RISK OF CANCER IN THE PRIMARY ALUMINIUM INDUSTRY: AN APPRAISAL

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INTRODUCTION

The last 50 years have seen dramatic improvements in the health of developed countries which have been brought about partly by increased scientific knowledge of the causes of disease and the ways in which disease can be prevented or cured, and partly by an increased standard of living and the spread of education. Mortality in infancy, childhood, and early adult life, in particular, has been so diminished that more than half of all loss of expectation of life under 85 years of age now occurs in the 55 to 74 year age group, and any further major progress in prolonging expectation requires an attack on those diseases that are principally responsible for death at these ages. In all Western countries, these are now ischaemic heart disease and neoplasms. In both England and Wales and in Norway each now accounts for nearly a third of all such deaths. It is not surprising, therefore, that so much attention should be concentrated on cancer: not because the risk of developing it at any particular age has increased, but because so many other diseases have been controlled that it is now relatively much more important.

That there might be an occupational hazard of cancer in the primary aluminium industry was realised by Kreyberg (1959) 25 years ago, when he drew attention to the presence of benzo(a)pyrene in the air of potrooms. Benzo(a)pyrene,

he believed, was likely to have been a cause of excess mortality from lung cancer in makers of coal gas while the same chemical (or group of chemicals) derived from the combustion of coal might also cause part of the excess mortality from lung cancer that was commonly found in urban dwellers compared to rural, and it seemed only logical that it should imply a specific hazard in the primary aluminium industry.

It was not, however, until 1971 that the first report was published suggesting that aluminium production workers did in fact suffer from an excess of lung cancer and perhaps also of skin cancer (Konstantinov and Kuzminykh, 1971). Since then many other papers have been published, the results of which have variously suggested that aluminium production workers might also specifically suffer from a wide range of other cancers or, conversely, that they might suffer no unusual hazard of cancer at all.

CHEMICAL EXPOSURES

The materials to which aluminium production workers are liable to be exposed in unusually large amounts as a result of their work are listed in Table 1. The list is taken from the IPAI Health Committee's (1982) review of the measurement of employee exposures in aluminium reduction plants. This classifies the materials in three groups, based on the likelihood of their contributing significant occupational exposure. Chemicals of primary importance are those met frequently and have high or low toxicity, while chemicals of secondary importance are those met infrequently or are

judged to have a very low toxicity. Chemicals of tertiary importance are seldom encountered, but are potentially hazardous because of their high toxicity. Another list, compiled more recently by the International Agency for Research on Cancer (1984), is identical save that it adds petroleum pitch to coal tar pitch, specifies cryolite and polynuclear aromatic compounds separately, adds cyanides and sodium hydroxide to the second category, and relegates asbestos to the second category from the first.

POTENTIAL CARCINOGENS

Of these materials, coal tar pitch and asbestos are known to be carcinogenic to Man: beryllium, cadmium, oil mists, and welding fumes may be; and lead can cause renal tumours in high doses in animals. For our present purpose, the last five can be ignored. Many workers have been heavily exposed to lead in other circumstances without apparently running any material risk of cancer (International Workshop on the Carcinogenicity of Metals, 1981) and the risks attributable to the other four materials, most of which are encountered only seldom in the aluminium industry, are still sub judice and are certainly relatively small. In each case the most probable hazard is cancer of the lung. The evidence is strongest for beryllium; but the conditions that may have produced lung cancer in beryllium workers occurred only during the war and were certainly extreme (Mancuso, 1979; Saracci, 1984). If welding fumes are hazardous, the risk is most likely to be associated with the welding of stainless steel, possibly because of its

content of nickel. Cadmium was at one time thought also to cause cancer of the prostate (International Workshop on the Carcinogenicity of Metals, 1981) but is now thought not to (Doll, 1984).

Asbestos

Whether asbestos has caused a hazard in aluminium reduction plants depends on the number of fibres released. I have no figures to indicate what this may have been but unless hundreds of men have been persistently exposed to average concentrations of more than 1 fibre ml^{-1} for many years, it is most unlikely that any risk of lung cancer attributable to asbestos could be detected. Asbestos also causes pleural and peritoneal mesotheliomas and may possibly cause cancers of the larynx, gastro-intestinal tract, and kidney. Mesotheliomas are produced less often than lung cancer, but they are so seldom produced by any other cause that the occurrence of even one case in an aluminium production worker would suggest that an asbestos hazard might exist. So far as I know, no such case has been reported. In the absence of a clear risk of lung cancer or mesothelioma due to asbestos, the possibility of a hazard of other types of cancer due to its inhalation can be ignored.

Pitch

We are left, therefore, with a potential hazard from exposure to pitch made from coal tar or petroleum. Both types consist predominantly of high molecular weight hydrocarbons, but only coal tar pitch contains any large amount of polycyclic aromatic hydrocarbons - the amount in

it being measured in percentage quantities while petroleum pitch contains only a few parts of them per million: that is, four orders of magnitude less. We may, therefore, ignore the latter, as both the chemical and the epidemiological data point to coal tar pitch as being the one likely cause of hazard.

Men have long been exposed in other industries to coal tar pitch, and more commonly still to the tar from which the pitch is derived, and a substantial literature exists relating these exposures to the risk of cancer. This shows unequivocally that three types of cancer have been produced by both tar and pitch - skin and scrotal cancer from direct contact, and lung cancer from inhalation of the fumes. A fourth type, cancer of the bladder, has also been observed in excess in makers of coal gas, which may perhaps have been related to the presence of 2-naphthylamine in the fumes; but the concentrations observed were small and other chemicals may have been responsible (Doll et al., 1972). It is unwise, however, to extrapolate directly from one industry to another as the conditions of exposure vary, as does the mix of polycyclic aromatic compounds (PACs) to which the men have been exposed.

Some 100 PACs have been identified in the atmosphere of aluminium production plants by Bjorseth and Eklund (1979), 34 of which were present in sufficient amounts for Bjorseth et al. (1978) to measure. Twenty of these have been reviewed by the International Agency for Research on Cancer (1983) in its monographs evaluating the carcinogenic risk of chemicals to Man. None of the individual PACs could be

specifically incriminated as human carcinogens, as human exposure has always been to a mix of many. For 4 of them, however, the Agency concluded that there was sufficient evidence of carcinogenicity in laboratory animals, while for 3 others there was limited evidence of carcinogenicity: that is, according to the Agency's definition, evidence that was limited either (i) because the studies involved a single species, strain, or experiment, or (ii) because the experiments were restricted by inadequate dosage levels, duration of exposure, or various similar technical defects, or (iii) because the neoplasms that were produced often occur spontaneously in the same species and are difficult to classify by histological criteria alone, such as adenoma and adenocarcinoma of the lung, or tumours of the liver in certain strains of mice. For 13 of the remaining compounds the evidence was considered to be inadequate to form a judgment, and only 3 were definitely classified as not carcinogenic at all.

In the absence of adequate data on humans, it is reasonable for practical purposes, according to the International Agency, "to regard chemicals for which there is sufficient evidence of carcinogenicity in animals as if they presented a carcinogenic risk to humans." Yet even for these chemicals it is now clear that they may act by different mechanisms and that extrapolation from one species to another is not always appropriate. They must, however, be regarded as prime suspects and the amounts of these compounds that Bjorseth et al. (1978) were able to measure in one Soderberg plant with vertical pins and one prebake

plant are shown in Table 2, along with the amounts of these compounds for which limited evidence of carcinogenicity has been obtained. All these compounds, it may be noted, are also found in cigarette smoke. Figures are given for the amounts measured in particulate form, as only very small additional amounts of any of them, apart from carbozole, were also present in the gaseous phase. Total PACs in particulate form varied from about 20 to 200μgm^{-3} in the Soderberg plant with an average figure around 100μgm^{-3} and were approximately two orders of magnitude less in the prebake plant. Personal sampling provided very similar figures, but showed that individuals employed as pinpullers in Soderberg plants might be exposed to average concentrations over 2 hour periods that were as much as 10 to 15 times greater. Such concentrations are not very different from those that have been reported during the manufacture of coal gas (Lawther et al, 1965), and although they are certainly not typical of the primary aluminium industry they strengthen the suspicion that men employed in aluminium production plants might be exposed to the same hazards of cancer as men employed in the manufacture of the former.

EPIDEMIOLOGICAL OBSERVATIONS

In the light of these findings, it should not be surprising if men employed in the aluminium production industry were found to experience an increased mortality from cancers of the skin and lung, and possibly also of the bladder. They might conceivably also be at risk of developing mesotheliomas, but there is no reason to suppose

that they should develop an increased number of cancers in any other site. Any excess of the first 3 types of cancer should, moreover, be observed characteristically for workers in Soderberg plants rather than for workers in prebake plants.

So far as cancers of the skin are concerned, these should not cause any material mortality if detected early. In industries in which they have occurred as a result of specific occupational hazards, they have commonly been noted in the company's medical records. If a suspiciously large number of cases have occurred on unusual sites (e.g. the forearms or scrotum), they have not been reported publicly since Konstantinov and Kuzminykh's (1971) first paper, and I shall leave aside any further consideration of skin cancers except in so far as they appear in mortality statistics.

The other potential occupational cancers in the industry have a high fatality rate and are best sought for in mortality data. It is useless looking for them in population surveys since the prevalence of cancers with a high fatality rate at any given moment is inevitably low, and the absence of even one affected man in a group of less than 1,000 employees can give a false sense of security, as happened in the British chromate producing industry in the 1950s.

By far the most useful technique to detect an occupational hazard of a potentially fatal cancer is the cohort study in which all men who have been employed in the industry for as long as good personnel records exist are followed forwards in time to discover how many have died

from the specified causes of death. The observed mortality rates are then compared with those that would have been expected from the experience of some standard unexposed population of the same age distribution observed over the same period. Exceptionally, as in Norway, incidence rates can be substituted for mortality rates with the gain of an increased number of cases and hence a better opportunity of observing a difference that is not confused by the play of chance, but this is possible only when the recording of incidence in the industrial and comparison cohorts can be guaranteed to be unbiased by a special concern for the industrial cohort. It is possible in Norway, where you have a system of cancer registration that covers the whole country with approximately equal efficiency, but it is seldom possible elsewhere, and it is only in extreme situations, in which the hazard is grossly increased, that comparisons of incidence data are of any material value in Britain and the USA.

The mortality (or incidence) data need to be broken down to show separately rates for men who have been employed on jobs that are likely to have different degrees of exposure and for men who have been employed for different lengths of time and for different periods after first exposure (since different cancers have characteristic induction periods which differ from one type to another, but are seldom less than 5 and often more than 20 years).

Such desiderata sound simple, but they are not easy to achieve in practice for a variety of reasons, including the

difficulty of choosing suitable standard rates for comparison and in classifying the extent to which individuals have been exposed in the past, and the paucity of numbers in the most crucial subgroups. Moreover, it is now becoming clear that we have insufficient theoretical background to know whether we ought to examine trends in the mortality ratio (that is, the ratio between the observed and expected numbers of deaths) or in the excess mortality rate (that is, the difference between the observed and expected rates). For our present purpose I shall look primarily at mortality ratios, as it is these that have been used routinely in the past.

