# Chemical Exposures of Rocket-Engine Test-Stand Personnel and Cancer Mortality in a Cohort of Aerospace Workers

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*We conducted a retrospective cohort study of 6107 aerospace workers to examine whether exposure to chemicals—primarily hydrazine fuels—during rocket-engine fueling and testing affects cancer mortality. When conditional logistic regression analysis was applied and adjusted for confounding variables, the estimated rate ratio for lung cancer mortality, comparing exposed to unexposed workers from the same facility, ranged from 1.68 (95% confidence interval, 1.12 to 2.52) to 2.10 (95% confidence interval, 1.36 to 3.25), depending on job-duration threshold (6 or 24 months) and lag (0 to 15 years). Similar results were obtained for hemato- and lymphopoietic cancer and for bladder and kidney cancer mortality, but estimates for these cancers were imprecise. We concluded that occupational exposure to hydrazine or other chemicals associated with rocket-engine testing jobs increased the risk of dying from lung cancer, and possibly other cancers, in this population of aerospace workers; however, our results need to be replicated in other populations.*

In the early 1990s, a worker health study was initiated in response to strong concerns voiced by area residents about the use of radioactive and toxic chemical substances at a nuclear reactor and rocket-engine testing facility in the Greater Los Angeles area (hereafter referred to as Santa Susana Field Laboratory [SSFL]). To examine the possible effects of chemicals used at these test stands on cancer mortality, we conducted a retrospective cohort study of workers employed at the SSFL between 1950 and 1993 who engaged in testing rocket engines. The company was a very active participant in the national space program in the 1960s. Rocket engines were fueled by a number of propellants, including hydrazine, 1-methylhydrazine (MMH), and 1,1-dimethylhydrazine (UDMH) (referred to collectively as hydrazine throughout this article). Hydrazines were used in large quantities at the facility, especially between 1955 and 1970. Hydrazine and UDMH have been classified by the US Environmental Protection Agency[1] as Group B2, a probable human carcinogen, and by the International Agency for Research on Cancer [2] as Group 2B, a possible human carcinogen, on the basis of sufficient evidence in animals and inadequate evidence in humans. On the basis of animal studies, MMH has been classified by the Environmental Protection Agency as a probable carcinogen (Group B2), but there is no evidence to date of its carcinogenicity in humans. In addi-

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tion, there is some evidence that the oxidation of UDMH results in nitrosamine formation,[2a,b] thus potentially exposing rocket-engine test-stand personnel to chemicals recognized as potent animal carcinogens. For example, the oxidation of UDMH would produce dimethylnitrosamine, a probable carcinogen according to the International Agency for Research on Cancer.[3] The processes that resulted in such oxidation would have exposed workers to both carcinogens.

Exposure to chemical carcinogens among rocket-engine testing personnel was not limited to hydrazine, ie, work in the areas of rocket-engine testing may have exposed some workers to trichloroethylene (TCE), asbestos, beryllium, and other chemicals. We did not have sufficient information to assess exposures to these other potential carcinogens in the workplace. Because hydrazine exposure is known to cause lung cancer in animals,[2] our study focused on the estimation of the possible effects of hydrazine exposure on lung cancer mortality. Because very little is known about the effect of hydrazine in humans,[2] we examined the associations between hydrazine exposure and death from a number of cancers: hemato- and lymphopoietic cancers, bladder and kidney cancers, upper aerodigestive tract cancers (oral cavity and pharynx, larynx, and esophagus), and pancreatic cancers.

## Methods

### Study Design and Subject Selection

Personnel records allowed us to define the study population and to obtain workers' employment information. From the total population employed at the Rocketdyne/AI division of Rockwell between 1950 and 1993 (approximately 55,000 employees, according to company personnel files), we selected all men who were employed before 1980 at the SSFL, who had worked for at least 2 years in any division, and who had never been monitored for radiation exposure, according to company records. The gender and employment restrictions were made to limit the size of the population, thus making a comprehensive mortality follow-up feasible.

Crosschecking personnel records against a transfer book documenting the transfer of employees between divisions revealed that the personnel office lacked records for 1690 of the listed Rocketdyne division employees. After an extensive record search involving all divisions, we were able to obtain 1063 (63%) of the missing records from other divisions. Only 248 of these retrieved records, however, belonged to workers who met our eligibility criteria. Thus we estimate that records were missing for 146 eligible workers (2.4% of all eligible cohort members). The final cohort included 6107 Rocketdyne division employees.

