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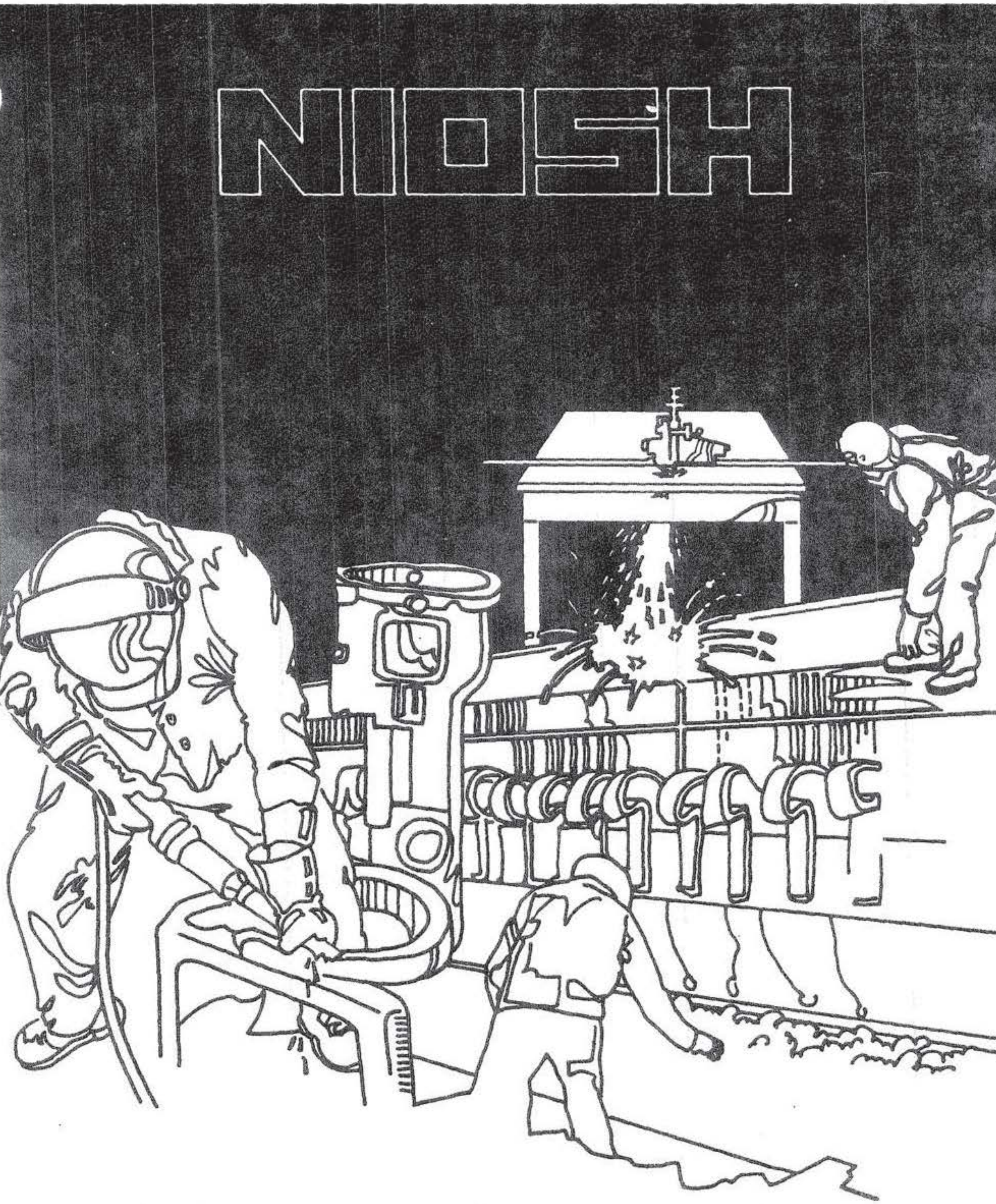

Health Hazard Evaluation Report

HETA 82-361-1437
KERR-MCGEE NUCLEAR CORPORATION
GRANTS, NEW MEXICO

PREFACE

The Hazard Evaluations and Technical Assistance Branch of NIOSH conducts field investigations of possible health hazards in the workplace. These investigations are conducted under the authority of Section 20(a)(6) of the Occupational Safety and Health Act of 1970, 29 U.S.C. 669(a)(6) which authorizes the Secretary of Health and Human Services, following a written request from any employer or authorized representative of employees, to determine whether any substance normally found in the place of employment has potentially toxic effects in such concentrations as used or found.

The Hazard Evaluations and Technical Assistance Branch also provides, upon request, medical, nursing, and industrial hygiene technical and consultative assistance (TA) to Federal, state, and local agencies; labor; industry and other groups or individuals to control occupational health hazards and to prevent related trauma and disease.

INTRODUCTION

On August 17, 1982, the National Institute for Occupational Safety and Health (NIOSH) received a request from the Oil, Chemical and Atomic Workers International Union (OCAW) and its Local 2-708 to conduct a health hazard evaluation at the Kerr-McGee Nuclear Corporation's uranium mill in Grants, New Mexico.* NIOSH was asked to evaluate health effects of exposure to uranium ore dust and to yellowcake; a concentrate of natural uranium produced by the mill. The union was particularly concerned about the nephrotoxic effects of yellowcake because a NIOSH Health Hazard Evaluation at another uranium mill had shown a correlation between increased B-2 microglobulin excretion (a marker of damage to the proximal tubules of the kidney) and duration of employment in the yellowcake area of the mill.(1)

NIOSH investigators visited the Kerr-McGee uranium mill on November 9-10, 1982. During the visit, we walked through the uranium mill from the crushing circuit through the yellowcake packaging and shipping area. We also microfilmed environmental and biological monitoring data, personnel records of workers employed during the preceding two years, as well as seniority rosters. Our goals in analyzing these data were (1) to determine the levels of exposure to yellowcake at the mill (2) to compare these levels to pertinent regulatory standards, guidelines and draft guidelines formulated by the Nuclear Regulatory Commission and (3) to characterize the population of workers exposed to yellowcake with respect to degree and duration of exposure.