Three cohort studies comprise the great majority of the epidemiological material that is of any substantial value in determining the reality and extent of any specific cancer hazards in the primary aluminium industry: those reported by Andersen et al. (1982) for Norway, by Gibbs and his colleagues (Gibbs and Horowitz, 1979; Gibbs, 1982; 1984) for Quebec, and by Rockette and Arena (1983) for the USA.

Canadian experience

The Canadian study involved 5,406 men who were working at one or other of two aluminium plants on 1 January 1950 (cohort I), and 485 who were working at a third plant one year later (cohort II), all of which were situated in Quebec. The men were followed until the end of 1977 (Gibbs, 1984) and interim results were published providing the observations that had been made before the end of 1973 (Gibbs and Horowitz, 1979; Gibbs, 1983). All but 0.5% were successfully traced, and 1,631 were found to have died.

Death certificates were obtained for 93% of the men who died in the larger cohort (I) and for 87% of those who died in the smaller cohort (II), and the causes of death were classified according to the certificated cause. It follows, therefore, that the specified causes have been underestimated by a small but material amount, and it would be reasonable to multiply the cause specific rates by 1.075 for cohort I and by 1.079 for the combined results of both cohorts.

No separation was made between process and non-process workers, but men were classified on the basis of their occupations according to whether they had no exposure to tar, some exposure to tar, or definite exposure to tar, and comparisons were made between the mortality rates in men who had ever or never been exposed and, within the ever exposed, between those who had had different numbers of years' exposure. For this purpose a year's employment with some exposure was taken as equivalent to a quarter of a year's 'definite exposure'. Understandably, perhaps, but still unfortunately, employment on the prebake process was classed as involving no exposure at all. Men were reclassified at the beginning of each year according to the amount of exposure they had had, and the expected numbers of deaths in each category were obtained by multiplying the man-years at risk in each exposure category by the corresponding sex and 5 year age group mortality rates for the province of Quebec for the same calendar years.

The results are summarized in Table 3 for all causes and for the four separate categories of cause for which data

were given for both cohorts combined, according to whether the men were ever or 'never' exposed to tar. The total mortality and that from non-malignant diseases was slightly less than expected which can reasonably be attributed to the healthy worker effect. The excess mortality from lung cancer in all men and in men whose work had exposed them to tar was in both cases statistically highly significant ($P<0.001$). More importantly, there was a progressive increase in the standardized mortality ratio with the amount of tar exposure measured in weighted tar-years and the excess after 21 or more tar-years of exposure was also highly significant ($P<0.001$). This is shown in Table 4. Other causes of death and other cancers (other than cancers of the lung and bladder) showed little change with number of tar-years exposed, and the results are most easily explained by a carcinogenic hazard from exposure to tar.

The small excess mortality from lung cancer which had been observed in men 'never exposed' to tar when the follow up was continued only to the end of 1973 (Gibbs and Horowitz, 1979) and which had raised the possibility of a small hazard in prebake plants (classified as having involved no exposure to tar) largely disappeared when the follow up was continued to the end of 1977, and what little did remain may be attributed either to chance or to the fact that the towns in which the men lived had a slightly higher mortality from lung cancer than the province of Quebec as a whole (Gibbs and Horowitz, 1979). Nevertheless, it would certainly have been preferable to have classified work on the prebake process separately and to have shown data for

men who had worked only on this process, if it had been practicable to do so.

Lastly, the figures that have been reported for other types of cancers in men at two out of the three plants followed to the end of 1977 (Gibbs, 1984) are summarized in Table 5. Cancers of the lung and bladder are shown separately, as there was a prior hypothesis that they might be observed in excess, and others are shown separately if there was an excess over expected in the combined group of ever and never exposed men. Other specified types of cancer for which the overall incidence was less than expected (i.e. cancers of the pancreas, genital organs, brain, and leukaemia) have been combined in one group.

The notable finding in this table is the excess from cancer of the bladder in the ever exposed. The number of deaths is small and the excess is not statistically significant (P=0.08, one-tailed), but it is more important to note that half the deaths were concentrated in men who had been exposed for more than 20 tar-years (Table 4) and that the trend in the SMR with duration of exposure was highly significant. These observations are, moreover, complemented by the results of a case-control study that has been carried out in the area of the plants by other and independent investigators. Thériault et al. (1981) noted that the mapping of cancer incidence in Canada by county revealed a part of Quebec where the rate was exceptionally high (Wigle, 1977), and successfully interviewed 81 out of the 96 residents in the area who were diagnosed as having bladder cancer between 1970 and 1979. Occupational histories

revealed that 25 had worked in the electrolysis sections of aluminium plants compared with only 14 out of 81 matched controls. From these and other data obtained in the study, Thériault et al. (1981) estimated that the relative risk of developing bladder cancer in aluminium electrolysis workers who did not smoke was 1.9, but that cigarette smoking and occupational exposure acted synergistically so that the relative risk for the combined exposures was 5.7.

Norwegian experience

The results of the Norwegian experience are of a high standard scientifically, but are less easy to interpret. Data were collected for all 8 plants that were able to provide complete personnel records for the entire period of their operation; but, because of the long induction period that is common before cancers are produced, only those obtained from the 4 plants in operation before 1954 have been utilized. Some of the main characteristics of the study are summarized in Tables 6 and 7. Unfortunately employment in Soderberg and prebake plants cannot be properly separated, as three of the plants used both processes and only one (Eydehamn which used the prebake process) used only one.

From Table 7 it is evident that the overall mortality was slightly less than the average for the country as a whole, but very close to that recorded for the counties in which the plants were located. This, Andersen and his colleagues (1982) suggest, could be due to a healthy worker effect, which is common for an employed population, being

counterbalanced by the employees' relatively low socio-economic status. Their idea could indeed be true; but most of the time that the men were under observation must have been many years after first employment, when the healthy worker effect tends to have passed off, and the results may just imply that the death rate was similar to that of other men living in the same areas, which was somewhat less than that for the country as a whole.

One minor defect of these data is that no attempt appears to have been made to ensure that the men who were not found to have died were still in Norway and alive. If some men had emigrated, which seems not altogether unlikely, some may have died abroad and the observed number of deaths may have been slightly underestimated.

Table 8 shows the numbers of cancers registered among men employed in the plants compared with the numbers that would have been expected at the corresponding county rates divided by type. Only cancers of the skin other than melanoma are excluded, because they are not recorded at the Registry. The data for the two old first war plants have been combined and so have the data for the two post second war plants. As with the Canadian data, types of cancer are shown separately if there was a prior hypothesis that they might be recorded in excess (cancers of the lung and bladder) and if there was an excess over expected in all four plants combined. Other specified types of cancer for which the overall incidence was less than expected, i.e. cancers of the stomach, colon and rectum, pancreas,

prostate, and lymphatic system, have been combined in one group.

Further analysis was limited to the 57 cases of cancer of the lung, as the numbers of the other individual types of cancer were thought to be too few for subdivision. The results are given in Table 9. This shows that the excess mortality was more marked in the processing departments than elsewhere, and was more marked in the old plants than in the new. When, however, the results were examined for men with different durations of employment, the high standardized incidence ratio in the processing department in the old plants was found to be much the same irrespective of the duration of employment. In the new plants a high standardized incidence ratio was observed mainly in men who had been employed for only a few years, and Andersen and his colleagues (1982) suggested that this excess in short-term workers might be due to the inclusion among them of an unusually high proportion of cigarette smokers. This may be so, but it is notable that a consistently high standardized mortality ratio, irrespective of duration of employment, was recently observed in a large group of English asbestos workers who were studied by Acheson et al. (1984), and that the American beryllium workers who were studied by Mancuso (1979) also showed an excess of lung cancer that was limited to the short-term employees.

A further difficulty in interpreting the Norwegian data is the gross difference in the incidence of lung cancer in the different parts of the country during the period when the aluminium workers were studied. The results are

therefore crucially dependent on the choice of the standard group for calculating the expected number of cases. This is illustrated in Table 10, which shows the different standardized incidence ratios that are obtained from the old and new plants if the rates chosen for comparison are respectively those recorded for the county in which each plant is located, the country as a whole, and Oslo city. Since some of the workers in the plants were recruited from parts of the country other than that in which the plant was located (just what proportion is unknown) it might be thought that their smoking habits, and hence their risk of lung cancer, might be more closely similar to the national rates than to those of the local county. Certainly the Oslo rates would be inappropriate, but the gross differences that are introduced by using different standard rates for comparison warn of the possibility that the results may be confounded by non-occupational factors.

In sum, the Norwegian data do not, by themselves, provide conclusive evidence of an occupational hazard, but the concentration of the excess mortality from lung cancer in men employed in the processing departments of the two old established plants plus the fact that the only types of cancer to show a significant excess in the old plants were the two which, it had been predicted, were most likely to be produced by occupational hazards in the industry, does strongly suggest that occupational hazards of cancer have existed in these plants. Unfortunately data are not given separately for men first employed before and after the second world war, so it is impossible to say whether the

hazards have been reduced. It was noted, however, that the excess of bladder cancer in the old plant was "mainly seen among those who started employment during the period 1916-29", and it may be that the hazard has already been reduced.

One further difficulty, which must be noted, is that no figures are given for the separate mortality rates from lung and bladder cancer for workers who were employed in the one plant which used only the prebake process (Eydehamn) and for workers who were employed in the other old plant (Tyssedal), which used both processes. It would be difficult to comment on such a breakdown, however, without detailed knowledge of incidence rates by duration and time of employment in each plant and such figures would be unreliable because of the small numbers involved.

U.S. experience

The last of the three major studies is the massive study of nearly 22,000 men employed in one or other of 14 aluminium reduction plants in the U.S.A. (Rockette and Arena, 1981 and 1983). All the men had five or more years cumulative employment in one or more of the plants between 01.01.49 and 31.12.77 and were followed from 01.01.50 (when the first men were qualified to enter the cohort) to the end of the period. All but 1.2% were successfully traced and 3,414 were found to have died. Death certificates were obtained for 97.6% of those who had died and these were used to classify the causes of death. Since no cause was discovered for 2.4% of deaths, the calculated SMRs for the

whole population were multiplied by 1.024 and by corresponding fractions for sub-populations whenever this was practicable.