### Death Certificates

Cause of death for deceased workers was obtained from death certificates that were retrieved from Rocketdyne/AI pension files and state vital statistics offices. If two independent company data sources identified an employee as active at the end of follow-up, we counted that person as alive. Vital status for approximately 10% of the hydrazine cohort was determined as alive, using this method. Employees not identified as alive or dead by company records were matched against three different record systems: the Social Security Administration beneficiary records files (for the period 1935 to 1994), the vital statistics files for the state of California (for the period 1960 to 1994), and the US National Death Index (for the period 1979 to 1994). Matches were verified by reviewing the information on the death certificates.

All death-identification systems together guarantee a vital-status search that is complete enough to justify that a person was treated as alive at the end of follow-up, if not identified as dead by at least one of the three computerized services or Rocketdyne/AI beneficiary records. From all sources combined, we identified 1391 subjects who died between 1960 and 1994. We were able to obtain all but one of the death certificates for the deceased workers.

A licensed nosologist coded the cause-of-death information recorded on each death certificate, using the *International Classification of Diseases—9th Revision* (ICD-9).[4,5] Both the underlying and associated (contributing) causes were coded, but the analyses presented in this article are based on underlying causes only. The coding was checked for accuracy, and discrepancies were discussed and reconciled by two members of the study team.

### Chemical Exposures

With the help of walk-through visits, interviews with managers and workers, and historical facility reports, we conducted an extensive industrial-hygiene review of the SSFL facility. The principal chemicals used at rocket test stands were hydrazines (hydrazine, MMH, UDMH), kerosene fuels, chlorine, fluorine, hydrogen peroxide, isopropyl alcohol, nitric acid, nitrogen tetroxide, TCE, and 1,1,1-trichloroethane. A smaller number of workers may have been exposed to asbestos and beryllium. Other chemicals were used at the test stands in considerably smaller quantities. Furthermore, workers may have been exposed to polycyclic aromatic hydrocarbons from the incomplete combustion of kerosene fuels and to nitrosamines formed via hydrazine oxidation. Having to rely on job titles for our exposure assessment made it impossible to differentiate between the three hydrazines or to identify workers exposed to nitrosamines derived from UDMH. Because we were not able to obtain air-monitoring data for any chemicals used at Rocketdyne/AI prior to 1985, we focused on one group of chemicals—hydrazines—with known animal carci-

nogenicity for which proxy measures of exposure could be developed from the available information.

We were unable to link workers with job locations such as a specific rocket-engine test stands because company records did not provide the necessary information. Thus we relied on job titles, job codes, and employment periods, combined with the information from worker and manager interviews and company records, to construct a job-exposure matrix for hydrazine exposure for personnel working at rocket-engine test stands. We were able to locate chemical inventories for hydrazine for the years 1955 through 1994, documenting the amount bought by the facility. Workers were assigned to four categories of presumptive exposure (high, medium, low, and unexposed) reflecting the relative probability of hydrazine exposure, rather than the amount of exposure. All exposure classification was done in a manner blinded to survival status and cause of death. The high-exposure group included workers who had been employed as propulsion/test mechanics or propulsion/test technicians for a minimum of 6 or 24 months (HYD-6 and HYD-24) and who had been responsible for pumping hydrazine into test-stand fuel tanks and rocket engines. Fuel-loading procedures officially involved "closed systems" to avoid exposure, but leakage of fuel from the system was allegedly a common occurrence. The medium-exposure (propulsion/test inspector, test engineer, research engineer, and instrumentation mechanic) and low-exposure (eg, flight-line mechanics and engineers) groups included employees who, according to their job titles, may have been present during engine test-firings but were unlikely to have had direct contact with hydrazine through fueling procedures. At any time during follow-up, a worker was assigned to the highest category for which he qualified. The unexposed group included all workers who did not qualify for any of the exposed categories (given the 6- or 24-month criterion).