BACKGROUND

The Kerr-McGee uranium mill has been operating at its present site since 1958. The primary function of uranium mills is to produce yellowcake, used commercially in the manufacture of nuclear fuel, from uranium ore. The main stages of the process involve (1) ore crushing and grinding, (2) ore leaching, (3) uranium recovery from leach solutions, and (4) drying and processing of the refined product (yellowcake). This product is a chemically complex mixture of diuranates, basic uranyl sulfate, and hydrated oxides.(2) The Kerr-McGee Nuclear Corporation has published a detailed description of the processes used at their facility.(3)

The Kerr-McGee Corporation is licensed to mill uranium by the Radiation Protection Bureau of New Mexico under authority delegated by the Nuclear Regulatory Commission (NRC). Both the Mine Safety and Health Administration (MSHA) and the NRC have regulatory jurisdiction over uranium mills. A Memorandum of Understanding signed by NRC and MSHA states that each agency will coordinate the development of standards with the other agency.

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* Effective October, 1983 Kerr-McGee realigned its uranium operations into two subsidiaries. The uranium mining and milling operations are now operated by the Quivira Mining Company.

MATERIALS AND METHODS

1. Review of the literature

a. Potential hazards by area of the milling process

Biological solubility influences the toxicity of a given uranium compound. Dusts containing only poorly soluble uranium compounds are cleared slowly from the lungs and pose a potential internal radiation hazard. More soluble compounds are absorbed rapidly from the lungs, decreasing the radiation hazard, but introducing the potential for renal toxicity.[2,4]

Before leaching (that is, in the ore crushing and grinding circuits) the uranium in ore dusts consists mostly of uranium oxides with a relatively small fraction of the more soluble uranium compounds. In addition to the radiologic hazards associated with inhalation of uranium dusts, significant concentrations in air of radon and its daughters may occur near ore storage bins and crushing and grinding circuits. The process of leaching converts much of the uranium to a complex mixture of sodium and ammonium salts. These salts are absorbed rapidly from the lungs, decreasing the radiation hazard, but introducing the potential for renal toxicity.[2]

Several recent studies have examined the solubility of uranium aerosols at various stages of the commercial milling process.[5,7] These studies have shown that uranium dusts encountered in industrial (mill) settings are seldom wholly soluble or insoluble. In reality, they are complex mixtures of compounds of varying solubility. For example, Eidson [5] found that the content of ammonium diuranate in yellowcake samples from four mills ranged from 12-82%, depending upon the temperature at which the yellowcake was dried. It is appropriate to regard the yellowcake drying and packaging area as the mill process involving the highest exposures to soluble uranium, and the crushing area as that primarily involving exposure to insoluble uranium.

b. Radiologic hazards

In mills, radiation is emitted primarily by radioisotopes in the U-238 decay series (Figure 1). The two exposure categories of particular concern are the daughter isotopes of radon-222, and the long half-life constituents of uranium ore dust (uranium-238, uranium-234, thorium-230, radium-226, and lead-210).

Radon daughters (polonium-218, lead-214, bismuth-214, and polonium-214) have rapid rates of decay and short half-lives. Polonium-218 and Polonium-214 emit alpha particles which penetrate poorly but produce intense local tissue damage when deposited internally. Since the parent compound, radon-222, is a gas, and since the daughter isotopes are small, they attach readily to respirable particles of dust. The hazard exists because inhaled dust deposits on the epithelial lining of the respiratory tract, allowing intense local irradiation.(8) Epidemiological studies have shown an excess of lung cancer in miners.(9,11) The occurrence of lung cancer relates closely to the levels of exposure to radon daughters. Because of these studies, the current standard for both mines and mills limits occupational exposures to 4 working level months (WLM)* over a 12 month period.(12) Unlike uranium mines, mills and other surface facilities have not been found to trap high concentrations of radon and radon daughters. While the release of radon complicates the problem of safe disposal of uranium mill tailings, occupational exposures of millers are lower than those of miners. Similarly, two published U.S. mortality studies of mill workers did not show an excess of lung cancer.(13,14)

Other potential sources of radiation are the long-lived isotopes contained within poorly soluble uranium dusts. Exposure to relatively insoluble compounds with a biologic half-life of greater than 50 days occurs in the crushing and grinding areas of uranium mills. Some data from toxicologic studies suggest that these less soluble uranium compounds may accumulate in the tracheobronchial lymph nodes. For example, in animals inhaling uranium dioxide dust at about 25 times the occupational limit for a prolonged period (5 mg/m^3, 6 hr/day, 5 days/week for up to 5 years) the concentration of uranium in tracheobronchial lymph nodes at autopsy was approximately 20 times greater than in lung tissue.(15) The lung concentrations in these studies were on the order of 3000 ug U/gm wet lung tissue. Autopsy data, available on a few humans in whom lung tissue concentrations wereless than 2 ug U/gm wet tissue, showed no evidence suggesting proportionally higher concentration in tracheobronchial lymph nodes.(16) Organ deposition of the uranium dust is hazardous because of prolonged internal alpha irradiation. Of particular concern is the epidemiologic finding by Archer,(13) who observed a nearly four-fold excess of deaths from lymphatic malignancies in a small cohort of uranium millers (4 deaths observed versus 1.02 expected). All of the cases worked in or near the crushing area of the mill where exposures to insoluble dusts were highest. However, a subsequent mortality study of a larger number of uranium millers found only a slight excess of lymphatic malignancies (excluding leukemia).(14) It remains possible that at high air concentrations of uranium dust, where much of the exposure is respirable, substantial chest deposition may occur and be associated with an increased risk of lymphatic malignancy.

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* A working level is defined as any concentration of short-lived radon-222 daughters, in one liter of air, that results in emission of 1.3 x 10^5 million electron volts (MeV) of alpha particle energy. A WLM is defined as the exposure to a concentration of one working level for a period of 170 hours.

