

Mortality Patterns Among Men Exposed to Methyl Methacrylate

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A cohort of 2,671 men, 1561 of whom were exposed to methyl methacrylate, was observed from 1951 to 1983 for mortality. This cohort consisted of men from two plants. This study utilizes detailed exposure estimates and smoking status to evaluate mortality patterns. We find no statistically significant excess all-cause or cause-specific mortality. Analysis of dose of methyl methacrylate with several cancer sites showed no trend. Men exposed to high levels of methyl methacrylate had cancer rates similar to those for men not exposed in the workplace, as well as for other men in the US population. An earlier epidemiology study reported a significant excess of colorectal cancer among persons exposed to several substances including methyl methacrylate. Our study results and results from animal studies do not support the hypothesis that methyl methacrylate is a human carcinogen.

Commercial production of methyl methacrylate (MMA) in the United States began in the late 1930s. MMA has many commercial applications including the production of acrylic sheet, acrylic molding and extrusion powders, as well as acrylic fibers. The National Institute for Occupational Safety and Health estimated in 1974 that 30,000 workers in the United States had been exposed to MMA.¹ Even with this widespread exposure there has been very little evidence that workplace exposure to MMA has caused serious health effects, other than possibly dermatitis.² Long-term effects such as cancer had not been addressed,¹ although animal studies do not indicate that MMA is a carcinogen.³

Recently, a study of workers at a Rohm and Haas

plant exposed to both ethyl acrylate (EA) and MMA among other substances reported a significant excess of colorectal cancers (K. Maher and L. De Fonso, unpublished data, March 1984). Because of the age of the operation in this study, it was difficult to determine levels and types of exposure. MMA was neither implicated nor eliminated as a possible cause of the excess (L. De Fonso and K. Maher, unpublished data, January 1986).

In view of the above, it was decided to examine the mortality patterns of two facilities in American Cyanamid where MMA is used. Both facilities had a large number of persons exposed to MMA for long periods. There were also exposure monitoring programs of long duration which allowed estimation of exposures for all workers at both plants. In addition, smoking histories had been recorded in the medical records of approximately 60% of the study population so that possible confounding of exposure with smoking could be addressed.

Materials and Methods

Two plants of the American Cyanamid Company are included in this study, the Fortier plant and the Santa Rosa plant. The Fortier plant manufactures MMA as well as several other materials and the Santa Rosa plant uses MMA in the manufacture of acrylic fiber. The study population consisted of all men who worked at either plant from start-up (1951 for Fortier and 1957 for Santa Rosa) until Jan 1, 1974. A total of 2,671 men, both whites and nonwhites, were identified and were observed to the end of 1983 using the Social Security Administration, the National Death Index, and American Cyanamid company records. At the end of the study, 2,373 (89%) were alive, 237 (9%) were dead, and 61 (2%) were lost to follow-up. Of the 237 deaths, death certificates were obtained and cause of death was ascertained for 224

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(95%). The 13 deaths for which no death certificate could be obtained were assumed to have died from unknown causes. Of the 2,671 men included in the study 1,302 were from the Fortier plant and 1,361 were from the Santa Rosa plant; eight persons worked at both plants. Combining men from both plants there were 2,473 whites and 198 nonwhites in the cohort.

Exposure Estimates

Exposure estimates for all jobs at both plants over time were developed. This process was made possible because of unique conditions at each plant. Industrial hygiene monitoring began at both plants in 1977 prior to many major engineering changes. Monitoring information in 1977 was, therefore, considered representative in most cases back to plant start-up. The exposure estimates were developed in five steps.

First, a department/job description was created for each plant. Job history information including department, job name, start date and stop date were taken from a job history card for each employee and entered into a computer file. A list by department and job with start and stop dates was then generated and sent to each plant. Similar departments and jobs in departments were combined until several plant personnel were satisfied that the resulting department/job description accurately represented the plant structure over time.