For a short period, facility employees experimented with beryllium as a solid rocket propellant, potentially exposing a small number of workers (fewer than 20). Asbestos was used at engine test stands as insulation around high-temperature wires and lines. Tasks involving the cutting, removal, and installation of asbestos materials may have involved some exposure. TCE was used in large quantities to clean engines fueled by liquid oxygen and kerosene and to remove combustible deposits and vapors. Nevertheless, because of the pervasiveness of solvent use throughout the facility and operations and the lack of exposure monitoring and worker-location data, it was impossible to identify exposed workers or to rate job titles with respect to solvent exposure.

## Personnel and Medical Records

Personnel records were used to create a three-category measure of pay type as a proxy for socioeconomic status: union employees paid on an hourly basis, salaried technical/administrative employees, and managerial/professional employees. Subjects who changed jobs during the follow-up period were categorized according to those jobs they had held for the longest period of time at Rocketdyne/AI.

Because Rocketdyne/AI did not systematically collect data on the race of its employees before 1972, we were unable to control for this factor in our analyses. According to the information on death certificates, however, 96% of all deceased workers were white.

Information about tobacco smoking was systematically recorded for two groups of workers in routinely administered medical questionnaires from different periods. Between 1961 and 1969, questionnaires completed by selected workers indicated current smoking status (smoker or nonsmoker); after 1980, questionnaires completed by selected workers indicated smoking history (current or former smoker or never smoker). Because information on smoking was not available for most subjects in our study population, we assessed the potential confounding of hydrazine effects by examining the association between smoking status and hydrazine exposure in the subset of subjects for whom smoking information was available (295 workers).

## Statistical Methods

Given the methods described above for the selection of workers for the hydrazine analyses, follow-up for this cohort began at the latest of three dates: (1) the start of work at the SSFL; (2) the start of work at any Rockwell division, plus 2 years; or (3) January 1, 1950. Follow-up ended on the date of death or December 31, 1994, whichever date came earlier.

Lung cancer mortality (ICD-9 code 162) was the major outcome of interest. In addition, we also examined the possible effects of hydrazine on death from hemato- and lymphopoietic cancers (including leukemias, lymphomas, lymphosarcomas, reticulosarcomas, myelomas, and Hodgkin's disease; ICD-9 codes 200 to 208), bladder and kidney cancers (exit organs; ICD-9 codes 188 and 189), upper aerodigestive tract cancers (oral cavity and pharynx, larynx, and esophagus; ICD-9 codes 140 through 150 and 161), pancreatic cancer (ICD-9 code 157), and emphysema (ICD-9 code 492). In addition to the exploratory objective of these analyses, there was also a methodologic objective: Because we did not have sufficient data on smoking to control for this variable in the analysis, we wanted to assess the associations between hydrazine exposure and several smoking-related diseases, aside from lung cancer.

To estimate hydrazine effects, we utilized the risk-set approach for follow-up data described by Breslow and Day.[6] In this approach, conditional logistic regression is used to

compare individuals who have died of the index disease with individuals still at risk of dying (the risk set of "survivors"). Risk sets for all analyses presented in this report were based on postmatching survivors to index deaths on calendar time. As an alternative approach in certain analyses, we postmatched survivors to index deaths on age, but this approach yielded approximately the same estimates for hydrazine effects.

Three time-dependent indicator variables, representing high, medium, and low exposure to hydrazine at the time of the index death, were included as predictors in the logistic-regression models. Because there were very few subjects in the low-exposure hydrazine group, however, effect estimates for this category are not reported. To allow for varying periods of induction/latency between exposure and death and to deal with possible selection bias, exposure measurements were lagged by zero, 10, and 15.[7] Lagging was achieved by ignoring exposure for each subject in a risk set within zero, 10, or 15 years of the index death. In addition, the number of years spent in high-exposure hydrazine jobs was treated as a time-dependent continuous predictor in separate models. Because the potential for hydrazine exposure within jobs probably changed over time, we also modeled the effect of high exposure (for at least 6 months) by decade of exposure (1950 through 1989). This was done by including in the model a binary variable for high exposure in each of four decades.