In mills, which process unenriched uranium, external radiation is of less concern than is internal. This is because the predominant emissions of natural uranium are alpha and beta particles, which penetrate the skin poorly. Alpha particles produce intense local irradiation when deposited internally. In contrast, gamma emissions, which do penetrate the body, are emitted in relatively small amounts.

c. Chemical Toxicity

The chemical toxicity of uranium has been studied extensively. During the 1940's the Manhattan project commissioned one of the most comprehensive toxicologic studies in history, the results of which fill over 2300 pages in four volumes.(17) That research, conducted on 12 different uranium compounds, identified the kidney as the critical organ for soluble uranium exposure, and defined the dose which would induce acute renal injury in both animals and man.

Subsequent medical research has confirmed the renal toxicity of soluble uranium.(18-23) Compounds such as uranyl nitrate and uranium acetate are used experimentally to create a model of acute renal failure. Injury following acute exposure affects predominantly the proximal tubules. Experimentally, abnormal excretion of glucose, albumin, and intracellular enzymes in the urine progresses to either death or clinical recovery. The kidneys of exposed animals who recover are more resistant to the acute effects of continued exposure.

Although the present occupational standard for soluble natural uranium (200 micrograms per cubic meter, or $1x10^{-10}$ microcuries per milliliter of air) is based upon the kidney as the critical organ, the standard is actually four times higher than the estimate derived from the original toxicological data.(4) In part the present standard was selected because of the absence of reported kidney disease in occupationally exposed workers, although this has never been adequately investigated.(23)

Medical surveillance of renal effects in occupational groups has been less thorough than the original toxicological research. A two year follow-up of an unspecified number of laboratory workers was reported by Howland.(24) The exposures were short, however, and since the subjects were scientists, may be considered atypical of most of the industry. Assessment of renal function was limited to measuring urine sugar and albumin, and examining urine sediment. Katz (25) measured urinary excretion of the tubular enzyme catalase among workers of two chemical plants processing uranium compounds. Although the average urinary catalase level was higher for these workers than for nonexposed controls, suggesting kidney tubular injury, the results were regarded as ambiguous because of differences in urine concentration. Case reports of transient albuminuria following occupational overexposures have been described from the United States[24] and England.[23] However, the transience of the albuminuria has been wrongly interpreted as evidence for the absence of chronic renal injury. Albuminuria is a marker of glomerular dysfunction, which can be produced by acute overexposures. Uranium is also known to damage the endothelium of the renal proximal tubules at exposure levels below those required to cause glomerular injury. Tubular injury is characteristically not detectable by increase in urine albumin, but rather, is reflected by an increase in exretion of low molecular weight proteins, such as beta-2-microglobulin. No systematic medical or epidemiological studies of renal function in uranium workers have been published, nor have sensitive markers of proximal renal tubular damage been used , such as urinary beta-2-microglobulin excretion or urinary protein electrophoresis. The existing mortality studies do not resolve the issue regarding nephrotoxicity in uranium mill workers. Archer et. al.[13] did not examine renal deaths as a distinct category. The study by Scott [26] did not take into account latency in its followup of 4,500 employees at a gaseous diffusion plant. Furthermore, the Scott study grouped together deaths from all causes in its analysis, and failed to separate the data according to exposure group. A more adequate paper by Polednak[27] examined mortality among 18,869 uranium enrichment workers in Oak Ridge, Tennessee. The study sheds little light on the chronic renal effects of prolonged exposure to soluble uranium, however, since the plant was only in operation for four years, and since the exposures were predominantly to insoluble or to moderately soluble uranium.

Waxweiler et al recently analyzed causes of mortality among a previously studied cohort of U.S. underground miners.[11] A 3-fold increase in deaths was observed from chronic and unspecified nephritis and renal sclerosis (ICDA 591-594, 9 observed, 3 expected). A slight excess of deaths from renal disease was also found in a cohort study of U.S. uranium millers (6 observed, 3.6 expected).[14] The authors did not view this excess as clearly occupationally related, because all of the cases were short term workers and four died within 8 years of their initial employment in a uranium mill. While neither of these studies is sufficient to establish that prolonged exposure to uranium causes renal disease, the findings underscore concern about the chronic renal effects of uranium exposure. Because renal disease is not always coded on the death certificate, even if present, clarification of this issue requires more sensitive techniques than mortality studies.

NIOSH conducted a Health Hazard Evaluation at the Cotter Uranium Mill, Canon City, Colorado in 1980-1981.[1] NIOSH investigators examined company industrial hygiene and bioassay records from 1975 to 1981. Both air samples and urine samples 1975 to 1978, prior to the construction of a new mill, indicated that exposures in the yellowcake drying and packaging areas frequently exceeded occupational standards. Both area air uranium monitoring and urine bioassay data in the new Cotter mill indicated that exposures since 1979 had generally been within the standard.

Because of the known nephrotoxicity of soluble uranium compounds, NIOSH assessed the kidney function of 39 uranium workers as compared with 36 age, race and sex matched local controls from a nearby cement production facility. The assessment of kidney function demonstrated statistically significantly increased urinary excretion of amino acids and of the small protein beta-2-microglobulin in the urine of uranium workers compared to local controls. The excretion of beta-2-microglobulin was significantly higher among the exposed when expressed in a variety of ways, including clearance and clearance relative to creatinine clearance. These findings were suggestive of renal (kidney) tubular injury, and are consistent with the known toxic effect of soluble uranium on the kidney tubules. Although the level of excretion was within some published population normals, the amount excreted by the uranium workers clearly exceeded that of local controls. Furthermore, within the uranium group, the level of excretion was significantly correlated with years of work in the yellowcake area of the old mill. No clear evidence of impaired glomerular function was evident in the uranium workers, compared to controls.