Second, and concurrent with the development of department/job description, the chemical exposures likely to be associated with the various jobs in departments were compiled, and an exposure profile for each department/job combination was developed. This was done by first identifying all processes that were present in each plant since start-up to Dec 31, 1983. Each of these processes was then evaluated to determine whether there was any potential for contact with known or suspected carcinogens. The substances with such potential were acrylamide, acrylonitrile, epichlorohydrin, formaldehyde, and methyl methacrylate at Fortier and acrylonitrile, methyl methacrylate, and vinylidene chloride at Santa Rosa.

The third step was to then determine which department/job/process combinations had exposure to the substances listed above. Persons at the plant familiar with department and processes working with the study staff made these determinations. In each case, department/job/process combinations were made time-specific by combining information from the personnel record with information on process start and stop dates. For Fortier there were 74 job titles in 15 departments which had exposure to one or more of the substances listed above and 25 job titles that had exposure to MMA. For Santa Rosa there were 15 job titles in 9 departments which had exposure to one or more of these substances, and all 15 jobs had exposure to MMA.

The fourth step was to obtain a history of each department regarding engineering controls, staffing changes, respirator use, and process changes which would affect exposure. This history was obtained by

interviews with persons who were familiar with the history of the department. All of these department changes were also related to a specific date.

The final step was to estimate exposures for each of the aforementioned substances for each department/job combination. This was completed by the use of extensive monitoring data and interviews of plant employees who had knowledge of past jobs and processes. Finally, exposures were assigned to each job/department combination using a coding form. When available, actual measurements were used to indicate a job exposure to the substance under study. In the absence of job-specific measurements, estimates of exposure were made. Through discussion with plant personnel, present day measurements were adjusted for changes in handling practices and installation of engineering controls. This method for quantifying exposure in an occupational study is similar to methods described elsewhere.⁴

Table 1 presents mean eight-hour time-weighted average estimated exposures to MMA by period of exposure at each plant. Exposure to MMA began earlier at Santa Rosa than at Fortier. In addition, exposure to MMA was higher at Santa Rosa than at Fortier. Generally, exposures declined over time at both plants, with the most recent exposures being the lowest. At both plants during each time period, large numbers of persons were exposed to MMA. However, few employees received exposure more than 20 years prior to the study cutoff date.

Smoking History

Smoking histories of workers were taken from medical records. Company physicians interviewed employees regarding smoking habits in the medical examination routinely administered at both plants since 1966. Prior to that time, some physicians would ask about smoking history and record it in the medical record. Although the medical examination was offered to all employees, it was voluntary and not all employees consented to take it. However, preemployment examinations were required for all employees. Periodic physical examinations were offered yearly to employees 40 years old or older and once every 2 years to employees younger than 40. Medical records were available on 74% of the men, and 78% of these records had smoking information recorded. Results of all medical examinations were reviewed and coded with one of three codes. If the medical record indicated a person smoked cigarettes, cigars, or pipe, this person was coded as a smoker. Medical records which stated a person was a nonsmoker or smoked less than 3 months were coded as "nonsmoker." When no information was provided in the medical record about smoking, a person's smoking status was coded as unknown. In all, 1,541 men had smoking information recorded. Generally, workers hired in earlier years were more likely to have smoking information. For instance, of the men hired prior to 1955, 64% had smoking information, whereas of the men hired between 1955

TABLE 1

Mean Established Eight-Hour Time-Weighted Average Exposures to Methyl Methacrylate in Parts Per Million (ppm) for the Fortier and Santa Rosa Plants by Period of Exposure

| Period of Exposure | Fortier | | Santa Rosa | |
|--------------------|---------------------------|-----------------|---------------------------|-----------------|
| | Average ppm, Mean (Range) | Persons Exposed | Average ppm, Mean (Range) | Persons Exposed |
| Prior to 1960 | — | — | 1.00 (0.05–4.00) | 137 |
| 1960–1964 | 0.38 (0.00–1.75) | 48 | 0.89 (0.05–9.00) | 373 |
| 1965–1969 | 0.69 (0.00–7.83) | 470 | 0.90 (0.05–7.50) | 546 |
| 1970–1974 | 0.44 (0.00–2.94) | 374 | 0.80 (0.05–11.00) | 292 |
| 1975–1979 | 0.35 (0.00–6.34) | 636 | 0.82 (0.05–11.50) | 398 |
| 1980–1983 | 0.13 (0.00–1.42) | 468 | 0.72 (0.05–5.40) | 118 |

and 1964 and 1965 and 1973, 54% and 60%, respectively, had smoking information.