The main results are summarized in Table 11, which shows the standardized mortality ratios for all causes of death and for four causes or groups of causes that were obtained by comparing the observed deaths with the numbers that would have been expected if the men had suffered the same risk of death as men of the same ages in the same calendar periods in the country as a whole. Ratios are given separately for men employed in the 7 prebake plants, the 6 Soderberg plants, and all 14 plants, including the one in which both processes had been used. With the single exception of lung cancer in the prebake plants, for which the standardized mortality ratio was 100, the ratios all lie between 76 and 97. That they should nearly all be less than 100 is not altogether surprising as men who were required to have been employed for five years before inclusion in the cohort will certainly have been healthier than the general sum of the population; but it is mildly surprising that the SMR for all cancers other than lung cancer should have been as low as 83 for all the plants combined, as the so-called 'healthy worker effect' does not generally have much effect on the cancer mortality rate after 5 years have passed, and manual workers might be expected to have a slightly raised mortality from cancer. One explanation for the deficiency could have been that the comparison with national mortality rates was inappropriate and that comparisons ought to have been made, as in Norway and Canada, with rates for the areas

in which the plants were located. This, however, was certainly not the explanation as the use of local county rates nearly always resulted in lowering the SMRs rather than raising them. The authors, correctly I think, thought it unwise to extrapolate county populations for more than a few years after the 1970 census and they limited their calculation of the numbers of deaths expected from the relevant county rates to the period 1950 to 1973, for which they were able to rely on populations estimated from the 1950, 1960, and 1970 censuses. The results showed that for all cancers the SMR was reduced from 85 to 80 and for lung cancer from 94 to 86, and to much the same extent or more for 7 of the other 11 types of cancer that they examined separately.

Further examination of the mortality from types of cancer other than cancer of the lung showed that there was a slightly raised mortality from cancer of the bladder in the Soderberg plants and from 5 other types of cancer when all types of plant were considered together (Table 12). That there should be an excess mortality from some types of cancer by chance is only to be expected, and none of the excesses were so great as to make this explanation unlikely in the absence of any prior hypothesis about them. It is notable, however, that cancer of the kidney was also found in excess in Norway and Quebec, while leukaemia was also in excess in Norway - and for neither of these types of cancer was the excess completely eliminated by using local county rates for comparison instead of national rates (SMR for the period 1950 to 1973 reduced from 113 to 107 for renal

cancer, and from 125 to 113 for leukaemia). The excess from cancer of the pancreas was, however, eliminated altogether (SMR reduced from 116 to 94).

Table 13 shows the principal results when cancer of the lung and the two types of cancer for which there was the greatest overall excess in comparison with the national rates (cancer of the pancreas and leukaemia) are examined in relation to duration of employment in all jobs and in Soderberg potrooms. Only cancer of the pancreas in Soderberg potroom workers showed any increase with increasing duration of exposure, but the number of deaths from this disease was very small and even the excess after 15 years' employment, in which 7 out of the 8 cases were concentrated, was not statistically significant (P=0.07, two-tailed test).

Taken at their face value the American data imply that in the industry as a whole there has been no measurable excess of any of the types of cancer that might have been anticipated to occur in it from prior knowledge of the types of pollution produced, and it does not provide any compelling evidence of the existence of any other unsuspected cancer hazard. We are left, however, with the uncomfortable feeling that, despite the very great efforts that were made to ensure that the whole population at risk was identified and followed up, there may have been some differential loss of records of men who had died in the early years. This is suggested by the very low death rates that were observed during the first 10 years of follow up. These are shown by the standardized mortality ratios for all causes of death

and for five individual causes or groups of causes separately for prebake and Soderberg plants in Table 14. The healthy worker effect may well vary quantitatively in different countries, but it is unusual for it to have as great an effect for as long as 10 years, as is suggested by these data, particularly when it is borne in mind that the SMRs for cancer were certainly overestimated by using national rates for comparison and are likely to have been for other causes as well.

Other data

The other studies of cancer in the aluminium reduction industry add little of value to those to which I have already referred. Konstantinov et al.'s (1974) report from the USSR, where the environmental levels of benzo(a)pyrene in the factories ranged from 1.1 to 1,695 ugm^{-3} indicated elevated mortality ratios for lung and skin cancer, but unfortunately did not give any detailed information about the population under study, and Milham's (1976 and 1979) work in Washington State is superceded by Rockette and Arena's (1983) study which included nearly all the workers who had contributed to his observations. Giovanazzi and D'Andrea's (1981) report provides a little additional data from Italy; but the number of workers involved was very small (212 potroom workers, 40 of whom died, and 282 workers in other departments of whom 13 died). Compared with national mortality rates there was a significant excess of deaths from cancer of all types in the potroom workers (14 against 8.0 expected) and a non-significant excess of lung cancer (4 against 2.3 expected).

CONCLUSION

What then can we conclude? First, there can be no reasonable doubt that there were occupational hazards of lung and bladder cancer in the Soderberg plants in the Quebec industry and, in the light of this finding it would be unreasonable not to conclude that at least some of the excess mortality from these two types of cancer in the old plants in Norway was also occupational in origin. Excess mortality rates from both these types of cancer can be confounded by smoking habits; but the use of local rates for comparison, which should partly allow for the effects of smoking, the concentration of the excess on those most heavily exposed to the specific industrial process, and Thériault et al.'s (1981) case-control study of bladder cancer patients, which enabled smoking habits to be taken into account, make it difficult to attribute the results to differences in smoking habits alone.

Secondly, it is not possible on epidemiological grounds, to exculpate the prebake plants entirely. Neither the Canadian nor the Norwegian study provides data for men who have been employed only in prebake plants and both present results that would be compatible with the existence of a hazard in them.

Thirdly, the U.S. data provide some assurance that it is possible to operate the industry without causing a detectable cancer hazard. It may be, too, that the same holds for the new plants in Norway, but in both cases there are findings that throw doubt on the conclusion that it has yet been achieved. In the U.S. the very low mortality rate

recorded in all the plants in 1950-59 could be due to a combination of the healthy worker effect and chance, but the conclusion that no detectable hazard did, in fact exist, would be greatly strengthened if normal rates continue to be observed in the population that has already been identified in the years after 1977. Moreover, it must be noted that there was an increased mortality from bladder cancer in the plants using the Soderberg process (SMR of 162) even though it was based on only 8 deaths. In Norway the standardized incidence ratio for lung cancer in men who had been employed for less than 15 years in the processing departments of the new plants is disturbingly high (220 based on 9 cases), and the number of cases in long-term employees (5) is too few to place any reliance on the fact that their incidence ratio is not raised. Only time will tell which of these two contrary indications is the more reliable.

Fourthly, the only other type of cancer which the data suggest might result from an occupational hazard is cancer of the kidney (Table 15). This occurred in excess in all three studies and may indicate a hazard in the prebake plants, where the cases were concentrated in the U.S. series and which were classed as not causing exposure to tar in Quebec. If there is such a hazard it cannot easily be identified with the hazard of bladder cancer, despite the close anatomical and physiological relationship of the two organs, as one appears to be related to prebake plants and the other to Soderberg plants. Of the three other types of cancer that at one time or another have been suggested might be related to exposure in the industry, two were in excess

in two series (leukaemia in the U.S.A. and Norway and cancer of the larynx in Norway and Quebec) and one only in one (cancer of the pancreas in the U.S.A.). The last can now be confidently dismissed, despite the increasing SMR with duration of employment in the potrooms in the American data, as it is not confirmed elsewhere and the apparent excess in the U.S. was eliminated when comparison was made with local rather than with national rates. There was no evidence to relate leukaemia specifically to heavy exposure in any particular set of conditions, but its future incidence should certainly be kept under review, and the same applies to cancer of the larynx, which it is natural to think might be associated with a hazard of cancer of the lung, although it is not, in fact, often found to be so in practice.

Fifthly, these hazards (cancers of the lung and bladder, possibly cancer of the kidney, and conceivably cancer of the larynx and leukaemia) must be presumed to be related to the volatile chemicals associated with exposure to coal-tar pitch. In our present state of knowledge, there is no way in which we can logically predict what effect a given amount of a mix of polycyclic aromatic compound will have, partly because the various sources all give rise to different mixes and partly because active carcinogens may exist in other forms. Moreover, if the hint of a hazard of renal cancer now provided by the experience of the industry means anything, it is unlikely that a single measurement of one index PAC or of all PACs combined will provide a realistic measure of the cancer hazard, as different hazards would seem to be associated with the different environments

of the different processes. We shall be better able to judge what the real situation is when we have the results of the second follow-up of the Norwegian industry's workers (Andersen et al., 1984) and much better able to if the U.S. industry reviews its experience again in a year or two's time, when it will have a few more years' experience and be able to use the 1980 census to calculate local county rates for the period around the census, and when there should be no possibility of a deficiency in the number of observed deaths.

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Table 1

CHEMICAL EXPOSURE IN ALUMINIUM REDUCTION OPERATIONS

Importance		
Primary	Secondary	Tertiary
Aluminium metal dust	Ammonia	Beryllium
Aluminium oxide	Copper dust or fumes	Beryllium oxide
Asbestos	Magnesium oxide fumes	Cadmium dust or fumes
Carbon monoxide	Ozone	Lead
Chlorine	Silica	Manganese dust or fume
Coal tar pitch	Welding fumes	Mercury
Fluoride dust		Phosgene
Hydrogen chloride		
Hydrogen fluoride		
Nuisance dusts*		
Oil mist (mineral)		
Sulphur dioxide		

*Including coke and glass and mineral wool fibre.

Table 2

AMOUNTS OF POLYCYCLIC AROMATIC COMPOUNDS IN THE ATMOSPHERE IN PARTS OF TWO ALUMINIUM PRODUCTION PLANTS

(after Bjorseth et al, 1978)

Evidence of carcinogenicity	Compound	µg Particulate matter m^{-3}	
		Vertical Pin Soderberg plant (10 samples)	Prebake plant (6 samples)
Sufficient	Benzo(a)anthracene	1.0-15.0	0.05-0.26
	Benzo(b)fluoranthene Benzo(k)fluoranthene	1.1-26.9	ND-0.28
	Benzo(a)pyrene	0.7- 9.0	ND-0.05
Limited	Carbazole*	ND- 2.8	-
	Chrysene	2.1-30.1	0.07-0.36
	Anthanthrene	ND- 0.32	-

*Also present in some gaseous phases samples in amounts up to 1.4 µg m^{-3} in the Soderberg plant and up to 0.20 µg m^{-3} in the Prebake plant.

Table 3

QUEBEC ALUMINIUM WORKERS: STANDARDISED MORTALITY RATIO BY CAUSE OF DEATH AND EXPOSURE TO TAR (all three plants, after Gibbs, 1984)

Cause of death	Exposure to tar		All men	
	Never	Ever		
Lung cancer	107 (33)*	144 (110)	133	(143)
Other cancer	79 (66)	113 (213)	102	(279)
Circulatory disease	80 (201)	91 (515)	88	(716)
Other causes	86 (140)	95 (353)	93	(493)
All causes	84 (440)	99 (1191)	94	(1631)

*Numbers of deaths in parentheses.

Table 4

QUEBEC ALUMINIUM WORKERS

STANDARDISED MORTALITY RATIO BY AMOUNT OF EXPOSURE IN TAR-YEARS:.