Based on these findings, NIOSH suggested that additional research be done to determine whether sensitive tests of kidney tubular function should be included in medical surveillance programs. In addition, NIOSH recommended that the relationship between chronic renal disease and occupational exposure to uranium be defined by epidemiologic studies.

d. Emerging concerns

Both uranium miners and millers have been observed to have an increased risk of death from non-malignant respiratory disease. Waxweiler et al, in a cohort mortality study of uranium miners, observed 83 deaths from "other non-malignant respiratory diseases" with only 16.6 such deaths expected, indicating that uranium miners had approximately a five-fold risk of death from this cause compared to the U.S. population.[11] Excess deaths from "other non-malignant respiratory disease" have also been reported in a mortality study of 2002 U.S. uranium mill workers.[14] In this study, mill workers were approximately 2.5 times more likely to die of these causes than would be expected by U.S. death rates, based on 39 observed and 15.6 expected deaths. Neither of these studies reported on the exact diagnoses contributing to this excess, but deaths from silicosis would be coded in this category. Both uranium miners and millers have potential exposure to silica; bulk samples of uranium ore were found to contain 42-95% alpha quartz in one study.[28] A large survey conducted by the U.S. Public Health Service in 1958-1961 found a 27.5% prevalence of silicosis among uranium miners with 20-29 years exposure, and a 3.2% prevalence among workers with 10-19 years exposure. Environmental silica levels in uranium mines were not reported.[28] A recent prevalence survey of respiratory abnormalities among 192 uranium miners who had accumulated at least 10 years underground experience by 1970 showed small but statistically significant effects of uranium mining on two measurements of respiratory function.[29] 12 of 143 survey participants

(8.4%) whose chest x-ray films were obtained had at least category 1/0 pneumoconiosis, which was equally prevalent in those with and without work experience in other types of mines. The opacities seen in the chest x-rays were of the nodular type commonly seen in silicosis.

It is possible that uranium ore dust and radon daughter exposures contribute to the excess mortality from non-malignant respiratory disease among uranium miners and millers. A recent morbidity study of workers in a uranium processing plant, in which silica exposure was minimal or absent, found increased non-malignant respiratory disease among workers with higher cumulative uranium exposure compared to lower.(30) No statistically significant association was demonstrated for the non-radioactive chemical exposures.(30)

Pulmonary lesions have been induced in experimental animals by administration of silica-containing uranium ore dust in conjunction with radon and radon daughters. In dogs, the lesions consisted of pulmonary hyalinosis, granulomatous reactions, vesicular emphysema and pulmonary fibrosis.(31) In Syrian hamsters, however, pulmonary emphysema was induced not only by inhalation of silica containing ore with and without radon daughters, but also by treatment with radon daughters alone.(32) In another experiment, monkeys exposed to UO_2 dust (which did not contain silica) developed patchy, hyaline pulmonary fibrosis.(15)

The increased risk of death from non-malignant respiratory disease found in mortality studies of uranium miners and millers reflects historical rather than recent exposures. The extent to which reduced exposures to silica, uranium ore dust and radon daughters among current mill workers has reduced their potential risk of developing non-malignant respiratory disease is unknown. However, given that silica, radon daughters and uranium ore dust have all been shown independently to induce pulmonary lesions in animals, there is some question as to whether separate occupational health standards would be protective when workers are exposed to all three substances concomitantly. Further research is necessary to clarify this issue.

2. Data collected and methods of analysis.

a. Urine bioassay data

The urine bioassay program began at Kerr-McGee in December, 1979. Operators in the yellowcake area submit routine samples every two weeks; maintenance personnel are required to submit a sample if they have worked for 4 hours or more in the yellowcake area in a calendar month. Additional samples may be taken if either bioassay or environmental monitoring results indicate excessive exposure. Employees are asked to submit morning urine samples after being off work at least two days. Four quality control samples (one uncontaminated plus three samples spiked at 15 ug/L, 30 ug/L and 45 ug/L) are submitted with each batch of routine samples. The samples are analyzed by an outside laboratory. At the time of the NIOSH visit, Kerr-McGee used a lower action level of 25 ug/L and an upper action level of 50 ug/L in interpreting the results of urine uranium bioassays.(33) However, an employee was placed on work restriction only if a confirming sample also exceeded 50 ug/L. In January, 1984, Kerr-McGee changed its urine uranium bioassay policy to allow higher exposures before work restriction is triggered. Currently, Kerr-McGee places an employee on work restriction only if two consecutive biweekly or special samples exceed 130 ug/L, or four consecutive biweekly samples exceed 30 ug/L.(34)

b. Air monitoring in the yellowcake area

Area air sampling data were obtained for the period 1970 to 1982. Currently, the yellowcake area is sampled weekly at 32 locations; in the early 1970's the area was sampled at least monthly at the same locations. Prior to May, 1979, a fluorometric method was used to analyze air samples; after that time a radiometric analysis was used. The results of the fluorometric analyses are expressed as u Ci/ml or ug/M^3, while the results of the radiometric analysis are expressed as disintegrations per minute per liter (DPM/L). In March of 1979, the health physics staff at Kerr-McGee used both methods to sample the precipitation and packaging areas and found that the results "were fairly close - for most samples they are within plus or minus 0.07 MPC of each other". The radiometric MPC°s were, however, almost always lower than the fluorometric results. Since 1972, a time weighted average has been calculated for each individual for a sampling period by weighting the uranium concentration at different job sites by the time the operator spends at the site. Time studies are conducted annually. NIOSH did not obtain a copy of the technical methods used for the fluorometric analysis; methods for the radiometric analysis are described in Kerr-McGee's "Environmental, Health Physics and Industrial Hygiene Standards and Technical Manual".(35) It appears that with both methods, it was standard practice to run sampling pumps for 5 minutes.

Our main purpose in analyzing the air data was to determine whether there had been a marked change in uranium air concentrations in the yellowcake area over the period 1970-1982. In order to do this, we calculated the arithmetic mean of air levels in the "precipitation" and "drier" areas in 1970 and 1975 and the ratio (x 100) of the mean annual air level to the current maximum permissible concentration (MPC). Since the 1980 samples were in units of DPM/L and could not be directly compared to the 1970 and 1975 values, we calculated the mean percent of the current MPC for air concentrations in the "precipitation" and "drier" areas for January, April, July and October of that year.

c.

There were three sources of data on personnel assignments. The urine bioassay data contained very specific work assignments for individuals sampled by two week periods 1979-82. Personnel records of workers employed during 1980-82 contained complete job histories since the worker was hired, but the job titles used were considerably more general than the bioassay assignments. In the personnel records, operators in the yellowcake area do not have a specific designation, but are included in the "SX-operator" category (SX is an abbreviation for solvent extraction). Approximately 30% of individuals with jobs coded in the SX category actually work in the yellowcake area. Union seniority lists, which were microfilmed for the period 1970-1982, also included yellowcake workers in the SX-operator category. It was therefore impossible to use the personnel and union seniority records to estimate, for each individual, the number of years worked in the yellowcake area. Instead, we tried to identify individuals with relatively high or long-term exposure by examining the work histories of individuals whose mean urine uranium equalled or exceeded 15 ug/L and individuals who had 25 or more samples taken in the urine bioassay program. As the urine bioassay program concentrates on workers in the yellowcake area, this would presumably identify individuals with long-term assignments in this area in the past several years. We also reviewed the union seniority lists and examined the duration of employment in the SX area for individuals who worked in this area 1970-75.