Analytic Procedures

Person-year accumulation for the cohort began either at plant start-up (1951 for Fortier and 1956 for Santa Rosa), when persons started employment at the plant (if after plant start-up), or upon the persons' first achieving a specified level of exposure, depending upon the type of analysis being undertaken. Person-year accumulation stopped either at the closing date of study (Dec 31, 1983), at the last day worked if the person was lost to follow-up, at the date of death, or at the date of reaching a higher exposure category.

Standard mortality ratios (SMR) and a direct standardized relative risk measure as described by Gilbert and Buchanan⁶ were employed to estimate risk. The relative risk measure not only avoids the biases associated with the use of external controls,⁶ but also has the added advantage of controlling for characteristics such as smoking, latency, and plant as well as race and sex. Both the SMRs and the relative risk measure were calculated by using a program developed by Marsh and Preininger.⁷ For the SMR 95% confidence intervals were calculated using the method of Bailer and Ederer⁸ and a χ^2 test was used to evaluate statistical significance of the trends.⁹ For the relative risk measure, 95% confidence intervals were based on the procedure of Miettinen¹⁰ and the test for the significance of trend is based on method developed by Mantel.¹¹

Results

Table 2 compares SMRs for total men exposed to MMA at either plant with total men who are not exposed. Exposure is defined as a cumulative exposure greater than 0.1 part per million (ppm), where cumulative exposure is calculated as the product of the number of days in the job and estimated average exposure in parts per million divided by 365. All persons contribute person-years to the unexposed category until they are exposed to 0.1 ppm cumulative exposure.

In Table 2, 1,971 persons contributed 26,391 person-years to the no exposure group. The SMR for all causes is 0.79, and for all malignant neoplasms, 1.01. For the

TABLE 2

Standardized Mortality Ratios (SMR) and Observed (obs) and Expected (exp) Deaths for Men by Exposure Grouping

| | SMR* (obs/exp) | |
|--|---------------------|----------------------|
| | Not Exposed | Exposed |
| All causes | 0.79 (123/156.5) | 0.67† (114/169.7) |
| All malignant neoplasms | 1.01 (30/29.7) | 1.04 (35/33.5) |
| Buccal cavity and pharynx | 1.95 (2/1.0) | 1.70 (2/1.2) |
| Digestive organs and peritoneum | 1.22 (9/7.4) | 0.74 (6/8.1) |
| Large intestine | 0.87 (2/2.3) | 0.39 (1/2.6) |
| Respiratory system | 0.75 (8/10.7) | 1.20 (15/12.5) |
| Bone | 5.77 (1/0.2) | — (0/0.2) |
| Skin | — (0/0.7) | — (0/0.9) |
| Prostate | 0.91 (1/1.1) | 2.01 (2/1.0) |
| Testis | — (0/0.4) | — (0/0.4) |
| Bladder | — (0/0.6) | 3.16 (2/0.6) |
| Kidney | 1.31 (1/0.8) | — (0/0.9) |
| Eye | — (0/0.0) | — (0/0.0) |
| Brain and other CNS | 1.63 (2/1.2) | — (0/1.4) |
| Thyroid | — (0/0.1) | — (0/0.1) |
| All lymphopietic cancers | 1.16 (4/3.5) | 1.06 (4/3.8) |
| Benign neoplasms | — (0/0.5) | — (0/0.1) |
| All infectious and parasitic diseases | 0.57 (1/1.7) | 0.67 (1/1.5) |
| All diseases of the circulatory system | 0.75† (49/65.6) | 0.69 (11/15.9) |
| All respiratory diseases | 0.53 (4/7.5) | 2.16 (4/1.9) |
| All external causes | 0.66 (20/30.5) | 0.87 (2/2.3) |
| Death certificate not available | 13 | 3 |
| Persons | 1,971 | 1,561 |
| Person-years | 26,391 | 28,021 |

* Indirectly standardized for race (white, nonwhite).