ALL CAUSES AND LUNG CANCER

(numbers of deaths in parentheses)

No. of plants	Cause of death	Exposure in tar-years			
		0	<10	11-20	21 or more
3+	Lung cancer	128 (27)*	91 (29)	170 (20)	286 (19)
	Other causes	71 (278)	92 (528)	84 (161)	94 (72)
2†	Lung cancer	101 (30)	97 (42)	172 (27)	271 (32)
	Bladder cancer	28 (1)	61 (3)	188 (3)	667 (6)
	Other cancers	85 (63)	115 (128)	96 (35)	132 (28)

*Numbers of deaths in parentheses.

+Followed to 31.12.73 (Gibbs and Horowitz, 1979).

†Followed to 31.12.77 (Gibbs, 1984)

Table 5

QUEBEC ALUMINIUM WORKERS

STANDARDISED MORTALITY RATIO FOR DIFFERENT CANCERS

AND EXPOSURE TO TAR

(two plants, after Gibbs, 1984)

Type of cancer	Exposure to tar		All men
	Never	Ever	
Lung	101 (30)*	143 (101)	131 (131)
Bladder	28 (1)	161 (12)	117 (13)
Hodgkin's disease	173 (2)	179 (5)	175 (7)
Kidney	185 (5)	129 (8)	146 (13)
Oesophagus and stomach	111 (17)	153 (50)	139 (67)
Larynx	84 (2)	131 (7)	117 (9)
Colon and rectum	84 (13)	110 (37)	101 (50)
Other specified† types	54 (11)	86 (37)	76 (48)
Other types	67 (13)	105 (47)	93 (60)
All	85 (94)	123 (304)	112 (398)

*Numbers of deaths in parentheses.

†Cancers of pancreas, genital organs, brain, and leukaemia.

Table 6

NORWEGIAN ALUMINIUM INDUSTRY'S CANCER INCIDENCE STUDY

Plant	Start of production	No. of men employed at least 18 months before Feb. 1970, and alive at 01.01.53	Follow-up period
Tyssedal	c.1915	1147	01.01.53 to 31.12.79
Eydehamn[1]	c.1915	1119	
Sunndal	c.1950	1488	
Aardal	c.1950	3656	
All		7410	

[1]Prebake system only: other plants used both Soderberg and prebake systems.

Table 7

NORWEGIAN ALUMINIUM INDUSTRY'S CANCER INCIDENCE STUDY

Plant	No.of deaths observed	Ratio of deaths observed to expected	
		1	2
Tyssedal	338	1.06	'nearly 1.1'
Eydehamn	435	0.97	
Sunndal	141	0.80	'nearly 0.9'
Aardal	457	0.83	
All	1371	0.92	

[1]Using corresponding national calendar year and 5 year age specific rates.

[2]After adjustment for county of location.

Table 8

NORWEGIAN ALUMINIUM INDUSTRY

INCIDENCE OF CANCER BY TYPE AND BY AGE OF PLANT

(numbers of cases in parentheses)

Type of cancer	Standardized incidence ratio		
	Old plants	New plants	All plants
Lung	172 (33)+	144 (24)	159 (57)+
Bladder	168 (18)+	72 (8)	119 (26)
Leukaemia	153 (9)	119 (8)	135 (17)
Kidney	129 (8)	116 (10)	122 (18)
Larynx	91 (2)	139 (5)	121 (7)
Unspecified type	105 (43)	114 (61)	110 (104)
Other specified* type	92 (106)	83 (93)	88 (199)
All	109 (219)	99 (209)	104 (428)

+P<.05 (one-tailed test).

*Cancers of stomach, colon and rectum, pancreas, prostate, and lymphatic system.

Table 9

NORWEGIAN ALUMINIUM INDUSTRY: INCIDENCE OF LUNG CANCER BY TYPE OF WORK AND DURATION OF EMPLOYMENT

(numbers of cases in parentheses)

Department	Length of employment (yrs)	Standardized incidence ratio		
		Old plants	New plants	All plants
Processing	1½-4	200 (9)+	294 (5)+	226 (14)+
	5-14	175 (7)	167 (4)	172 (11)+
	15 or more	208 (11)+	100 (5)	155 (16)
	All	196 (27)+	154 (14)	179 (41)+
Other	1½-4	77 (1)	176 (3)	133 (4)
	5-14	91 (1)	100 (2)	97 (3)
	15 or more	133 (4)	128 (5)	130 (9)
	All	111 (6)	132 (10)	123 (16)

+P<.05 (one-tailed test).

Table 10

NORWEGIAN ALUMINIUM INDUSTRY

STANDARDISED INCIDENCE RATIO FOR LUNG CANCER

USING DIFFERENT SETS OF RATES FOR COMPARISON

Plants	No. of lung cancers	Rates for comparison		
		Local counties	Whole country	Oslo city
Old	33	172	147	72
New	24	144	77	42
All	57	159	107	55

Table 11

U.S. ALUMINIUM INDUSTRY: STANDARDISED MORTALITY RATIO BY CAUSE OF DEATH AND TYPE OF PLANT

(numbers of deaths in parentheses)

Cause of death	Type of plant		All plants*
	Prebake	Soderberg	
Lung cancer	100 (161)	88 (64)	96 (272)
Other cancer	85 (315)	84 (128)	83 (524)
Major cardiovascular disease	97 (1352)	76 (416)	87 (2093)
Other causes	77 (605)	79 (315)	76 (1065)
All	91 (2433)	82 (923)	84 (3954)

*Including one plant employing both processes.

Table 12

US ALUMINIUM INDUSTRY

STANDARDISED MORTALITY RATIO FOR DIFFERENT CANCERS

BY TYPE OF PLANT

(numbers of deaths in parentheses)

Type of cancer	Type of plant		All plants*
	Prebake	Soderberg	
Lung	100 (161)	88 (64)	96 (272)
Bladder	73 (11)	162 (8)	78 (19)
Leukaemia	128 (25)	131 (11)	128 (43)
Pancreas	133 (39)	106 (13)	125 (63)
Kidney	151 (19)	55 (3)	120 (26)
Lymphosarcoma and reticulosarcoma	132 (15)	117 (6)	112 (22)
Stomach	113 (35)	106 (13)	105 (55)
Unspecified	64 (98)	78 (54)	67 (178)
Other specified+	80 (73)	60 (20)	77 (118)
All	92 (476)	86 (192)	89 (796)

*Including one plant employing both processes.

+Cancers of larynx, colon, and genital organs.

Table 13

US ALUMINIUM INDUSTRY

STANDARDISED MORTALITY RATIO BY JOB

AND YEARS OF EXPOSURE: SELECTED CANCERS

(numbers of deaths in parentheses)

Type of cancer	Job	Years of exposure		
		< 15	15-20	20+
Lung cancer	Soderberg potroom	81 (23)	115 (12)	81 (7)
	All jobs	104 (94)	87 (60)	91 (118)
Pancreas cancer	Soderberg potroom	20 (1)	234 (4)	224 (3)
	All jobs	120 (20)	142 (17)	113 (26)
Leukaemia	Soderberg potroom	247 (9)	- (0)	
	All jobs	136 (18)	118 (25)	

Table 14

US ALUMINIUM INDUSTRY: STANDARDISED MORTALITY RATIO*
BY TYPE OF PLANT AND CALENDAR PERIOD
(numbers of deaths in parentheses)

Cause of death	Prebake plants		Soderberg plant	
	1950-59	1970-77	1950-59	1970-77
Lung cancer	90 (16)	90 (81)	0 (0)	96 (46)
Other cancer	80. (45)	83 (147)	85 (10)	84 (73)
Ischaemic heart disease	88 (129)	105 (475)	37 (10)	78 (164)
Accidents and violence	66 (37)	97 (87)	70 (13)	88 (59)
Other causes	40 (67)	86 (378)	42 (18)	92 (201)
All causes	66 (294)	93 (1168)	49 (51)	86 (543)

*Omitting 2.4% of deaths of unknown cause.

Table 15

CANCER OF THE KIDNEY IN ALUMINIUM REDUCTION WORKERS

Country	Standardised mortality ratio+		
	All plants	Highest rate	
Canada (Quebec)	146 (13)*	185 (5)	in 'never exposed' but including work in prebake plant
Norway	122 (18)	129 (8)	in old plants
USA	120 (26)	151 (19)	in prebake plants

+Incidence ratio for Norwegian data.

*Number of cases or deaths in parentheses.

Chairman

Thank you very much Professor Doll for the extensive overview and appraisal of the risk of cancer in the primary aluminium industry. You made particular reference to the following:

i. First you described the risk potential in the primary aluminium industry associated with exposure to PAH compounds from the use of pitch and resulting from the partial combustion or destillation of coal and oil.

ii. Second you indicated similar groups of substances in other industries than the Aluminium industry which may cause hazards of cancer of the skin, lung and bladder but that it is not known which of the specific PAH compounds are responsible for producing such cancers in humans.

iii. Third you implied that risks of developing these cancers attributed to occupation in the industry must be considered to occur until shown not to.

iv. Fourth you drew attention to some studies suggesting a risk of cancer at other sites than the lung and that such sites should be borne in mind also in the Aluminium industry.

The audience will no doubt like to participate in a wide discussion of these important aspects which I suggest be done after the two subsecuent papers of this morning's session have been presented. In the meantime, however, brief comments are invited also after each of the individual papers. Are there any such comments now after Professor Doll's paper?

Langård: I wonder if Professor Doll would comment on the large American study which he said included 14 different plants and a large number of people. These and other experiences in the USA (petrochemistry, chromates a.o.) have a tendency to lower the risk sometimes observed in smaller European studies.

Doll: For the research workers responsible for an overall study it becomes increasingly difficult to ensure the maintenance of an equally high standard throughout when you increase the number of plants involved. That is not a criticism of the research workers. It is just a fact of life. There is sometimes the

possibility too that some plants may be operating in different ways. The degrees of pollution may be substantially different. Plants with important hazards associated with heavy exposure may be "diluted" by the inclusion of plants with very restricted exposure. It becomes extremely difficult to maintain standards of collection of data over so many plants. But this can be overcome by continuing the study, because the research done on these workers can be followed through in subsequent years. It then becomes very much more like a single study in a single plant. I hope very much that the tripartite study in the States is continued for a further 5 years so that the information collected becomes scientifically easier to rely on. One would, however, still be left with the fact that hazards and conditions vary from plant to plant. In the full report of the American tripartite study to the government and unions, detailed data were published for each plant separately. But I would like to return the question. What is your feeling about such studies?

Norseth: I seem to recall that there was an excess of kidney cancers at Bremanger which is a ferro-vanadium plant in Norway. It was based on quite a few cases. At the Institute of Occupational Health we tried unsuccessfully to find some common explanatory pattern for the cases. I would like to ask Elkem to look into that again.

Guthe: Norseth may also recall a PAH Symposium we attended in USA some years ago when an excess of kidney cancers was suggested in a study of American Steel Industry workers (coke oven workers) by Redmond & Lloyd, I believe.