EVALUATION CRITERIA

Federal standards for protection against radiation are contained in 10 CFR Part 20.(12) These standards stipulate a Maximum Permissable Concentration (MPC) for natural uranium in air of 1.0×10^{-10} microcuries per milliliter of air (uCi/ml), 200 ug uranium per $meter^3$ of air or 0.222 disintegrations per minute per liter of air gross alpha activity average for a 40 hour workweek. New Mexico's licensing conditions are consistent with federal regulations pertaining to external radiation dose and air and water concentrations promulgated in 10 CFR Part 20.

In addition to federal standards, the Nuclear Regulatory Commission issues regulatory guidelines which may or may not be adopted in State licensing conditions. Draft regulatory guides which have been used in the preparation of this report are:

1. Health Physics Surveys in Uranium Mills (issued August 1980).(2)
2. Information Relevant to Ensuring that Radiation Exposures at Uranium Mills will be as low as is Reasonably Achievable (issued August 1980).(36)
3. Bioassay at Uranium Mills (issued 1978).(37)

There are no regulatory standards pertaining to bioassay results at uranium mills. However, guidelines for obtaining and evaluating samples were provided in Draft Regulatory Guide 8.22, Bioassay at Uranium Mills.[37] This guide stipulates that workers in the yellowcake concentrate areas of mills should have urine collected for bioassay every two weeks and that samples should be collected at least 48 hours, but not more than 96 hours, after the most recent occupancy of the yellowcake area. The regulatory guide recommends a lower action level of 15 micrograms per liter (ug/L) and an upper action level of 30 ug/L to protect against chemical toxicity to the kidneys. The basic formula for calculation of the action level is:

[3ug U/gm kidney x 300 gms. x (.693/15) days] /1.4 liters/day

where 3 ugU/gm = the experimental nephrotoxic limit for human kidney dose[3,38]

300 grams = the average human kidney weight[38]
(.693/15 days) = the first order elimination constant, where 15 days represents the biological half life determined experimentally[38]
1.4 liters/day = the average human urine excretion rate.[38]

Actions recommended by the draft NRC regulatory guide when the urine uranium concentration is 15 to 30 ug/L include repeat urinalysis, determination of why air samples did not warn of excessive concentrations of airborne uranium, identification of the cause of airborne uranium and initiation of control measures. Bioassay should be performed for other potentially exposed workers, and work assignment limitations should be considered to ensure the worker does not exceed a urine uranium concentration of 30 ug/L. When the urine uranium concentration exceeds 30 ug/L, operations should be continued only if it is virtually certain that no worker will exceed a urinary uranium concentration of 30 ug/L, and work restrictions should be established for affected employees.[37]

The current Kerr-McGee bioassay policy is at variance with the NRC guidelines in several respects. The company policy disregards the recommendations to curtail worker exposure at the lower (15 ug/L) and upper (30 ug/L) action levels recommended by the NRC. The NRC guidelines state that at the urinary uranium concentrations now used to trigger work restrictions for Kerr-McGee employees, 30 ug/L for four consecutive specimens or greater than 130 ug/L for any specimen*, there is a "possibility of renal damage to the worker." At these levels, the NRC recommends having an additional urine sample tested for albuminuria.

* The NRC guide stipulates 130 ug/L for any specimen, while the Kerr-McGee bioassay policy stipulates two consecutive biweekly or special samples.

RESULTS

1. Urine uranium bioassay data

a. Urine uranium standards

Figure 2 summarizes the results obtained from 0, 15, 30 and 45 ug/L standards analyzed along with urine samples. There is fairly high variability in the measurements which could result from problems in the method of spiking samples or problems in the analytical method. There was also a tendency for the 15 ug/L standards to give readings higher than the true value, while the 30 ug/L and 45 ug/L standards tended to have results lower than the true value. If the results on standard samples reflect the degree and direction of laboratory measurement error, rather than errors in spiking, we would infer that results of bioassays in the lower end of the range might be overestimated and those in the upper end of the range might be underestimated. In addition, caution must be used in interpreting the results of a single sample because the measurement error appears to be so large.

b. Urine uranium bioassay results - overall

1181 samples were taken from 156 workers (including control samples) during the period December, 1979 to October, 1982. The number of samples taken per individual ranged from 1 to 51, with a mean of 7.2 samples and a median of 3 samples.

Table 1 shows the overall distribution of results (excluding control samples). 23.9% of samples exceeded the NRC lower action level of 15 ug/L and 9.3% of samples exceeded the NRC upper action level of 30 ug/L. Table 2 compares the urine uranium bioassay results at the Kerr-McGee uranium mill with those at the Cotter uranium mill previously studied by NIOSH. The urine uranium results at Kerr-McGee from 1979-1982 were in the same general range as those at Cotter 1976-1981. (The sampling results at Cotter were considerably higher during 1975.)

c. Urine uranium levels, by person

Table 3 shows the distribution of individuals by mean and median uranium values. 45.5% of individuals had median urine uraniums of less than 5 ug/L; 42.9% had medians of 5-14.99 ug/L and 10.9% had medians of 15-29.99 ug/L. No individual sampled repeatedly had a median of 30 ug/L or greater. In general, urine uranium levels of individuals sampled repeatedly were highly variable, probably reflecting changing job and work assignments and varying levels of air contamination in work areas over time.

d. Urine uranium levels, by job

Table 4 shows the distribution of urine uranium levels by job (jobs were grouped as shown in Appendix I). Workers in "yellowcake precipitation" had the highest mean urine uranium level (20.5 ug/L), followed by workers in yellowcake drier (19.0 ug/L) and yellowcake "general" (11.4 ug/L) areas of the mill. Supervisors generally had low levels of uranium in their urine (mean of 4.1 ug/L).