† $P < .05$.

no exposure group, all diseases of the circulatory system is significantly less than expected with an SMR of 0.75.

There were 1,561 men exposed to MMA with 28,021 years of follow-up. For men exposed, the SMR for all causes was 0.67, significantly less than expected. All

TABLE 3
Tests for Trends for Indirectly and Internally Standardized Risks for Exposure to Methyl Methacrylate for Several Categories of Cancer

| Cause of Death | Cumulative Dose, ppm-yr | | | | P Values for Trend |
|--|-------------------------|--------|----------|-------|--------------------|
| | None | 0-0.19 | 0.20-2.0 | 2.0+ | |
| All malignant neoplasms | | | | | |
| Observed deaths | 30 | 6 | 11 | 18 | |
| Expected (indirect standardization)* | 29.7 | 5.5 | 12.6 | 15.5 | .622 |
| Expected (internal standardization)† | 31.3 | 5.8 | 12.0 | 15.9 | .957 |
| Cancer of the digestive organs and peritoneum | | | | | |
| Observed deaths | 9 | 2 | 1 | 3 | |
| Expected (indirect standardization)* | 7.4 | 1.2 | 3.0 | 3.8 | .533 |
| Expected (internal standardization)† | 6.4 | 1.4 | 2.6 | 4.6 | .390 |
| Cancer of the large intestine | | | | | |
| Observed deaths | 2 | 1 | 0 | 0 | |
| Expected (indirect standardization)* | 2.3 | 0.4 | 0.9 | 1.2 | .246 |
| Expected (internal standardization)† | 1.9 | 0.2 | 0.3 | 0.6 | .484 |
| Cancer of the respiratory system | | | | | |
| Observed deaths | 8 | 1 | 6 | 8 | |
| Expected (indirect standardization)* | 10.7 | 1.9 | 4.5 | 6.1 | .229 |
| Expected (internal standardization)† | 10.6 | 2.1 | 4.5 | 5.8 | .388 |
| All lymphoietic cancer | | | | | |
| Observed deaths | 4 | 0 | 1 | 3 | |
| Expected (indirect standardization)* | 3.5 | 0.7 | 1.5 | 1.5 | .293 |
| Expected (internal standardization)† | 4.3 | 1.1 | 1.5 | 1.1 | .667 |
| Persons | 1,973 | 1,561 | 1,190 | 577 | |
| Person-years | 26,391 | 7,218 | 12,818 | 7,985 | |
| Death certificate not available | 13 | 0 | 2 | 1 | |
| % with unknown smoking history | 42.1 | 32.8 | 25.3 | 9.9 | |
| % smokers (excluding persons with unknown smoking history) | 72.2 | 72.4 | 73.0 | 73.8 | |

* Indirectly standardized for age, period, and race with the US population used as the comparison.

† Directly standardized for age (<45, 45-54, 55-64, 65+ years), period (<1965, 1965+), race (white, nonwhite), latency (<10, 10-20, 20+ years), and smoking (smokers, nonsmokers, unknown) with the comparisons being all remaining groups.

malignant neoplasms were almost exactly as expected compared with the U.S. population (SMR = 1.04) and no different than among those men at the same plants unexposed. Cancers of both digestive organs and peritoneum (SMR = 0.74) and large intestine (SMR = 0.39) were at expected levels, unlike the single previous study done on workers exposed to MMA.³ All other categories were at expected levels. In general, men exposed to MMA had mortality levels similar to the US population and to men at the same plants not exposed. No excess mortality from colon cancer was observed. No rectal cancer occurred in this study population.

