

8. INTERNAL COMPARISONS—THE TIME FACTOR

8.1 Introduction

The question of whether a certain work site in the smelter can increase the individual's risk of dying from a specific cause can be analysed by means of *internal comparisons*. There are, however, a series of difficulties in this type of analysis, primarily connected with the time aspect. The environment has not remained constant and the individuals have been of different ages when employed at various work sites. It is not evident whether a hazardous area of exposure would have the same effect on a worker at the age of 30 as on an older individual. A hazardous exposure on a certain individual during a specific time period may not be visible until much later.

Three different types of time-related notations will be used for each individual:

Latency time, here the period from initial employment (exposure) to death. Analogously, latency time can be calculated with respect to a certain work site within the smelter.

Exposure time, i.e. the total time that an individual has been employed at the Rönnskär smelter. Exposure time can also be confined to a certain work site within the smelter.

Calendar time, i.e. at which time (30s, 40s etc) a certain exposure took place.

We have chosen to use the expression 'latency time' in the same manner as in certain works of occupational medicine, e.g. Selikoff (1975). Of course, the illness need not necessarily have been present (latent) in an individual during the whole time period in the same way as a contagious disease for example, where, traditionally, the latency time is the time interval between the transmission and the outbreak of the disease.

8.2 Latency time

It takes a long time for certain chronic diseases to develop. Therefore, it need not follow that the cases which

occur at the beginning of a certain employment period have been caused by the particular occupational exposure connected with that job. Conversely—if a case of such an illness has experienced a very long exposure, it need not be the last part of the exposure time that played an etiological role.

The lung cancer cases in the Rönnskär cohort are all plotted in Fig. 8.1 a) and b). The four lines in Fig. 8.1a demarcate cases who have died within 0, 5, 10 and 15 years of employment. The three cases who died less than 15 years after first employment, were individuals first employed at Rönnskär when they were comparatively old, and it does not seem plausible that their illness was caused by occupational exposure at the smelter. As can be seen, these deaths occurred early in comparison with the other lung cancer deaths in the cohort.

The so-called latency time effect has been studied by simply disregarding the first years (deaths as well as person-years) in the statistical analysis. SRR values have been calculated, utilizing the age distribution of the entire cohort, eliminating the first 5, 10, 15 and 25 years of follow-up. This does not result in any large effects in terms of total cancer mortality but, for lung cancer, we observe an increase of 5% when truncating the first 15 years and a 9% increase at a 25-year truncation. The rate ratios become even more sensitive to changes in latency times when we only consider older age groups. If, for example, an analysis is based upon a follow-up of >15 years, both the healthy worker effect and the early cases not caused by occupational exposure are reduced. The consequence of such a procedure would necessitate the adjustment of the external comparisons in Fig. 7.3. Thus, when comparing with the entire Swedish male population we get an adjusted SMR = 122 instead of 116, while SMR = 138 for the total cancer mortality and SMR = 278 for lung cancer instead of 134 and 263 respectively. It would, perhaps, be more important to consider different latency times when studying the mortality rates connected with various work sites within the smelter.



Fig. 8.1. The latency times of the 76 lung cancer cases with regard to age and calendar time (at employment and death respectively).

8.3 Exposure time

A statistical association between exposure time and death rate does not always lead to a self-evident conclusion. For example, it could be that the longer a person has been subject to a certain exposure, the greater are his chances of dying from a specific cause. The display of such an association is obviously influenced by the fact that a person could not have had a long exposure time unless he had a long survival time, and the exposure time could not have been very long if he had a short survival time.

Death specific SRR values have been calculated for non-exposed (less than 5 years) and exposed (5 years or more). In accordance with the PAR definition, the accumulation of person-years for the exposed group has begun

five years after employment. Since this group is numerically larger, it dominates the cohort and its SRR values can not be expected to be far from 100. The pattern for those employed for less than five years, is of interest. With regard to the overall mortality, the non-exposed group has an SRR value of somewhat larger than 100, which is in accordance with the mechanism we have just described. Therefore, it is especially remarkable that the cause-specific SRR values for cancer and for cerebrovascular diseases (neglecting the first 15 years) are considerably smaller for the non-exposed than for the exposed group. There appears to be a risk reduction with regard to cancer and cerebrovascular diseases for those employed less than five years (Fig. 8.2).

8.4 Calendar time

It is obviously of great importance to find out whether the environmental changes have resulted in health improvements.

The analysis of time trends in this context is, however, complicated by the facts that individuals are ageing and that some deaths due to exposure occur after a very long time.

We have already demonstrated (Figs. 7.1-7.2) that cancer mortality, especially lung cancer mortality, has increased more among Rönnskär workers than among the surrounding populations. This does not necessarily reflect environmental changes but probably effects of latency time. We have therefore defined sub-cohorts by using the variables birth year and employment year.