Langård: Further to my previous remark about the American petrochemical studies where one or two plants were included in the first instance, an increased risk of about 1.5 to 1.6 was found. Subsequently 9 or 10 large plants were included. The risk was thereby diluted and no hazard was considered to be present. I don't think that is a right conclusion. It is certainly possible that the exposure in the particular plants was much higher than in other plants.

Mowé: One comment on methodology: A main problem in all

epidemiological studies of occupational cancer is evaluation of the exposure. You showed the very complex exposures in the aluminium industry. Would it be better to try to undertake case reference studies in this industry in the future e.g. with regard to bladder cancers? Would it not be possible to take the 18 bladder cancers and 3 or 4 controls and go into details concerning exposure, look at combination and interaction of exposures etc. An additional comment directed to the plant physicians in the aluminium industry concerning bladder cancer, is that we should recognise bladder cancer in old plants as occupational cancers. Such cases should be notified to the Directorate of Labour Inspection.

Doll: When a cohort study has suggested a specific hazard of a particular type of cancer it is an excellent thing to complement that by a case reference study of individual subjects to see if the exposures that caused the hazard can be specified. But this does not always give as clear an answer as one might hope. If one is dealing with risks that are double or less than double the normal risks, then there will be a 50-60% dilution of cases that in fact haven't had any special hazards but will have had all sorts of experience in the industry. On small numbers it isn't always all that clear just what the specific exposures have been. Nevertheless I think one should always try case reference studies of individual patients. One may well get indications for what to concentrate on in future studies.

I would like to add one point to the interesting question of the reliability of looking at data for an entire industry. I think one has to distinguish between situations in which you already have a prior hypothesis that there might be a hazard, and situations in which a hypothesis is thrown up by one particular plant. The risk of cancer of the prostate in cadmium workers was thrown up by one plant and perfectly properly reported. Concern was felt by everyone that there might be a hazard on the basis of the experience of 4 cancers, with 0.8 expected. Then another plant in America reported the same thing. There was absolutely no experimental reason why this should be so and no prior hypothesis why there should be a hazard. In such a situation one must look at the whole industry to get a large enough population. There are now results reported for the whole

cadmium industry in Britain covering 21 plants. I have also seen results for the American industry. The total number of prostate cancer deaths in the British industry is now 31, and on national rates we would have expected 30.9. One had to treat the first findings as hypothesis forming and then look for confirmation elsewhere. This does not detract from the validity of Dr. Langård's remarks in other situations where there is good reason to think that there may have been a hazard in one plant.

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7. PANEL DEBATE

Special Panel Guest: Sir Richard Doll

Panel Members:
K. Berg
A. Bjørseth
B. E. Dahlberg
S. Langård
T. Norseth
H. H. Tjønn
J. R. Vale

Chairman: T. Guthe

Chairman: Welcome to this panel debate. It is hoped that our special panel guest, Prof. Doll, the panel experts and the symposium participants in the hall will all feel free to take part in the debate. Dr. Dahlberg has kindly agreed to open the debate with some introductory remarks.

4.1. Introductory Remarks

Dahlberg: Prof. Doll, members of the panel, ladies and gentlemen, I invite you all to place yourselves in the position of an Occupational Health Doctor in a Norwegian Aluminium plant. Among the many problems he/she has to face are PAH (or PAC). The plant doctor is supposed to evaluate the health risks and give advice and information to workers and management. If the plant doctor does not have the required competence and knowledge, he/she must obtain the necessary information elsewhere. In fact this is what we are doing at this symposium both yesterday and today. When facing employees and management, radio and newspaper reporters, the plant physician is the one to represent the expertise. We are by now used to such situations and I believe that this is how it has to be. It is part of the plant physician's work, and a challenging task. We ask your professional guidance and knowledge in handling this difficult question.

Since the first reports on the excess occurence of Lung Cancer among potroom workers appeared, the plant physicians have been working hard to get to the heart of the problem. The interpretation of published data has been most difficult. It has been suggested that the causing agent is PAH. But do

we know that for certain? Is it the whole truth? What are the combined effects? We have been awaiting results of further investigations, pushing the answers ahead of us. The Norwegian Authorities have nevertheless decided on a maximal exposure level - or an administrative norm of 40 microgram PAH pr m^3 air to be allowed in the working environment of the aluminium plants. There are, however, the very wide limits in the precision of the analytical methods used as previously mentioned during this symposium. It is understandable in the circumstances that the union asks for a report in plain understandable terms on PAH, exposure levels and health risks. I ask the panel's assistance in this and to put PAH in the wider context of all other pollutants in in the working atmosphere of the aluminium plant. On the one hand the plant physicians are sceptical of belittling the possible health hazards of PAH in view of the focussed interest on its association with Cancer. On the other hand we have been careful to draw attention to the other known serious health hazards in the potrooms, i.e. lung diseases other than cancer, e.g. potroom asthma.

The industry pays great attention to the Nordic Aluminium Industries' Health Committee's recommendations. We therefore have the responsibility to give the best advice possible. But giving advice concerning the working atmosphere, leads to discussions of the processes of production and technology. Indeed, what we think, mean and do in this respect today will be reflected in the potrooms 10-20 years from now. Will we at that point be able to say that we did our best? We ask you to help us define PAH as a health hazard generally and in the primary aluminium industry, and in conjunction with other pollutants in the potroom atmosphere. This would make practical formulations possible to serve the purposes mentioned. I will now try to formulate some questions requiring answers, even if some have already been discussed before.

(i) First Is there an excess of cancer in potroom workers in the primary Aluminium industry?

(ii) Second If so, what is the extent of the problem and which are the cancer sites?

(iii) Third If in the affirmative,

a. what is (are) the causative agent(s)?

b. what is the role of PAH compounds in carcinogenesis?

c. how will one or more of the PAH compounds affect pregnant and/or fertile women, keeping in mind the increasing number of female potroom workers?

d. is it possible that also gametic cells can be damaged?

e. what is the effect of exposure to PAH compounds and asbestos together, keeping in mind the relationship between smoking and asbestos?

(iv) Fourth Do we have enough epidemiological, medical and industrial hygiene knowledge to make recommendations to management of a preferred type of smelter?

(v) Fifth What should be the plant physician's attitude to the Norwegian maximal "administrative norm" for PAH of 40 $\mu g/m^3$ work air which is often exceeded in the plants, and taking into account the incidental character of this level, the limited reliability and precision of sampling and PAH analytical methods.

(vi) Sixth How should the plant doctors' attitude be to the possible mutagenic activity and ecological meaning of reported PAH levels in the atmosphere of areas and villages adjacent to aluminium production plants, when asked by community doctors and politicians?

Chairman: Dr. Dahlberg has in his introductory remarks to the panel debate raised a number of questions. Taking up the question of the administrative norm of 40 $\mu g/m^3$ and PAH, analytical methods and the wideness of their limitations reference was made to the papers and our discussion of this topic yesterday. To open the debate I wonder if Fjeldstad would discuss that further in the context of the question raised by Dr. Dahlberg?

Fjeldstad: Yesterday I described the administrative norm and how the present value of 40µg/m^3 was established. It was based on the percentage PAH in the tar on the filter. This percentage can vary from 10-40%, which is quite a wide range. With 10% you will get half of this norm value, or about 20 µg/m^3. At 40% you have it doubled. Dr. Tjønn and others might possibly amplify on this question.

Norseth: As to the administrative norm of 40 µg/m^3 air it should be made clear that there is absolutely no available data allowing any kind of risk estimation related to this or any other level of exposure to PAH in any industry. The value of 40 µg is guess work. When discussing this value we must ask why we have it. One answer is to force industry to develop technology which will reduce exposure. As such 40 µg/m^3 is a fairly reasonable level since industry has not yet reached that value. When it does I would suggest that we lower it to 20, maybe 10. The value is thus not at all related to epidemiological research although I think the presence of a cancer risk in the aluminium industry is reasonably well shown to exist. We do not know if there exists a cancer risk for people starting to work in the aluminium industry today. But we know that they will be exposed to carcinogenic substances. There is possibly a very small risk, so small that it may be impossible to detect it in an epidemiological study which really is a very coarse method. If you are exposed to carcinogenic substances a cancer risk must be assumed to be present. For this reason the Aluminium industry should be forced to improve its production technology.

Dahlberg: I agree that we want to see the smelter technology improved. On the other hand attention has been focussed so much on PAH compounds that the special technological solutions sought have cost hundreds of millions of kroner. Companies have built and rebuilt the Søderberg pots to Sumitomo technology mainly to get rid of PAH compounds in the environment. Following such investments the company will stick with that technology maybe for 10 years or longer. I would prefer technology allowing improvement of the total working environment and also implementing our needs with regard to all other environmental factors and not only PAH compounds on which so much interest has been focussed during the past few years.

Tjønn: Concerning the 40 µg/m^3 as an administrative norm for PAH it is fair to say that this figure is drawn out of a hat. But we should also bear in mind that it is very difficult to measure the PAH concentration in a potroom. Moreover, - as we have heard - the various plant laboratories carrying out the measurements come up with results greatly different from those of the reference laboratories. I agree with Dr. Norseth that we should encourage improvements to the working environment in the aluminium plants; there is room for it. The administrative norm is not a limit below which we are safe or above which we are unsafe. It is a value of convenience to encourage improvement of the total environment in the aluminium industry. I am not concerned about the large sums of money invested by Norwegian industry in order to improve the environment. One main point is what to do with Søderberg anodes. In my view they should perhaps be dropped in the deep sea! A new technology is needed.

Langård: We should not spend too much time on the administrative norms. It was confirmed this morning by Prof. Doll that there is no way to predict the cancer hazard related to the levels of PAH exposure today. Therefore the figure of 40 µg/m^3 probably is - as has been said - "drawn from a hat". But when governmental institutions put forward such values they should also consider the cost of such requirements as compared with other improvements obtainable with the same amount of money. Norseth's comment that administrative norms are instruments to force the industry to improve technology can only be considered as political limit value for environmental pollutants. In that case we have left the basis we have had for threshold limit values up until now.