Mortality among men by increasing intensity of exposure is shown in Table 3 for selected causes of death. Causes of mortality were selected if they had eight or more deaths occurring. Cancer of the large intestine was also selected because of the excess in a previous study. Four exposure categories were chosen: none, or less than 0.000247 ppm (0.1 divided by 365), 0.0 to 0.19, 0.20 to 2.0, and more than 2.0. These categories were chosen to place large numbers of persons in the highest exposure category.

Both indirect and internal standardization techniques were used to test for trend. The indirect standardization method took into account age, calendar year, and race whereas the internal standardization method additionally took into account latency and smoking status. Controlling for smoking status appeared important, as there was an increase in smokers with increasing cumulative

exposure. The difference, however, among all categories was small, ranging from 72.2% in the "none" exposure group to 73.8 in the highest exposure group.

There also appears to be better ascertainment of smoking status with increasing exposure. In the highest exposure group only 9.9% of the persons have unknown smoking status compared with 42.1% for the none exposure group. This would seem to result from a high proportion of long-term workers in the higher exposure categories. Since all workers are offered yearly medical examinations where smoking status is recorded, long-term workers have more opportunities to have a medical examination. The effect this bias has on the results of the trend analysis is unknown but is probably small, since excluding persons with unknown smoking status does not significantly change the results.

Examination of the P values for the test of trend in Table 3 revealed no significant trends for either the indirect or direct standardization methods for any category of cancer examined. Cancer of the large intestine, the site elevated in a previous study, demonstrated no trend, with no deaths occurring in the two highest exposure groups.

Discussion

This study examined the mortality experience of 2,671 men, 1,561 of whom were exposed to significant

amounts of MMA at two plants. The exposures varied considerably by plant, by era, and by job assignment. This study was somewhat unique in view of the extensive amount of available industrial hygiene data. These data in conjunction with detailed work histories made it possible not only to determine who was exposed to MMA, but also to estimate levels of exposure for each employee.

The Fortier plant manufactured MMA and the Santa Rosa plant used MMA in the manufacture of acrylic fiber. Exposures were generally higher at Santa Rosa, ranging from 0.05 to 11.50 ppm. Exposures at the Fortier plant ranged from 0 to 7.83 ppm.

Follow-up of the cohort members in this study was good for a study of this type. Only 2% of population was lost to follow-up, and death certificates were obtained for all but 5% of persons presumed dead. Of the 114 deaths occurring among men exposed to MMA, only three death certificates, or 2.6% could not be located.

The observed deaths for both exposed and unexposed groups for all causes of death were less than expected. Among those not exposed, the SMR was 0.79 and among the exposed, 0.67. These low SMRs most likely are the result of both initial selection and the ongoing benefits of employment,¹⁸ although an SMR of 0.67 observed among those persons exposed to MMA is very low for any population.

Thirty-five cancer deaths occurred among the 1,561 persons exposed to MMA, whereas 33.5 cancer deaths were expected. For specific cancer sites among the exposed group, no significant excesses were observed in the 16 sites examined. Of special interest in this study were the sites that were elevated in the previous epidemiology study, namely colorectal cancers.⁹ No excesses were observed at these sites among men exposed to MMA. For cancer of the large intestine, 1 death was observed and 2.6 expected. There were no deaths from cancer of the rectum.

Analyses of trends by cumulative exposure and duration of exposure showed no increasing risk of mortality by increasing exposure. These analyses took into account smoking status, age, calendar year, race, and latency. For all cancers, and for cancers of the digestive organs and peritoneum, large intestine, respiratory, and all lymphoietic cancers, no increasing trend in risk is observed with increasing exposure or duration of exposure.