Rate ratios and 95% confidence intervals (CIs) for exposures were derived from the estimated logistic parameters and standard errors. To control for confounding, we included the following covariates in each model: age at death (continuous), pay type (two fixed binary variables), and time since hire or transfer to SSFL (continuous) at the time of death of the worker. Time since hire or transfer was included to control for the selective loss of less healthy workers.[8]

**TABLE 1**
Description of the Cohort

| | |
|---|---|
| Number of subjects | 6,107 |
| Percent Male | 100 |
| Average follow-up time, years | 29.0 |
| Average age at start of follow-up, years | 34.6 |
| Number of person-years of follow-up | 176,886 |
| Number of total deaths | 1,391 |
| Number of cancer deaths | 404 |
| Total mortality rate, per 100,000/year | 786 |
| Total cancer-mortality rate, per 100,000/year | 228 |
| Pay type, percentage of total | |
| Salaried managerial/professional | 45.1 |
| Salaried technical/administrative | 43.7 |
| Hourly/union | 11.3 |
| Unknown | 0.0 |

## Results

Our cohort is characterized by a long average follow-up time (29 years) and a high percentage of salaried employees (89%) (Table 1). There were 1391 total deaths (19% of the total), of which 404 (29%) were from cancer as the underlying cause, yielding a total cancer mortality rate of 228 per 100,000/year. During the follow-up period, approximately 28% of the men in the cohort were classified as presumptively exposed to hydrazine, using the HYD-6 criterion for defining exposed jobs, and, using the HYD-24 criterion, approximately 24% were exposed; most of these exposed subjects were in the high-exposure group (Table 2).

The estimated effects of high and medium hydrazine exposure on lung cancer mortality are listed in Table 3. The estimated rate ratio (RR) for the high-exposure group, relative to the unexposed group, ranged from 1.68 (95% CI, 1.12 to 2.52) to 2.10 (95% CI, 1.36 to 3.25), depending on the minimum criterion for defining exposure (6 or 24 months) and the lag for measuring exposure (0 to 15 years). We observed a slight increase in the rate ratio with increasing lag. No excess rates were observed for subjects in the medium-exposure group (RR $\leq$1). By modeling years of work in high-exposure jobs as a continuous time-dependent predictor with zero lag, the RR per 10-year increment of high exposure was 1.65 (95% CI, 1.18 to 2.32); ie, each 10-year increase in the number of years spent in high-exposure jobs is associated with a 65% increase in the rate of lung cancer mortality.

Table 4 shows the adjusted effects of hydrazine exposure on other cancers possibly affected by the exposure, according to our criteria. For hemato- and lymphopoietic cancers, the RR for high exposure ranged from 1.27 (95% CI, 0.51, 3.14) to 2.83 (95% CI, 1.22, 6.56). The observed effect was noticeably stronger for HYD-6 than for HYD-24 and increased slightly with increasing lag (no shown); nevertheless, all of the estimates were imprecise (ie, all 95% CIs were relatively wide, and all but one included the null value). The RRs for medium exposure showed a similar pattern, but they tended to be smaller and even less precisely estimated. Separate analyses for leukemia and lymphoma mortality were not informative enough to distinguish which cancer type was associated with hydrazine exposure. The effects of hydrazine exposure on bladder and kidney cancer mortality are also imprecisely estimated. Depending on the lag, the RR for high exposure ranged from 1.50 (95% CI, 0.55 to 4.12) to 2.55 (95% CI, 0.94 to 6.86), and there were no deaths in the medium-exposure group. Again, sep-

**TABLE 2**
Number of Subjects, by Category of Presumptive Hydrazine Exposure* and by Minimum Duration (6 or 24 months)†

| Exposure Variable‡ | High | Medium | Low | Unexposed | Total |
|---|---|---|---|---|---|
| HYD-6 | 1,053 | 654 | 32 | 4,368 | 6,107 |
| HYD-24 | 827 | 592 | 42 | 4,646 | 6,107 |

* For each exposure variable, a worker is classified in the category of his or her highest exposure level at the end of follow-up.

† Either 6 or 24 months of employment in selected jobs is required for classification in the high-, medium-, or low-exposure categories. These categories reflect the relative probability of hydrazine exposure, rather than the amount of exposure.

‡ HYD-6, minimum of 6 months exposure to hydrazine; HYD-24, minimum of 24 months exposure to hydrazine.

arate analyses for bladder and kidney cancer mortality were not informative enough to distinguish which cancer type was associated with hydrazine exposure.

We also examined whether hydrazine affected smoking-related diseases other than lung cancer. We found that there was no apparent effect of high exposure with an excess mortality rate for all other smoking-related cancers together or for upper aerodigestive tract and pancreatic cancers separately, or for emphysema mortality (Table 5). The RRs for medium exposure tended to be greater than one but were estimated rather imprecisely.

