years, that is, 1959, 1964, 1969 and 1974, were chosen on the basis of our preliminary analysis of data by single-year age groups in such a way that the centre of the high-mortality generation is always located at the centre of a five-year age group.

[7] Goldstein has criticized the model on the ground that, since it is logically impossible to vary either age, period or cohort while holding constant the other two, results of data analysis using the model may be illogically conceived. (H. Goldstein, "Age, period and cohort effects—a confounded confusion", *Bulletin in Applied Statistics*, vol. 6, No. 1, 1979, pp. 19-24.) In the present research, however, age, period and cohorts can be considered as proxies for some mortality determinants that are logically separable from each other. Aging proceeds with physiological deterioration of the human body. Period factors represent health-related environmental variables, including the level of available medical technology and the accessibility to health services. Cohort factors are considered to reflect impacts of past experience on current health conditions. In fact, age factors, period factors and cohort factors are of our research interest as surrogates of impacts of aging, environment and past experience, respectively, on mortality. Since these mortality determinants can be considered to exert their influences more or less independently of each other, the use of model (4) does not seem totally inadequate for the purposes of the present study.

[8] G. A. Sacher, "Analysis of life tables with secular terms", *The Biology of Aging*, American Institute of Biological Sciences Symposium, No. 6.253, 1960.

[9] J. C. Barrett, "Age, time and cohort factors in mortality from cancer of the cervix", *American Journal of Hygiene*, vol. 71, 1973, pp. 253-259; "The redundant factor method and bladder cancer mortality", *Journal of Epidemiology and Community Health*, vol. 32, 1978, pp. 314-316; "A method of mortality analysis: application to breast cancer", *Revue d'Epidémiologie et de Santé Publique*, vol. 26, 1978, pp. 419-424; "Cohort mortality and prostate cancer", *Journal of Biosocial Sciences*, vol. 12, 1980, pp. 341-344.

[10] Model (4) is under-identified unless some additional restrictions are imposed on it. Since the age, period and cohort factors are interrelated such that $k = 10 + j - i$, they are computationally inestimable.

A simple way to make the model estimable is to assume that two or more of the factors are equal. In the present research, however, it does not seem valid to assume any pair of age factors or period factors to be equal, since substantial variations of mortality with respect to age and period are expected on the basis of previous research findings. Mortality in middle and old ages increases steeply with age, so that no pair of age factors should be set equal. It was also decided to pose no restrictions on period factors prior to the data analysis, because developed countries are known to have experienced a substantial decline of mortality after the Second World War, with some slowdown in the 1960s.

On the other hand, although cohort variations are the main subject of the present study, our focus is not necessarily on all observed cohorts. The study of Okubo (note 1) has shown that effects of the Second World War on mortality began to appear in late middle age, suggesting that the long-term impacts of warfare on the mortality of the survivors in their younger ages may be minor. The factors for the two youngest cohorts (δ_{12} and δ_{13}) that were under 45 during the study period (1959-1974) are therefore set to be equal to each other. Although this still is a strong assumption, the choice of the two factors seems more reasonable than the other alternatives.

[11] A few other methods for detecting cohort variations in mortality were considered. First, age-specific death rates observed in the study population can be compared to those in the model life tables. Since the model life tables show representative age patterns of mortality on the basis of existing national life tables of reliable quality, it is expected that deviations from model life tables indicate cohorts with relatively high or low mortality. The comparative analysis, however, did not satisfy the expectation because the age patterns of male adult mortality in many developed countries in the 1960s and the 1970s are systematically different from those of existing model life tables that are mostly based on national life tables before 1960 (for example, *Age and Sex Patterns of Mortality: Model Life Tables for Under-Developed Countries*, United Nations publication, Sales No. 1955.XIII.9; A. J. Coale and P. Demeny, *Regional Model Life Tables and Stable Populations*, Princeton, Princeton University Press, 1966). Death rates of older males in developed countries during the last two decades tend to be higher than those extrapolated from those of younger males on the basis of the age patterns of mortality in the model life tables, perhaps reflecting the increasing effects of smoking on old-age mortality in the recent periods (S. Preston, "An international comparison of excessive adult mortality", *Population Studies*, vol. 24, No. 2, 1970, pp. 5-21). Therefore, deviations from model life tables due to cohort variations tend to be masked by the departures due to the excessive old-age mortality for males.