Two different cohorts have been analysed separately: workers born before 1910 and workers born 1910 or after. A comparison between these two cohorts can, at least in part, reflect the changes in the work environment, since

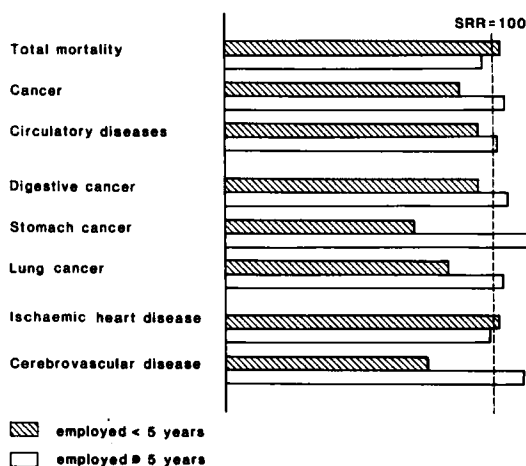


Fig. 8.2. Directly standardized mortality rate ratios (SRR) for various categories with regard to cause of death and exposure. For cancer diseases, a latency time of 15 years has been applied. (SRR = 100 for the whole Rönnskär cohort).

Table 8.1

Directly standardized mortality rate ratios (SRR) for various causes and birth cohorts. (Ages 30–64 years). A latency time of at least 15 years has been applied for all cancer diseases. (SRR = 100 for the whole Rönnskär cohort)

Cause of death	SRR for workers born	
	–1909	1910–
Total mortality	100	98
Cancer	100	98
Digestive organs	93	113
Lung cancer	105	93
Circulatory diseases	94	102
Ischaemic heart diseases	83	111
Cerebrovascular diseases	92	100

Table 8.2

Directly standardized mortality rate ratios (SRR) for various causes of death and employment cohorts. (Ages 15–74 years). For cancer diseases, a latency time of at least 15 years has been applied. (SRR = 100 for the whole cohort)

Cause of death	SRR for those first employed		
	–1939	1940–49	1950–
Total mortality	103	104	88
Cancer	97	119	60
Digestive organs	105	111	60
Lung cancer	103	129	54
Circulatory diseases	103	101	97
Ischaemic heart diseases	96	103	109
Cerebrovascular diseases	106	109	99

those born before 1910 have very rarely been exposed to the 'cleaner' environment of the sixties and seventies. The cohorts are, however, not completely comparable: Those born 1910 or after are no older than 66 years at the closing date for data collection in this study. Furthermore, the workers in this group were on average older at the time of their employment, which means that they contribute fewer person-years to the lower age groups. We will therefore only compare the mortality pattern for the age interval 30–64 years.

Table 8.1 shows that the groups do not differ greatly with regard to total mortality. The same is true for the overall cancer mortality but, for cancer of the digestive organs and lung cancer, there are differences—in opposite directions. The question is whether the declining lung cancer mortality rate indicates that the younger cohort has experienced a

Table 8.3

Directly standardized mortality rate ratios (SRR) for different cancer diseases and employment cohorts. Latency period 15–24 years. (SRR = 100 for the total Rönnskär cohort)

Cause of death	SRR for those first employed		
	–1939	1940–49	1950–
Cancer	117	95	71
Digestive cancer	140	51	83
Lung cancer	103	99	67

somewhat less hazardous work environment. An argument for this interpretation is that the tendency is completely contrary to findings for the total population. The increasing mortality from circulatory diseases is, however, in accordance with the development for the entire population.

As an alternative, we have made a comparison between three employment cohorts, namely those employed before 1940, between 1940 and 1949, and 1950 and thereafter (see also Fig. 1.3). Age-specific mortality rates for each cohort have been weighted, using the total Rönnskär cohort as standard population. A latency time of 15 years has been applied for cancer diseases.

We have the impression from Table 8.2 that there is a considerably reduced mortality among those employed 1950 or later, especially with regard to cancer. It is necessary to recall, however, that the follow-up period for this cohort is at most 26 years (of the total group of 76 lung cancer cases in the cohort, only 19 occurred within 25 years of first employment). A further analysis was therefore made, applying a latency period of 15–24 years to all three cohorts (Table 8.3).

The declining trend in Table 8.3 must be interpreted with caution. It should be observed that the employment age was lower (median age 24 years) among those employed after 1950 compared to those employed in the thirties (median age 29) and in the forties (median age 27 years). This might contribute to a longer latency time for those employed in the fifties.

Has the cancer mortality rate among Rönnskär workers employed after 1950 been higher than expected on the basis of official statistics? Prior to 1977, there were only 20 cancer cases in this group, of which four had lung cancer. These numbers are too small for further calculations. Using official statistics, 22 cancer cases were expected, of which 3.4 lung cancers. As already pointed out, the latency time was 26 years at the most. A continued follow-up of the actual cohort is thus motivated.