2. Air monitoring in the yellowcake area

Table 5 summarizes air monitoring results obtained in the drier and precipitation areas of the Kerr-McGee uranium mill 1970, 1975, and 1980. The results were divided by the current MPC (in appropriate units) in order to provide a basis of comparison over time. The results do not indicate that there has been a trend toward either lower or higher average exposures in these areas 1970-1982. At the Cotter uranium mill the means from 1975-1981 ranged from 7.4% to 227% of the MPC.

3. Number of workers in the yellowcake area and their duration of employment

At the time NIOSH investigators visited the Kerr-McGee uranium mill, 65 hourly workers and 40 salaried workers were involved in mill production jobs. In addition, there were 44 hourly and 12 salaried workers assigned to the maintenance and electrical departments of the mill. The yellowcake area was operating on 3 shifts, 4 days a week, with one person assigned to operate the drier and one person assigned to operate the precipitator in the yellowcake area. In addition to workers assigned to full-time jobs in the yellowcake area, supervisory personnel and maintenance and electrical personnel may walk through or work in the area. Currently, maintenance and electrical workers performing non-routine operations in the yellowcake area are required to wear respiratory protection.

In the hazard evaluation conducted by NIOSH at the Cotter uranium mill, there was a correlation of B-2-microglobulin excretion (a marker of renal tubule damage) and years of exposure to yellowcake (Figure 3). Workers with less than 8 years of employment in the yellowcake area showed little or no effect. Although a medical evaluation of kidney function at the Kerr-McGee mill would not necessarily include only long term yellowcake workers, this is the group in which we are most likely to observe an effect. We therefore tried to identify the numbers of workers with recent and long term exposure to yellowcake from available records (as described under "methods"). The results are shown in Table 6. Eleven workers were identified from bioassay records as having worked more than one year in the yellowcake area within the past 3 years; however, none had worked in yellowcake over eight years. There were, in addition, several supervisors who had over 10 years employment in the SX area prior to their transfer to supervisory jobs, but we do not know if they actually worked in yellowcake.

According to union seniority rosters, as of 1975, 4 individuals had worked in the SX area for over 5 years; as of 78 there were 3; as of 80 there were 2 and as of 82 there were 5 (4 of whom started in 1977). We conclude from this that there may be a few workers with long term exposure to yellowcake who were no longer working at the Kerr-McGee mill when the bioassay program began in 1979. Most workers assigned to full time jobs in the yellowcake area have worked there for less than 5 years. This obviously limits the ability of a medical study to detect renal changes associated with longer-term exposure.

DISCUSSION AND CONCLUSIONS

1. Evaluation of exposures

a. NIOSH investigators based our evaluation of exposures primarily on urine bioassay results obtained from 1979 to 1982. We chose to examine urine uranium levels because they are a direct measure of the amount of uranium that has been absorbed by the body and excreted by the kidney, and are therefore a more direct correlate of potential renal damage than are air uranium measurements. The majority of Kerr-McGee workers sampled in the urine bioassay program had mean and median urine uranium values below the NRC lower action level of 15 ug/L, but workers with jobs in the yellowcake drier and precipitation areas had mean urine uraniums above 15 ug/L. As shown in Table 4, 16.4 % of samples from workers in the yellowcake drier area and 19.0% of samples in the yellowcake precipitation area exceeded 30 ug/L, the level at which the NRC guidelines require work restrictions for affected employees, as well as other actions to prevent continued worker exposure. The NRC action levels are set to maintain uranium concentrations in the kidney below concentrations that produce renal damage in animal studies.

b. NIOSH's analysis of extensive air monitoring data obtained from Kerr-McGee was primarily directed at determining whether there was evidence for a substantial increase or decrease in air uranium measurements from 1970-1982. We found that the ratio of the mean divided by the current MPC (x 100) was similar in 1970, 1975 and 1980 for samples taken in the drier and precipitation areas. Although these annual means were approximately one-third of the MPC, we did not fully evaluate the air monitoring data in terms of specific sampling sites or weekly and quarterly exposures, and cannot draw further conclusions about worker exposure based on these data.

2. Evaluation of Kerr-McGee bioassay policy.

We question the rationale for the latest revision in the Kerr-McGee bioassay guidelines. The current policy places employees on work restriction only when urinary uranium concentrations exceed levels at which the NRC suggests that renal damage may occur (30 ug/L for four samples or 130 ug/L for two samples). In so doing, it ignores the NRC's recommended upper action level of 30 ug/L for a single sample. Since 90% of the urine uranium concentrations measured among Kerr-McGee workers were below 30 ug/L, it would seem feasible for Kerr-McGee to adopt the NRC-recommended action level (30 ug/L). Such a policy would ensure prompt identification of excessive exposures not otherwise detected by the monitoring program, and would better protect individual workers against renal damage. In view of the ALARA, or "as low as reasonably achievable concept," there appears to be no justification for making the evaluation criteria more lenient.

3. Feasibility and usefulness of a medical study.

a. Rationale for conducting a study

As was discussed in the literature review, there is a need for additional research to define the relationship between chronic renal disease and occupational exposure to uranium. We therefore have considered the feasibility of examining Kerr-McGee workers to determine whether there are detectable changes in proximal renal tubular function associated with degree or duration of exposure to soluble uranium. In doing so, our main source of data regarding predicted effect is the Cotter uranium mill hazard evaluation.

b. Comparability of exposures to uranium at Kerr-McGee and Cotter

From 1976 on, urine uranium measurements at Cotter were roughly in the same range as those at Kerr-McGee. In 1975, however, urine uranium levels at Cotter were considerably higher. It is difficult to compare air measurements from Kerr-McGee and Cotter because we did not analyze them in great detail and because there was such high variability in mean air levels at Cotter from 1975-1981 (from 7.4% to 227% of the MPC). Based on the urine values, we would conclude that exposures at Kerr-McGee and Cotter were reasonably comparable.