Kristoffersen: Hvis vi skulle diskutere 40 µg/m^3 PAH bør det gjøres i et annet forum dvs med folk fra fagbevegelsen, Yrkeshygienisk institutt, Arbeidstilsynet og arbeidsgiverene. Det er sagt ting her som jeg føler jeg må gå imot. Det ble sagt at det ble brukt flere hundre millioner kroner for å få vekk PAH. Det er ikke riktig. Industrien i Norge gikk inn for Sumitomo for å klare flere ting. Først, ville de øke levealderen på elektrolysecellene fra 2-3 år til 6 år. Regn ut og se hvor mye penger de ville tjene på det. Videre

ville elektrisitetsforbruket bli nedsatt betraktelig. Jeg var med fra begynnelsen for tillitsmennene i diskusjonen om Sumitomo. Vi lå vel på 17-17.5 kwh pr. tonn aluminium. Jeg tror japanerene sa at vi skulle komme ned i 14-14.5 kwh. Det har imidlertid ikke slått til. Vi gikk over til Sumitomo pga at arbeidstilsynet satte så harde normer. Investeringene ble ikke 200 millioner men kanskje 400-500. Det er deres egen feil idet Sumitomo i innkjøp kostet 90 millioner kroner. Resten er hevet ut av vinduet fordi det ble tullet med å få teknologien i gang. Sumitomo teknologien gir tørre anodetopper. Det minker PAH og det blir helt andre forhold i elektrolysehallene bl.a. fordi en får kniver istedenfor å kjøre rundt med en hakkespett med 110 dB støynivå. Hele arbeidsmiljøet kan altså tas vare på med denne teknologien. Jeg synes at før en kommer med uttalelser bør en sette seg inn i hva det er gjort på arbeidsplassene. Forholdene er så pass gode idag at vi ligger under de administrative normene så og si både for PAH og for støv.

Skogland: As representative of Norsk Hydro's Karmøy Fabrikker I should like to make some comments. The epidemiological studies reported at this meeting have not taken the exposure level into account. We have even seen that the workers in prebaked plants, and transport workers in Søderberg plants are registered as non-exposed persons. As a comment to Kristoffersen's remarks, I have here the results of the measurement of the exposure levels at Karmøy plant. You will see from the figure that the group "others" - which mainly consists of transport workers at the Søderberg plant - has almost the same exposure as the cell operators. If the epidemiological studies put transport workers into groups of non-exposed workers - then the results of the epidemiological studies are unreliable. In the aluminium industry a lot of time is spent measuring the exposure level and great efforts have been made to reduce it. What is the point of measuring exposure levels and reducing the levels if the exposure levels are not taken into account in the epidemiological studies? What is then the purpose of an administrative norm for exposure (40 $\mu g/m^3$)? And if the exposure level has so little importance, does this mean that we can now reduce the number of measurements when the exposure level gets below 40 $\mu g/m^3$?

Doll: I would like to make two comments.
(i) The first is in relation to Dr. Norseth's statement that no data allows any risk estimate to be related to specific values of PAH's. I strongly agree with him, but would like to link that with the last speaker's remarks. The fact that epidemiological studies have not made much use of environmental measurements is because they have not been available for use. It is the ambition of every epidemiologist to be able to relate his observations of disease incidence in groups of men and women to their environmental exposure and to their behaviour measured in quantitative ways. So every epidemiologist supports the last speaker in saying how important it is to collect such measurements and then to use them to interpret the observations we can make on men and women.

(ii) I would then like to go back to Haugen's paper yesterday. I think it is unwise to rely on some environmental measure as a means of controlling hazards, without taking into account that for the pollution from that chemical in the atmosphere to have any effect on the body it has to be absorbed into the body and metabolised and brought to the site of action. I would urge that whilst using, for the time being, these environmental measures of PAH as a means of controlling or attempting to control the hazards and for monitoring the extent of pollution in the working environment we should recognise that these are only guesses at what is the correct thing to do. We should be monitoring the biological exposure of the actual workers the whole time. Because it may well be that people are exposed to large amounts of chemicals which - if you treat them in the appropriate way - will cause enormous amounts of cancer in animal species and yet will not cause a single cancer in the human because the chemicals are absorbed in a different way or are not biologically available. On the other hand there may be chemicals which have not been demonstrated to have any effect which in the particular circumstances in which humans are exposed and with the specific metabolic processes of man are in fact causes of disease. I think therefore it is most important that whilst we continue to operate the threshold limit values, we should always bear in mind that these are just regulations which are the best that can be recommended at any given moment. We should continue to keep the work force of any industry under observation and if we suspect that it is being exposed to any unusual hazard or disease we should make biological measurements because it is only by controlling the

actual metabolised chemical that we can really be sure that we are controlling the risk to the individual.

Bjørseth: Dr. Tjønn mentioned that we should drop the Søderberg anodes "in the deep sea". As an environmental chemist I am of course against all kinds of marine dumping! Addressing myself to Prof. Doll's comment, I would like to show a table listing all the techniques discussed yesterday. We talked about chemical analysis, short-term testing, animal experiments and epidemiology. On the left hand side I have suggested how easy it is to perform the analyses on these measurements. In the second row the relevance of the data are given. For the chemical analyses it is quite easy to do the measurements, but there is still a question mark about the relevance of the data. The reason is that we are concerned with the atmospheric concentration of compounds that are inhaled. We can say nothing about the body dose of these compounds and definitely nothing about the target dose. I desist from saying anything about how we should rate the short-term animal experiments and the epidemiology findings since I am not a professional in those areas. But I maintain that there are question marks concerning the relevance of the chemical data. We should also keep that in mind when we evaluate the data from the aluminium plants.

Reisæter: I am a doctor at Husnes aluminium plant which is a prebake smelter. I have some remarks to the debate and questions to the panel concerning occupational, environmental carcinogenesis and the presence of substances in the working domains. In later years warning of cancer risks has been proclaimed by various medical research institutions. What implications do the different cancer risk factors have for humans in general and for the individual person? A one-sided approach against cancer can be misleading or wrong. Generally, diseases might take the form of bone marrow or brain damage, lung tissue destruction or serious allergic reactions etc. The combined effects of chemical substances must be noted. Over 2,000 chemical substances are suspected to be carcinogenic for experimental animals. In comparison one has reliable information that only about 30-50 of these are carcinogenic

to humans. Knowledge of cancer risk factors for humans comes from costly bought experiences in exposed groups and/or single outstanding observations by skilled medical doctors. So the limitations of epidemiological methods and doubts about the reliability of research observations justify conclusions which tend to create fear among employees? It is not only a medical problem in the larger perspective it is also an ethical, political and legal one. What is demanded from us is an open and honest presentation of our incomplete knowledge, inviting political organs together with the employers, employees and their organisations to establish guide lines and limit values of the potential importance of the situation in preventive health care.

Norseth: I have a comment to the last speaker. The forming of a committee with all parties involved to decide recommendable exposure levels is exactly what is done. As a comment also to Langård, all administrative norms are political and have always been so. In this country the administrative norms are established by discussion between labour organisations and the employers, i.e. a political decision. The medical background is often very weak. As a comment to Dahlberg, I agree with him that we should be careful not to concentrate on one factor and ignore other hazards. As to Bjørseth, he overlooked an important factor by not mentioning biological marker monitoring. Maybe they are the most important ones at present. Unfortunately we do not yet know the importance for the single worker of a biological marker like DNA protein adduct or genotoxic substances in urine or in sputum or wherever you look for it. What is really needed is a systematic monitoring of biological markers combined with proper epidemiological screening so that hopefully we will know what biological markers really mean. We would then have a good way of estimating health risks.

Drabløs: We industrial doctors have to take measures because of suspicions that some environmental component may cause carcinoma and we cannot wait for proof. We have heard that there is an increased risk of leukemia. Can anyone suggest which environmental components are causative for leukemia?

Langård: I would like to comment on the question of interaction raised by Dahlberg, Reisæter and Norseth (yesterday). Experiments have shown that the clearance of PAH from the airways of animals is reduced by a combination of dust and PAH exposure. A combination of aerosol and PAH probably makes exposure to PAH more extensive. The human situation is similar (Cohan in "Science" some 3/4 years ago) and we have indication that a combination of smoking and aerosol results in more airways retention of the aerosol among smokers than among non-smokers. This could indicate that the same exposure actually gives a higher or stronger exposure in smokers than among non-smokers, since the retention of the particular particle is much longer in smokers than in non-smokers. This might in part explain the recognized interaction between asbestos exposure and smoking. At least one of the "other factors" in the Norwegian plants refered to by Dahlberg may be asbestos. The excess risk observed in the Norwegian study is about 2/1 for lung cancer. This is not very great as compared with the asbestos/ tobacco situation. It could be that just the amount of asbestos used in the aluminium industry could explain some of this increase. I assume that a lot of potroom workers have been exposed to asbestos during a long period of time. Smoking is probably more prevalent among these populations and interaction between asbestos and tobacco could also explain part of the excess. In combined exposure groups Italian colleagues have in a case referent study attempted to determine how great the cause fraction is, or - what is the attributable risk referable to asbestos and tobacco respectively and how much is referable to the combination of the two. In that particular study asbestos as a causative fraction contributed only 6% and tobacco with about 40%. This phenomenon could explain a part of the excess in the Norwegian aluminium study if asbestos had been present.

Sanner: The possible cancer risk in the primary aluminium industry was evaluated by the International Agency for Research on Cancer (IARC) late last year. As a participant in that meeting I would just like to cite the conclusion with regard to epidemiological studies. The evaluation was: "The available epidemiological studies provide limited evidence that certain exposures in the aluminium production industry are cancerogenic to humans, giving rise to cancer of the lung and bladder. A

possible causative agent is pitch fume. There is inadequate evidence that occupational exposure in the aluminium production industry results in hæmatolymphopoietic and pancreatic cancer".

Chairman: One of Dr. Dahlberg's questions was that if there is a cancer excess, which are the sites? Can it be answered more precisely than the discussion has brought out so far?

Doll: I think we are all in agreement with the International Agency for Research on Cancer's conclusion that there is evidence to make us accept that there is a hazard of lung cancer and bladder cancer in certain parts of the primary aluminium industry. But we haven't yet faced up to the question of the reality of the existence of the other hazards that have been mentioned. When a large number of studies are done of any group of workers and a large number of cancers are looked at, inevitably there will be a substantial proportion in which the standardised mortality ratio (SMR) is above 100. What tests are we now going to apply in order to accept these excesses as being occupational in origin rather than the chance findings that must occur regularly in such circumstances? Obviously if one can relate them to specific chemicals which are known to produce that type of cancer in other circumstances one will be inclined to accept the existence of a hazard in the industry and one would also be inclined to accept it, if you could show a relationship with length of exposure and with a specific period after exposure. Now, to my mind none of the other cancers that have been mentioned really pass any of these tests, though I wouldn't be prepared as yet to dismiss them altogether. I think we can dismiss the possibility of a hazard of pancreatic cancer because that has not been shown anywhere outside the USA where it was first suggested. Milhams suggestion should not be regarded as independent of the study carried out by Rockette and Lorraine because they both covered the same set of data. Added to this the latest studies show that when the incidence of pancreatic cancer is compared with the incidence in the local counties rather than with the national figures the excess of that disease disappears. So far as I am concerned there is no evidence to concern us about pancreatic cancer. Concerning larynx cancer the average intelligent person would say that

if you accepted a risk of lung cancer there should be a risk of laryngeal cancer. In practice this does not apply epidemiologically though possibly because laryngeal cancer is so much more uncommon and one does not obtain enough data to test the hypothesis. But I would at any rate keep larynx cancer with a question mark against it, although I don't believe there is sufficient positive evidence to say that it is produced. But on common sense grounds, one has to consider the possibility of it being produced if the chemicals going past it produce cancer in the bronchial mucosa. Though of course they may go past the larynx without actually depositing on it. Leukemia is I think extremely difficult. I am not aware of any chemicals in the PAH group which have been shown to cause leukemia in other situations though there is now some suggestion that cigarette smoking may produce leukemia. If this is confirmed, the possibility that the aluminium industry is also producing leukemia must be considered seriously. One of the problems with leukemia is that we do not know what tests to apply epidemiologically about the relationship we would expect between the date of first employment (or first exposure) and the development of the disease. Certainly we cannot expect it to be the same as for the epitelial cancers. We cannot say that we should look for an excess 15 or 20 years after first exposure, when we know that the leukemias that are produced in humans by chemical exposure - or by ionizing radiations occur with maximum incidence within 5 years of first exposure. To my mind at the present moment we should keep an open mind about leukemia.. We should not dismiss it, but neither should we accept it as an occupational hazard. I will add to that cancer of the kidney which I spoke about this morning. I think Dr. Guthe's comment on the existence of an excess among coke oven workers in the steel industry is an interesting point. But if there is a risk the evidence points to it being associated with the pre-bake process. This reminds us once again that we don't really know what are the specific agents responsible for the carcinogenic hazards.