The estimated effects of hydrazine (HYD-6) on lung cancer mortality and hemato- and lymphopoietic cancer mortality, by decade of exposure, are shown in Table 6. For both cancer outcomes, the effect of hydrazine exposure seems to be limited primarily to exposure received during the 1960s. The RR for high-exposure versus unexposed workers was 2.01 (95% CI, 1.21 to 3.33) for lung cancer and 2.45 (95% CI, 0.91 to 6.58) for hemato- and lymphopoietic cancer.

## Discussion

We observed positive associations between our proxy measure of hydrazine exposure and the rates of dying from cancers of the lung, blood and lymph system, and bladder and kidney. Nevertheless, consistent effects were not observed for medium-exposure levels, except for hemato- and lymphopoietic cancers, and effect estimation was relatively imprecise, except for high exposure and lung cancer, because of the small numbers of cancer deaths in the exposed categories. On the other hand, the RR for lung cancer mortality tended to increase with increasing lag, which is consistent with the long induction/latency for this cancer, and it was consistent across two operational definitions of exposure (HYD-6 and HYD-24). In addition, we were able to control for the potentially confounding effects of age, socioeconomic status (pay type), time since hire or transfer to SSFL (by statistical adjustment), calendar time (by postmatching), and radiation exposure (by selection of the cohort).

Although hydrazine is known to be carcinogenic in animals, there is no clear epidemiologic evidence linking hydrazine exposure with cancer risk in humans. In a British study of hydrazine production workers, Morris et al[9] found no effect of hydrazine exposure on cancer mortality, but this was a very small study of only 427 workers (25 cancer deaths, eight of which were from lung cancer). In an Italian study of workers at a thermoelectric power plant, Cammarano et al[10] reported an excess mortality rate for all cancers among workers with 10 or more years of employment (12 cancer deaths; 4.35 expected). These investigators, however, were not able to link the excess cancer mortality with hydrazine exposure because several carcinogens were present in the workplace.

Despite the findings of this study linking our measure of hydrazine exposure with cancer mortality, there are several methodologic problems that limit causal inference. Aside from the low precision in estimating effects, our exposure classifications were based entirely on job titles, not on quantitative estimations of exposure. Thus there was certainly exposure misclassification, which could have biased the results. Not only was it impossible to determine from job titles which workers were actually assigned to specific rocket test stands or buildings, but the exposure to hydrazine at any test stand typically resulted from accidental and unpredictable occurrences. Because we have no reason to believe, however, that this exposure misclassification was differential with respect to cancer outcome, we would expect the bias to be toward the null value. Such misclassification therefore implies that our hydrazine effects might be underestimated. Furthermore, the frequency of test firings and the amount of hydrazine, specifically UDMH, used at Rocketdyne was greatest between the 1950s and the early 1970s, which is roughly consistent with our results: We found that the effects of hydrazine exposure on lung cancer mortality and hemato- and lymphopoietic cancer mortality were limited primarily to exposure received during the 1960s. Nitrosamines are formed during hydrazine oxidation, and this suggests that workers were exposed to these known animal carcinogens while working at the rocket-engine test stands. Thus the effect we are observing for hydrazine exposure could also be due to the combination of exposure to both carcinogens at the test stands. Because of the lack of

**TABLE 3**
Adjusted Rate Ratios (RRs) and 95% Confidence Intervals (95% CIs)* for the Effects of High and Medium Hydrazine Exposure Versus No Exposure on Lung Cancer Mortality,† by Hydrazine Exposure (6- or 24-month criterion) and Lag (in years) for Hydrazine Exposure ($n$ = 6107; 146 cancer deaths)

| | | High Exposure | | | Medium Exposure | | |
|---|---|---|---|---|---|---|---|
| Hydrazine Variable | Lag (years) | No. of Cancer Deaths | RR | 95% CI | No. of Cancer Deaths | RR | 95% CI |
| HYD-6 | 0 | 44 | 1.68 | 1.12 to 2.52 | 5 | 0.41 | 0.17 to 1.02 |
| | 10 | 42 | 1.70 | 1.13 to 2.56 | 4 | 0.36 | 0.13 to 0.98 |
| | 15 | 41 | 1.93 | 1.27 to 2.93 | 4 | 0.42 | 0.15 to 1.16 |
| HYD-24 | 0 | 36 | 1.70 | 1.11 to 2.59 | 7 | 0.66 | 0.31 to 1.44 |
| | 10 | 34 | 1.76 | 1.15 to 2.71 | 6 | 0.65 | 0.28 to 1.49 |
| | 15 | 34 | 2.10 | 1.36 to 3.25 | 5 | 0.65 | 0.26 to 1.62 |