A previous study found a significant excess of cancer of the large intestine and rectum (SMR = 1.67 or 52 observed, 31.2 expected) in a group of persons who worked in processes where, among other operations, acrylates and methacrylates were produced, polymerized, and fabricated (K. Maher and L. De Fonso, unpublished data, March 1984). However, this excess could not be related to dose or latency. Furthermore, a case-control study on this same population indicated that working in plant production departments that involved exposure to EA and/or MMA as the monomer, the polymer, or the polymer dust is not associated with significantly increased risk of colorectal cancer (L. De Fonso and K. Maher, unpublished data, January 1986). In this study no excess of colorectal rectal was observed.

There were no rectal cancers, and the SMR for cancer of the large intestine among those exposed to MMA is 0.39 (1 observed and 2.6 expected). The SMR for the category of cancer of the digestive organs and peritoneum is only 0.74 (6 observed and 8.1 expected). From these results it does not appear that exposure to MMA is related to increased risk of colorectal cancer. The excess of colorectal cancer observed in the previous study may be a chance finding, related to an unmeasured or poorly measured occupational exposure, or be related to a background factor such as diet. MMA exposure is an unlikely cause of this excess.

However, it should be noted that there were several notable differences between the previous study on MMA and this study. First, persons in the previous study most likely had higher MMA exposures because of the older age of the facility under study. Second, persons in the previous study had much longer latency intervals than did those in the present study. Finally, the previous study's population had longer durations of exposures. These differences in the two study populations could conceivably account for differences in risk for colorectal cancer between the two studies. However, this is considered unlikely since the trend of risk with increasing dose was absent in the present study when latency was considered.

Another important difference between the two studies was plant location. In the previous study, the plant was located on the Pennsylvania and New Jersey borders in an area of the country with very high colorectal cancer rates.¹³ Colorectal cancer has high regional variability with high rates in the Northeast.¹³ In the 1970s, Bucks County, Pennsylvania, the county in which the previous study's plant was located, was at the 75th percentile for age-adjusted rates of death from cancer of the large intestine.¹⁴ This large colon cancer rate in the region where the previous study's plant was located may explain the colon cancer excess when this plant population was compared to the United States.

This study was large enough to have detected among MMA workers a 45% increase in total cancers, a 100% increase in respiratory cancers, and a six-fold increase of cancer of the large intestine, based on a two-tailed 5% significance level with a power of 80%. No significant excesses were observed for any cancer grouping. In addition, all tests for trends taking into account several possible confounding factors showed no increasing risk with increasing dose of MMA. Based on these study results, there is no evidence to support the hypothesis that MMA is a human carcinogen.

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The Regressed Moral State

The working model of moral conflict has been that of emotion warring against reason, with only reason's mastery offering trustworthy guidance. A more careful analysis of the regressed state would see that the moral conflict is usually a case of one immature thinking-emotive moral scenario in conflict with another more wholly owned and appropriately mature moral scenario. . . .

Rational persons may have a more difficult time noticing and assessing those less dramatic but equally disabling disorders consisting of conflicts of emotion. In philosophical arguments the problem of such deficits is regularly ignored and that of excessive emotion emphasized. Yet in our technological culture perhaps the greatest moral danger arises not from sentimentality, but from devaluing feeling and not attending to or nurturing moral emotions.

Some persons are too "burned out" from stress to see or care about moral dilemmas. Others are so accustomed to isolating and not attending to their emotions that when they inadvertently must confront feeling, they are overwhelmed by what seems to them an alien external force. They are all the more susceptible to moral collapse and making poor ethical decisions.

Habits of numbing or suppressing emotion spread to other domains in a personality and impair moral thinking as surely as excessive, infantile emotions do. The human mind for brief periods can go into detached, depersonalized overdrive and function automatically like a computer. We have seen detached analysis destructively employed by the best and brightest. The maintenance of moral emotions and the care and cultivation of moral sentiment should be seen as all-important; after all, the rational tutoring of emotion depends upon people who already possess a highly developed emotional repertoire. Those concerned with educating health care workers know that the absence of emotional responses of empathy and sympathy become critical bioethical issues.

—From "The Role of Emotion in Decision Making," by
Sidney Callahan in *Hastings Center Report*, 1988;
18:12.