Guthe: In the aluminium industry the question of possible health effects of magnetic fields has come up and leukemia has been mentioned. Would Dr. Tjønn care to say something about that?

Tjønn: Magnetic fields is an interesting area and some health effects have been described. The subject will be discussed in a forthcoming seminar in Sweden. In connection with leukemia benzene should be mentioned, since I believe it can be destilled from pitch and mineral coke of the anodes.

Norseth: I would like to put a question to the panel. If we did not find by epidemiological investigations an increased cancer risk but still know that there is an exposure to carcinogenic substances, will the question if there was an epidemiological risk really change our attitude? Something had to be done to improve the working atmosphere even if the results of the epidemiological results were so called negative. In my view epidemiological results are not negative. There are both positive and inconclusive epidemiological results since you always have to figure out what is the lowest possible risk which could be detected. If you figure this out I feel that the highest acceptable risk is still lower than the lowest that can be detected by the epidemiological investigation. Then a positive epidemiological investigation should not change our attitude to the problem. This is an ethical problem.

Doll: The debate which Dr. Norseth has initiated is one in which I should like to join if you could set aside two days for discussion! I would however like to say a few words in response, relating to the meaning of words. It is easy to say that some agent is a carcinogen and translate that in our minds to meaning it causes cancer in the individual. But a "carcinogen" means that in certain circumstances somebody has been able to produce cancer with it in an animal by for example injecting it or doing something else to it that succeeds in producing cancer. But first of all we have to know something of this chemical's bio-availability. It may be completely immaterial that it exists in the atmosphere, as it may not reach the target tissue or it may not be absorbed. We cannot just say that it is a carcinogen and therefore it must be controlled. We want to know if there is something which humans are absorbing in such a way that it will produce a risk to man. Very often we have to assume that it will do so because we want to act on the safe side.

Langård: Commenting on the same aspect there is a type of economic priority in the industry and in the community. If we know we have certain carcinogens in the work places should we invest a lot of money to reduce or eliminate these instead of using the same amount of money in areas where the effect would be more efficient for preventive purposes?

Chairman: Another question was raised by Dr. Dahlberg in this context of the total environment related to conditions in focus in the aluminium plants in addition to cancer. Looking first to the lungs, I wonder if Prof. Vale would like to say something about lung conditions other than cancer. You have discussed these at previous health conferences concerned with the Aluminium industry.

Vale: As a chest clinician I know little about the various PAH compounds. I think however, that the question we have been discussing today, namely the carcinogens in the potroom atmosphere is actually a rather small fraction of the health problem in the plants of the primary aluminium industry. I would like to remind everyone of the other kinds of respiratory disorders which occur: obstructive diseases, asthmalike or chronic airflow obstruction. These are much more frequent than lung cancer and undoubtedly have a far greater impact on the health of aluminium workers. I agree with those on and outside the panel who have stressed the danger of focussing too much on PAH compounds, leading to technological "improvements" which may actually deteriorate the atmosphere with regard to so-called inert dusts and other pollutants. I consider it important to make these points here and now even if they appear to be a little outside the topic of this symposium.

Dahlberg: Even if it can be considered to be a little on the side of this symposium it is important that the questions of PAH is put in a proper context in the total environment. As

plant physicians we should go back and discuss it in such a context. It would also be interesting to know which role PAH compounds have as agent(s) or element(s) in the mechanisms behind the asthma reaction and the chronic diseases. We do not have any answer to this at the moment, and research is needed in this area. Such research work should not be secondary to cancer research.

Astrup: Dr. Dahlberg put forward the question if PAH compounds destroy or harm gametic cells. As many know it has been recommended to remove pregnant women from the potrooms of aluminium plants. In this connection I would like to ask Prof. Berg if we should allow fertile women to work in the potrooms at all? If they continue to work there, would he recommend studies recording the frequency of births, spontaneous abortions, malformations etc.

Berg: Yesterday I quoted some circumstantial, indirect data from experiments with mice in which relevant compounds had been given to pregnant mice. This resulted in tumours in adult life after intrauterine exposure. Also, in an experimental animal series (Tomatis) an increased frequency of tumours was found in later generations. Taken at face value this should mean that genetic transmissible changes predisposing to cancer had been created by the intra-uterine exposure. It seems to point to gametic cell damage in the animals so exposed. Also, a reduced number of premordial oocytes was found in prinatally exposed animals. So it seems that the exposure could cause significant damage to the gametic cells. Having said that I must stress again how indirect and circumstantial this evidence is. I have no idea of how the doses employed would relate to the amount of exposure in the Aluminium industry. If we believe that exposure in the industry has caused cancers, it would seem reasonable to assume that DNA damage has occured. You would then have an obligation to suspect that DNA damage could also take place in gametic and not only in somatic cells. But again, man is a complicated animal. To my mind more important than what you have been discussing most of the day, is the question if PAH and several other compounds in the industry do in fact cause transmissible genetic damage. I referred to this yesterday. It is difficult

to examine this question. The kind of studies one should do, would involve very extensive experiments. It is easy to say that they would be too expensive.
Concerning pregnant women in Aluminium plant working environments, anybody with good sense would not want a pregnant woman in such an environment as germinal tissue might be quite vulnerable at the foetal stage. There is also the broad question about teratogenicity of these compounds. In many ways we know far too little to draw any practical conclusions concerning fertile women. But mutations can also take place in fertile men. The very long term approach to this would be to try to establish - perhaps through collaboration with other countries - systems to uncover if there exist mutant proteins which I alluded to yesterday; and by utilising some of the other new techniques which are now coming up, notably the new possibilities of examining DNA itself.

Astrup: I wonder if I could reformulate my question. As it has been recommended that pregnant women should not be allowed to work in the potrooms, do you think that they should be removed earlier?

Berg: I meant to say yes to that and my main reason was the Tomatis data from animals.

Chairman: If there are no more comments on this aspect, I suggest we move on to one of Dr. Dahlberg's further questions. It concerned production plant communities in which there were local doctors and politicians asking questions about pollution of the external environment and if this pollution could have an ecological effect. We had a paper on that subject, yesterday. We also have in the audience representatives of the SFT.

Jæger: SFT has only begun to consider the question and have as yet no answers. Clearly we want to know what the levels of PAH compounds in the surroundings of Aluminium plants are. We also hope to get help from the experts as up to now we are somewhat bewildered by what these PAH levels mean.

Thrane: Avoiding repetition of what was said yesterday, I want nevertheless to draw attention to the total PAH burden in the environment. There were high atmospheric concentrations of PAH in the area. I also pointed to the burden of the PAH in the dust settling on the ground. We found quite an amount of PAH in such dust. It could get into the soil and also cover the vegetation in the area. By this pathway it could enter into the ecological system.

Doll: Can anyone tell me what the comparable measurements of PAHs are in the Finish fir forests in strong sunlight? I have been unable to trace the reference, but I have been told that a recent study in Finland has shown a very substantial increase in PAH in the forests following clear sunny days.

Chairman: Since nobody seems to have information on this point I suggest that we move on to one of Dr. Dahlberg's further questions, namely if we have enough knowledge of technology to make recommendations today for the type of smelters which are going to serve us into the 1990's. Can Dr. Dahlberg first amplify on his question?

Dahlberg: The reason for this question is that we are often asked by the management, if we know enough and if we can give recommendations. We have already had a long debate about the 40 $\mu g/m^3$ administrative norms for PAH, but in formulating this question my hunch was that Norwegian engineers have competence to build smelters that are very good and which can give us the very fine working atmosphere we all want. We are all aware of the fact that improvements cost money and that the money should be invested in the right place so the effect on the working environment can be seen. Are we doing this? If we are, we must realize that we are also pointing to some of the ways to develop future smelters. Possibly a management representative present may wish to comment on this?

Chairman: Perhaps Mr. Ellingsæter who is also a member of the governing council of the Aluminium Industry's Secretariat for Health, Environment and Safety would comment?

Ellingsæter: I have been listening to the presentation of the papers and discussions for two days and have so far refrained from making any comments. However I would like to ask Prof. Doll to excuse my commenting in Norwegian for the benefit of those who have difficulties in following the discussion in English.

Dahlberg har i det siste spørsmål tatt opp hvilke råd han skal gi til ledelsen og bedt om hjelp til å gi slike råd. Noen unnviker å svare, andre gir svar som er av liten hjelp selv om man har lyst til å dumpe Søderberg anodene i havet. Faktum er imidlertid at Søderberg ovnene står der og det tar tid å dumpe dem i havet.

Jeg har på mange måter sympati for bedriftslegene. De er i en vanskelig stilling. På den ene side må de justere seg til det praktisk liv og på den annen side skal de være profesjonelt ærlige.

Vi er nødt til å finne en løsning og jeg viser til Langårds relevante innlegg: at vi er nødt til å være praktiske og fornuftige og å se hvordan vi bør bruke investeringsmidlene på best mulig måte.

Vi i industriledelsen og som også er medlemmer av styret for Aluminiumindustriens Miljøsekretariat er nødt til å trekke praktiske konklusjoner. Vi kan ikke i det uendelige diskutere nye prøver. Verkene står der.

Den konklusjon jeg trekker er at det ligger i luften at det er en overhyppighet av kreft ved Aluminiumverkene. Overhyppigheten synes ikke så stor at det er noen umiddelbar grunn til omgående å slå alarm. Det arbeid som er gjort har vært verdifullt idet det har skaffet oss ny kunnskap. Usikkertheten som er kommet til uttrykk på dette seminaret er også en nyttig erfaring.

Det er dessuten en konklusjon at vi bør overvåke situasjonen fremover i tiden.