* Estimated RRs were adjusted for age at death (continuous), pay type (two fixed binary variables), and time since hire or transfer to the Santa Susana Field Laboratory SSFL (continuous).
† International Classification of Diseases—9th Revision code 162.

**TABLE 4**
Adjusted RRs and 95% CIs* for the Effects of High and Medium Hydrazine Exposure Versus No Exposure on Cancer Mortality, by Cancer Site and Hydrazine Exposure (6- or 24-month criterion) for a 15-Year Lag

| | High Exposure | | | Medium Exposure | | |
|---|---|---|---|---|---|---|
| Cancer | No. of Cancer Deaths | RR | 95% CI | No. of Cancer Deaths | RR | 95% CI |
| Hemato- and lymphopoietic cancers† ($n$ = 41) | | | | | | |
| HYD-6 | 11 | 2.83 | 1.22 to 6.56 | 5 | 1.79 | 0.65 to 4.94 |
| HYD-24 | 6 | 1.42 | 0.54 to 3.72 | 4 | 1.32 | 0.45 to 3.90 |
| Bladder and kidney cancers‡ ($n$ = 22) | | | | | | |
| HYD-6 | 7 | 1.65 | 0.59 to 4.56 | 0 | — | — |
| HYD-24 | 6 | 1.80 | 0.63 to 5.12 | 0 | — | — |

* Estimated RRs were adjusted for age at death (continuous), pay type (two fixed binary variables), and time since hire or transfer to the SSFL (continuous).
† ICD-9 codes 200 to 208.
‡ ICD-9 codes 188 and 189.

worker-location data, we were unable to distinguish the rocket engine test personnel who primarily loaded hydrazine-fueled engines from those who used other types of fuel, such as kerosene.

Another potential source of bias in our study is the confounding by risk factors that were not included in the analyses, such as smoking and occupational exposures to other chemicals. We do not believe that confounding by smoking was appreciable in the hydrazine cohort, however, because we observed little association between smoking status and hydrazine exposure in a subset of 295 subjects (Table 7). In addition, an effect of hydrazine was not observed for smoking-related diseases other than lung, bladder, and kidney cancers (Table 5).

Nevertheless, potential confounding from other chemical exposures is more problematic. Workers assigned to test stands, for example, were probably exposed to TCE that was used in cleaning ("flushing") the rocket engines after test-firings. There is some human evidence that TCE may be a risk factor for cancers of the liver, biliary passages, kidney, and non-Hodgkin's lymphomas,[11–13] and animal studies have found links with cancers of the lung.[14] Thus TCE exposure might have confounded the estimated effects of hydrazine exposure on these cancers if workers in the same jobs were exposed to both hydrazine and TCE. Indeed, this scenario is very likely. Unfortunately, we had no way to observe the associations between hydrazine exposure and other chemical risk factors for cancer.

Another methodologic problem is that all outcome variables in this study were based on mortality, not cancer incidence. Because several cancers, such as bladder cancer, are not highly fatal, incident cases would not have been counted as outcome events if they were still alive at the end of the follow-up period. Furthermore, even if workers with non-fatal cancer had died during follow-up, the cancer would not be listed as the underlying cause of death on death certificates, or it might not be listed at all. Thus the findings reported for