c. Comparability of duration of exposure at Kerr-McGee and Cotter

As can be seen in Figure 3, 14 of 39 individuals included in the Cotter medical study had 8 or more years of full- or part-time exposure to yellowcake. At Kerr-McGee, as shown in Table 7, we were unable to identify any individual with 8 or more years of work in the yellowcake area of the mill. At Cotter, workers with fewer than 8 years of exposure to yellowcake showed little or no correlation between clearance of beta-2-microglobulin relative to creatinine and duration of exposure. Because the effect observed at Cotter appeared to be associated with long-term, cumulative exposures, we consider it highly unlikely that a medical study utilizing B-2 microglobulin clearance relative to creatinine as a marker of proximal renal tubular damage would show a significant correlation with duration of exposure or a significant difference between Kerr-McGee workers and a non-exposed population. A study showing no evidence for renal toxicity in this population would not be truly reassuring because it may be that such damage would occur if exposure continued for 10 or more years.

d. How the potential renal toxicity associated with long term exposure to yellowcake might be studied

There are several ways in which to assess whether long term exposure to yellowcake causes renal damage. One type of study would be a mortality study in which causes of death among workers employed in uranium mills as much as 20 or 30 years ago would be examined. The difficulties in this type of study would be that historical records might not contain information about who worked in yellowcake, as opposed to other areas of the mill, and that renal disease, even if present, often is not recorded on the death certificate. A more intensive study could be done by identifying the same group of workers, but in addition to collecting the death certificates, contacting workers who are still living to determine if they have ever had renal disease as well as interviewing next-of-kin and obtaining medical records of deceased individuals. This type of study would be very expensive and difficult to conduct if a large group of workers were followed (as would be necessary for a good study). The third option would be to examine a group of workers with ten or more years exposure to yellowcake who are still employed in the industry. In this type of study, which is similar to the medical study conducted at Cotter, we could use techniques that are sensitive to the type of renal damage induced by uranium to determine whether kidney function has been affected by the exposure. Kidney function in long term yellowcake workers could be compared to kidney function among individuals with shorter exposure and individuals who reside in the same area but have not worked in a uranium mill or mine. In our opinion, this last type of study is the next logical step in answering questions about the renal toxicity of uranium.

Prior to initiating such studies, NIOSH investigators consider how many people should be included to have a reasonable chance of detecting an effect. We do this by calculating statistical power, which is the probability that a study with a given number of participants could detect the specified effect if it truly existed in the study population. Although a number of different statistical analyses could be done, the simplest one is the test of whether clearance of B-2 microglobulin (relative to creatinine clearance) differs in the yellowcake exposed and non-exposed populations.

In the Cotter study, NIOSH found that the mean of this relative clearance was 1.5 times greater among exposed workers than controls. If we selected a population composed exclusively of long term workers, we would like to be able to detect a difference at least this large between the long term workers and unexposed controls. If the mean relative clearance (ml/min beta-2-microglobulin divided by ml/min creatinine) among control workers is 2.62×10^{-4} (as it was at Cotter) and the mean among exposed workers was 3.95×10^{-4} (as it was at Cotter) we would like to select a sample size such that the confidence intervals of the "control" means (Mcont) and the "exposed" means (Mexp) do not overlap.[39] The null hypothesis in this case is that the "control" mean and "exposed" means are equal (that is Mexp - Mcont = 0) and the alternative hypothesis is that (Nexp-Mcont) = 1.33×10^{-4}. We therefore wish to select the number of exposed (Mexp) and control (Ncont) so that:

$$t_{\alpha/2}\, s \sqrt{\frac{1}{N_{exp}} + \frac{1}{N_{cont}}} \leq 1.33 \times 10^{-4}$$

$$s \approx 0.00024$$

$$t_{\alpha/2} \approx 2$$

$$N_{exp} = N_{cont}$$

$$\text{So: } 2(.00024)\sqrt{\frac{2}{N_{exp}}} \leq 1.33 \times 10^{-4}$$

The minimal value of Nexp for which this is true is 27. Thus, in planning a study comparing the renal function of long term yellowcake workers and unexposed individuals, the minimal number of people we should study is 27 exposed and 27 controls.

RECOMMENDATIONS

1. Kerr-McGee should use the most recently proposed NRC regulatory guideline for urine bioassay, which specifies 15 ug/L as the lower action level and 30 ug/L as the upper action level.

2. Worker exposures to uranium dust in the yellowcake drier and precipitation areas should be reduced so that few urine samples from workers in these areas exceed 15 ug/L. When urine uranium concentrations do exceed 15 ug/L, actions specified in the NRC regulatory guideline should be taken. These actions include identification of the cause of airborne uranium and initiation of

control measures. When the urine uranium concentration exceeds 30 ug/L, operations should be continued only if it is virtually certain that no worker will exceed a urinary uranium concentration of 30 ug/L, and work restrictions should be established for affected employees.[35]

3. Although a medical study of the effects of long term exposure to yellowcake is not feasible at Kerr-McGee, because of the relatively short duration of exposure of most workers to yellowcake, such studies would be valuable in assessing the health risks of uranium. In order to achieve adequate sample size for such a study, it may be necessary to examine workers with long-term exposure to yellowcake from several mills or other parts of the uranium industry.

REFERENCES

1. HETA 81-055-954. MTA 80-107. September, 1981. Cotter Corporation, Canon City, Colorado.

2. United States Nuclear Regulatory Commission, Office of Standards Development: Health physics surveys in uranium mills, Draft regulatory guide and value/impact statement, August, 1980.

3. Uranium Mining and Processing, Kerr-McGee Corporation.

4. Spoor NL, Hursh JB. Protection Criteria. In: Hodge HC, Stannard JN, Hursh JB, eds. Uranium, Plutonium, and Transplutonic Elements. New York, Heidelber, Berlin: Springer-Verlag, 1973: 241-270.

5. Eidson AF. Biological characterization of radiation exposure and dose estimates for inhaled uranium milling effluents. NUREG/CR-1669, LMF, 1980.

6. Kalwarf DR. Solubility classification of airbourne products from uranium ores and tailings piles. NUREG/CR-0530, PNL-2870, 1979.

7. Dennis NA, Blauer HM, Kent JE. Dissolution fractions and half-times of single source yellowcake in simulated lung fluids, Health Physics 42: 469-477 (1982).