En annen konklusjon er at det fra Aluminiumindustriens siden og fra Miljøsekretariats side må sees praktisk på om vi skal rette fokus på miljø og helse noe bort fra kreftsykdommen og i retning også av andre helseplager i industrien slik som bl.a. også Prof. Vale understreket.

Jeg har ikke nevnt noe særlig om nye annlegg som ville forbedre ihvertfall arbeidsatmosfæren. Installasjonene f.eks. på Karmøy viser at det fysiske arbeidsmiljøet er blitt vesentlig bedre sammenlignet med driften av de gamle Søderberg ovnene. Det er imidlertid praktiske og økonomiske grenser. Vi kan bekalge at de er der, men en kan ikke komme utenom.

Kristoffersen: Dahlberg's siste spørsmål angår ikke bare ledelsen på bedriften. Fag-bevegelsen stiller det hver dag. Mange innenfor fagbevegelsen har gjerne den mening at hvis de arbeidene som er satt i gang på aluminiumsverkene i tilknytning til rammekravene blir fullført så kan vi benytte Søderberg ovnene et stykke inn i 1990 årene. Jeg har i så måte vært optimist i lengre tid. Hvis all parter på bedriften, fagbevegelsen og bedriftsledelsen, jobber sammen så klarer de å få til bra forhold på arbeidsplassen. Men ut i nittiårene er Søderberg ovnene blitt så gamle at de må skiftes ut, uansett. Det er da bare en måte å skifte dem ut på, nemlig med prebake annlegg. Pr. dags dato er vi ikke så glad i prebake annlegg. Det er kanskje litt rart å si. I prebake hallen på Karmøy er luften sånn som den er herinne, dvs helt til man åpner dekslene, da kommer røk og støv ut, men det er ikke så mye. De som gjør de verste operasjonene får en god del støv eksponering mens de øvrige får svært lite. Når det skal legges om må det iallefall gjøres litt etter litt. Pr. dags dato er det ca. 6000-7000 mann ansatt i aluminiumindustrien i landet. Når en går over til prebake generelt så blir det ca. 2500, ihvertfall ikke mer.

Tjønn: En uttalelse jeg har kommet med er blitt sitert noen ganger, nemlig å dumpe Søderberg anodene i havet. Den er blitt protestert av generelle miljømessige grunner idet havet ikke skal forurenses. La meg si det på en annen måte: i hallene hvor det elektrolyseres aluminiumoksyd bør en ikke varme opp petrol koks og bek og jeg tror ikke at roterende kniver og endret Søderberg teknologi vil forbedre forholdene særlig f.eks. for boltetrekkere selv om man har plikt til å ha verneutstyr. La meg til slutt si at det har vært interessant å være til stede på dette møte som har

holdt en høy vitenskapelig standard. Vi har fått bedreftet oppfatninger som har foreligget tidligere. For Arbeidstilsynet er dette verdifullt i den period vi nå går inn i, nemlig det siste år for oppfyllelse av nåværende rammekrav for Aluminiumindustrien som går ut pr 1.1.85. Når det gjelder kreft situasjonen føler jeg meg forsåvidt beroliget. Kreftrisikoen er vel til stede og det er blitt reist interessante spørsmål om andre former for kreft enn lungekreft. Jeg håper det blir anledning til å følge disse opp. Det som er uttalt om den administrativ norm på 40 $\mu g/m^3$ bekymerer meg, nemlig spørsmål om 41 er galt og svært risikabelt og om 38/39 er riktig og absolutt sikkert. Dette er selvfølgelig ikke en realistisk måte å stille spørsmål på, særlig tatt i betraktning den store analyseusikkerthet som råder og hvor vi så ringeprøver som avdekket at PAH konsentrasjonen i samme prøve kunne variere med en faktor på 3-4 og muligens opp i 8-9 i enkelte prøver. Det er klart at analyse verdien må taes med forbehold for prøvetakning og analysemetoder, og brukes som veiledning, retningslinjer. Norske aluminiumsverk kan neppe komme ned til lik standard uten å gå over til en annen teknologi, dvs prebaked. Vi i arbeidstilsynet takker for invitasjonen fra Aluminiumindustriens Miljøsekretariat til å komme her. Jeg håper at vi får anledning til å følge arbeidet videre og at vi fortsatt vil få informasjon og også kanskje bli invitert neste gang!

Skogland: Jeg vil gjerne gå tilbake til det Bjørseth stilte et stort spørsmålstegn ved, nemlig hvorvidt det var relevant å gjøre målinger og analyser på PAH. Det er ikke å komme forbi at det er vanskelig å ta representative prøver, og det er vanskelig å gjøre analyserer med en tilstrekkelig grad av nøyaktighet, slik som det har viste seg i ringtestene som ble presentert igår. Figuren jeg viste angikk PAH eksponeringen for årene 1981-82-83 i Søderberg annlegget på Karmøy fabrikker.

Rutineundersøkelsene ble foretatt vår og høst. Hver undersøkelse omfattet 2 skift på tilsammen 80 operatører hvorav halvparten ble undersøkt m.h.p. PAH-eksponering. Målingene er helt i tråd med driftssituasjonen vi hadde på Karmøy fabrikker. Ved den først rutineundersøkelse av stort omfang høsten, 1981, var driftssituasjonen relativt god, slik som det fremgår av verdiene for cellepasserne og transport-operatørene. Boltetrekkerene var mindre å skryte av. Dette forundret oss fordi Karmøy fabrikker var en av de første som fikk innebyggete boltetrekkerkraner. Vi satte igang med å finne en forklaring. Parallelt med dette fikk vi imidlertid store driftsforstyrrelser. Det var strømbrudd på Karmøy 24. november 1982. Alle cellene "frøs" til. De ble started opp igjen ved årsskiftet 1981/82. Vi anså det nødvendig å gjøre en ekstraordinær arbeidsmiljøundersøkelse i januar 1982 for å se hvordan eksponeringen da var hos operatørerene. Hos boltetrekkerene var den skyhøy. Dette er operatører som sitter i innelukkete førerkabiner og utfører automatisk boltetrekkingen ved å betjene spaker inne i kabinen. Etterhvert som vi fikk skikk på driftssituasjon, stabiliserte også miljøsituasjonen seg for celleoperatørene, og for de som jobbet på gulvet i elektrolysehallene. Det er min og andres erfaring at en stabil god driftssituasjon gjenspeiler seg i arbeidsmiljøet. Hva angår boltetrekkere har vi hatt et prosjekt i gang for å rense lufttilførselen til boltetrekkerkabinen. Vi valgte å tilføre luft, ikke bare filtrert for støv, men også med innlagt filter med aktivt kull for å adsorbere de gassformige PAHene, eventuelt også PAH som måtte strippe av det som avsettes på filterne. Vi opererer med overtrykk inni førerkabinen. Det punktet som viser en eksponering på 75 $\mu g/m^3$ for boltetrekkerene er en gjennomsnitts verdi tatt i høstundersøkelsen 1983 og representerer innebygde boltetrekkerkraner uten den nevnte forbedring som ikke ennå var kommet i gang. En kran ble ombygget på prøve. Jeg har desverre ikke tabellen her for de ca. 15 kranmålingene som ble gjort. Gjennomsnittelig lå eksponeringen på 10 $\mu g/m^3$ for boltetrekkerene over den tiden de oppholdt seg i kabinen. Det dreier seg ikke om veiet gjennomsnitt over 8 timer. Vi måler kun den tiden de oppholder

seg i elektrolysehallen. De forsøkene vi har gjort med denne boltetrekkerkranen er lovende. Om vi kan komme ned i en PAH eksponering av boltetrekkerene på 10 ug/m^3 så tilsvarer dette samme lave PAH-eksponering i prebake annlegget. Jeg vil gjerne konkludere med at måling og analyse av PAH eksponeringsnivået ikke er bortkastet. Det har tvertimot hatt betydning.

Ellingsæter: I begynnelsen av panel-debatten ble jeg noe bekymret. Det var atskillig engasjement angående 40 ug/m^3. Det er vel egentlig i industrien et tilbakelagt stadium å diskutere denne på praktisk grunnlag. Det er en administrativ norm som må aksepteres og vanskelighetene som oppstår ved å oppfylle normen må vi greie å takle mellom industrien, fagorganisasjonen og arbeidstilsynet.

Jeg er imidlertid forbløffet over Norseth's kommentar at når vi klarer 40 ug/m^3 så ønsker han krav om lavere normer, 20, 10, osv. For å si det svakt: dette er en meget robust politisk uttalelse som ikke er noe særlig praktisk informasjon for bedriftslegene å gå hjem til sjefene i aluminiumindustrien med.

Chairman: We will now turn to Dr. Dahlberg for his comments after the panel and others have endeavoured to answer the questions he raised in his introduction.

Dahlberg: Thank you all for the answers and comments given. Possibly Prof. Doll would be patient and round off this debate by summarising briefly the present state of knowledge concerning the excess of Lung Cancer. Everybody who has spoken on this issue appears to admit that there is an excess. We have also discussed other sites of cancer. What is the extent of the problem? Different rates are given in different studies. The union is also asking for a number. What is the extent of the risk of cancer in the primary aluminium industry?

Doll: We have been discussing the industry as a whole which includes the Canadian industry and the American industry as well as plants in this country. The majority of workers in the industry in this country are, I believe, working in plants

for which we have had no data presented since they are plants which have been instituted after 1954. I understood the question to be what I estimate the current increased risk of Lung Cancer to be for a man working in the potrooms of the aluminium industry in Norway at the present moment. My answer to that must be that we have had no evidence presented which would allow us to give a precise figure. But as conditions have been improved regularly over the past 50 years, and taking into account the experience in the American industry the workers at present moment in the industry should not be suffering an overall excess risk of Lung Cancer of more than 20 or 30% above the normal figure. Now, that is a figure drawn out of the hat - like Dr. Norseth's PAH figure of 40 $\mu g/m^3$. But this risk must be less than the risk in the old plants that have ceased operating. The risk in the new plants that have been reported in Norway seems likely to be partly an artifact due to not knowing what is the correct reference population. Therefore it is reasonable to postulate that the workers in the industry at present moment are experiencing a risk of not more than 50% above the normal after 15 years exposure and perhaps even less. Now that does not take into account cancer of the bladder and possibly cancer of the kidney but these two diseases are both much less common than cancer of the lung. Proportionately the additional risk in these conditions is still quite small. I don't know if that is a sufficiently precise answer to Dr. Dahlberg's question, but I can assure you it is a more precise one than the facts really allow me to give.

Dr. Dahlberg: Thank you. I appreciate that.

Chairman: Time is up and the debate ended. Everyone has had a great deal to say on the scientific experimental, medical epidemiological and industrial hygiene sides as well as on behalf of industry, management, labour, the regulatory agencies and official institutions. There have indeed been a lot of interesting angles and valuable contributions in the discussion. I wish particularly to thank the panel members and the symposium participants from the floor for their contributions to the debate.