**TABLE 5**

Adjusted RRs and 95% CIs for the Effects of High and Medium Hydrazine Exposure Versus No Exposure on Mortality From All Smoking-Related Cancers (except lung) and From Emphysema, by Disease and Hydrazine Exposure (6- or 24-month Criterion) for a 15-Year Lag

| Disease | High Exposure | | | Medium Exposure | | |
|---|---|---|---|---|---|---|
| | No. of Cancer Deaths | RR | 95% CI | No. of Cancer Deaths | RR | 95% CI |
| Upper aerodigestive tract cancers[†] ($n$ = 25) | | | | | | |
| HYD-6 | 3 | 0.69 | 0.19 to 2.53 | 3 | 1.69 | 0.47 to 6.06 |
| HYD-24 | 2 | 0.57 | 0.13 to 2.61 | 2 | 1.18 | 0.26 to 5.27 |
| Pancreatic cancers[‡] ($n$ = 23) | | | | | | |
| HYD-6 | 2 | 0.48 | 0.10 to 2.25 | 4 | 1.95 | 0.62 to 6.12 |
| HYD-24 | 1 | 0.32 | 0.04 to 2.51 | 4 | 2.26 | 0.72 to 7.09 |
| All smoking-related cancers (except lung)[§] ($n$ = 70) | | | | | | |
| HYD-6 | 12 | 0.94 | 0.47 to 1.86 | 7 | 1.22 | 0.54 to 2.76 |
| HYD-24 | 9 | 0.90 | 0.42 to 1.92 | 6 | 1.17 | 0.49 to 2.79 |
| Emphysema deaths ($n$ = 27)[‖] | | | | | | |
| HYD-6 | 3 | 0.54 | 0.15 to 1.93 | 4 | 2.18 | 0.72 to 6.62 |
| HYD-24 | 3 | 0.74 | 0.21 to 2.65 | 3 | 2.26 | 0.64 to 8.02 |

* Estimated rate ratios are adjusted for age at death (continuous), pay type (two fixed binary variables), and time since hire or transfer to the SSFL (continuous).

† ICD-9 codes 140 through 150 and 161.

‡ ICD-9 code 157.

§ ICD-9 codes 140 through 150 and 157, 161, 188, 189.

‖ ICD-9 code 492.

**TABLE 6**

Adjusted RRs are 95% CIs* for the Effects of High Hydrazine Exposure (HYD-6) Versus No Exposure on Lung Cancer Mortality and Hemato/Lymphopoietic Cancer Mortality, by Decade of Exposure (zero lag)

| Decade of Exposure | Lung Cancer | | Hemato/Lymphopoietic Cancer | |
|---|---|---|---|---|
| | RR | 95% CI | RR | 95% CI |
| 1950 to 1959 | 0.88 | 0.54 to 1.44 | 0.86 | 0.32 to 2.28 |
| 1960 to 1969 | 2.01 | 1.21 to 3.33 | 2.45 | 0.91 to 6.58 |
| 1970 to 1979 | 1.45 | 0.70 to 3.01 | 0.00 | 0.00 to —[†] |
| 1980 to 1989 | 0.46 | 0.06 to 3.64 | 0.89 | 0.00 to —[†] |

* Estimated RRs are adjusted for age at death (continuous), pay type (two fixed binary variables), and time since hire or transfer to the SSFL (continuous).

† Upper limits could not be estimated because of the small numbers of outcome events in the high-exposure category.

**TABLE 7**

Number (and percentage) of Current and Former Smokers Among 295 Workers Who Were Included in Three Medical Surveys at the SSFL, by Exposure Category and Period

| Hydrazine (HYD-6) Exposure Category | 1961 to 1969 | | 1983 to 1992 | | |
|---|---|---|---|---|---|
| | No. (%) of Current Smokers | Total Subjects | No. (%) of Current Smokers | No. (%) of Ex-Smokers | Total Subjects |
| High | 14 (58.3) | 24 | 8 (23.5) | 19 (55.9) | 34 |
| Medium | 14 (63.6) | 22 | 1 (11.1) | 6 (66.7) | 9 |
| Low/Unexposed | 88 (57.1) | 154 | 12 (23.0) | 24 (46.2) | 52 |
| Total | 116 (58.0) | 200 | 21 (22.1) | 49 (51.6) | 95 |

these cancers in our study might not be an accurate reflection of risk-factor effects. Empirical evidence for this problem comes from an occupational study by Demers et al,[15] who found that estimated effects on bladder and colon cancers differed for incidence and mortality data in the same population.

In conclusion, the results of this study suggest that occupational exposure to hydrazine and/or other chemicals associated with the same rocket-engine testing jobs increased the risk of death from lung cancer, and possibly other cancers, in this population of aerospace workers; however, causal inference is limited

and our results need to be replicated in other populations.

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