Second, if cohort variations are different between males and females, the sex ratio of death rates may be unexpectedly high or low at some ages, indicating male or female cohorts of relatively high or low mortality. There is, however, a strong age pattern of sex ratio of death rates widely found in West European countries, including the Federal Republic of Germany, with a peak about age 60 to 64 (see A. D. Lopez, "The sex mortality differential in developed countries", in A. D. Lopez and L. T. Ruzicka, *Sex Differentials in Mortality: Trends, Determinants and Consequences*, Canberra, Australian National University Press, 1983, pp. 53-120; and *Levels and Trends of Mortality since 1950*, United Nations publication, Sales No. E.81.XIII.3, figure II.7). This age pattern is quite strong and dominates the sex ratio of death rates for the elderly of the Federal Republic of Germany, masking most variations of cohort origin.

[12] The cohort variation in $k(x)$ among males of the Federal Republic of Germany was initially discovered by Ansley Coale, when he and the author were conducting a study on old-age mortality at the Office of Population Research of Princeton University.

[13] The model of geometric increase of adult mortality has been proposed by G. Gompertz and elaborated by Strehler and Mildvan, and Abernethy. See B. L. Strehler and A. S. Mildvan, "General theory of mortality and aging", *Science*, vol. 132, 1960, pp. 14-21; J. D. Abernethy, "The exponential increase in mortality rate with age attributed to wearing-out of biological components", *Journal of Theoretical Biology*, vol. 80, 1979, pp. 333-354.

[14] R. Schoen, "The geometric mean of the age-specific death rates as a summary index of mortality", *Demography*, vol. 7, 1970, pp. 317-324.

[15] L. Grebler and W. Winkler, *The Cost of the World War to Germany and to Austria-Hungary* (New Haven, Yale University Press, 1940), pp. 81-82.

[16] F. Bumm, ed., *Deutschlands Gesundheitsverhaeltnisse unter dem Einfluss des Weltkrieges* (Berlin, *Deutsche Verlago-Anstalt*, 1928), vol. 2, p. 22.

[17] Suda L. Bane and Ralph H. Lutz, eds., *The Blockade of Germany After the Armistice 1918-1919* (Stanford University Press and Oxford University Press, 1942), p.v.

[18] Okubo, *op. cit.*

[19] Unfortunately, the classification of causes of death changed during the study period. The change was substantial with respect to cardiovascular diseases, thereby making a further time-series analysis of cardiovascular mortality in more detail very difficult to conduct in a rigorous manner.

[20] The historical data shown in table 3 were kindly provided to the author by John Knodel and Neil Bennett at the Population Studies Center of the University of Michigan.

[21] The calculation has been made under the assumption that the rate of mortality increase with age is 0.093, the median value of all $k(x)$'s in figure I.

[22] United States Public Health Service, *Smoking and Health: A Report of the Surgeon General* (Washington, D. C., Department of Health, Education and Welfare, DHEW publication No. PHS79-50066, 1979); S. H. Preston, *Older Male Mortality and Cigarette Smoking: A Demographic Analysis* (Berkeley, University of California, Institute of International Studies, Population monograph series No. 7, 1970).

[23] Substantial cohort variations have been found both in exposure to cigarette smoking (for example, United States Public Health Service, *The Health Consequences of Smoking for Women: A Report of the Surgeon General* (Washington, D. C., Department of Health and Human Services, United States Government Printing Office, 1980 0-326-003, 1980)) and lung cancer mortality (for example, R. A. M. Case, "Cohort analysis of cancer mortality in England and Wales, 1911-1954 by site and sex", *British Journal of Preventive and Social Medicine*, vol. 10, 1956, pp. 172-199).