8. Cohen BL. Radon: characteristics, natural occurrence, technological enhancement, and health effects, (1979) Prog in Nuclear Energy, Vol 4. 1-245.

9. USDHEW, PHS, National Institute for Occupational Safety and Health (NIOSH), and National Institute of Environmental Health Sciences, Joint Monograph No. 1 (1971) Radon daughter exposure and respiratory cancer, quantitative and temporal aspects.

10. BEIR (National Academy of Sciences Committee on Biological Effects of Ionizing Radiation) (1972) Report, the effects on populations of exposures to low levels of ionizing radiation, Washington DC.

11. Waxweiler R, Roscoe R, Archer V, Thun M, Wagoner J, Lundin F. Mortality follow-up through 1977 of the white underground uranium miners cohort examined by the United States Public Health Service. In Gomex M ed. Radiations Hazards in mining: Control, measurement, and medical aspects. N.Y. Soc. of Mining Engineers, 1981: 823-830.

12. United States Nuclear Regulatory Commission, Rules and Regulations: Title 10, Chapter 1, Code of Federal Regulations-Energy. Part 20 Standards for protection against radiation. pp. 20.1-20.21, June 1979.

13. Archer VE, Wagoner JK, Lundin FE. Cancer mortality among uranium mill workers. JOM 1973; 15,1, 11-14.

14. Waxweiler, RJ, Archer, VE, Roscoe, RJ, Watanabe, A and Thun, MJ. Mortality patterns among a retrospective cohort of uranium mill workers. In: Epidemiology Applied to Health Physics, Proceedings Sixteenth Midyear Topical Meeting, Health Physics Society, Albuquerque, New Mexico, January 9-13, 1983.

15. Leach LL, Maynard EA, Hodge HC, Scott JK, Yuile CL, Sylvester GE. A five-year inhalation study with natural uranium dioxide (UO_2) dust-1. Retention and biological effect in the monkey, dog, and rat. Health Physics 1970; 18, 599-612.

16. Donoghue JK, Dyson ED, Hislop JS, Leach AM, Spoor M. Human exposure to natural uranium, a case history and analytical results from some post-mortem tissues. Br. J Ind. Med. 1972; 29, 81-89.

17. Voeglin C, Hodge HC, Eds. Pharmacology and Toxicology of Uranium Compounds, Vol. 1-4, McGraw Hill, 1949.

18. Bowman FJ, Foulkes EC. Effects of uranium on the rabbit renal tubules. Toxicol. and Appl. Pharm. 1970;16: 391-399.

19. Nomiyama K, Yamamoto A, Sato C. Assay of urinary enzymes in toxic nephropathy. Toxicol. and Appl. Pharm. 1974; 27: 484-490.

20. Blantz R. The mechanism of acute renal failure after uranyl nitrate. Journ. Clin. Invest. 1975; 55: 621-635.

21. Rothstein A. Berke H., Aminoaciduria in uranium poisoning J. Phar. 96: 179-187, 1949.

22. Clarkson TW, Kench JE., Urinary excretion of amino acids by men absorbing heavy metals, Biochem. J. 62: 361-371, 1955.

23. Butterworth A. Human data on uranium exposure. USAEC Report-58 (1979) 41-46.

24. Howland JW. Studies on human exposures to uranium compounds. In: Voeghtlin C, Hodge HC eds. Pharmacology and Toxicology of Uranium Compounds. 1949: Vol. 3, 993-1017.

25. Katz EJ, Holt LG, Schwartz S. The effect of uranium exposure on urinary catalase excretion. In: Tannenbaum A Ed. Toxicology of Uranium. 1951: 283-289.

26. Scott LM, Bahler KW et al. Mortality experience of uranium and nonuranium workers, USAEC report Y-1729, TID-4500. 1970.

27. Polednak AP and Frome EL. Mortality among men employed between 1943 and 1947 at a uranium processing plant. J Occup Med 23: 169-178, 1981.

28. Flinn, RH, Brinton, HP, Doyle, HN, Cralley, LJ, and Harris, RL. Silicosis in the metal mining industry; A reevaluation 1958-1061. Public Health Service Publication No. 1076. U.S. Government Printing Office, Washington, 1963.

29. Samet, JM, Young, RA, Morgan, MV, Humble, CG, Epler, GR, McLoud, TC. Prevalence survey of respiratory abnormalities in New Mexico uranium miners. Health Physics 46: 361-370 (1984).

30. Wilson, J. An epidemiologic investigation of nonmalignant respiratory morbidity in a uranium mill. Presented at the American Public Health Association Conference, November, 1983.

31. Cross, FT, Palmer, RF, Filipy, RE, Dagle, GE and Stuart, BO. Carcinogenic effects of radon daughters, uranium ore dust and cigarette smoke in beagle dogs. Health Physics 42: 33-52 (1982).

32. Cross, FT, Palmer, RF, Busch, RH, Filipy, RE and Stuart, BO. Development of lesions in syrian golden hamsters following exposure to radon daughters and uranium ore dust. Health Physics 41: 135-153 (1981).

33. Bioassay Program, Kerr-McGee Nuclear Corporation, Grants, New Mexico, revised October, 1982.

34. Bioassay Program, Kerr-McGee Nuclear Corporation, Grants, New Mexico, revised January, 1984.

35. Environmental Health Physics and Industrial Hygiene Standards and Technical Manual. Kerr-McGee Nuclear Corporation, Grants, New Mexico.

36. U.S. Nuclear Regulatory Commission. Draft Regulatory Guide and Value/Impact Statement. Information relevant to ensuring that occupational radiation exposures at uranium mills will be as low as is reasonably achievable. August, 1980.

37. United States Nuclear Regulatory Commission Draft Regulatory Guide 8.22, Bioassay at uranium mills. July, 1978.

38. Alexander RE. Applications of bioassay of uranium. Directorate of Regulatory Standards. U.S. Atomic Energy Commission. Wash-1251, UC-41 June, 1974.

39. Mendenhall, W. Introduction to Probability and Statistics. Fourth Edition. Duxbury Press, North Scltuate, Massachusetts.

AUTHORSHIP AND ACKNOWLEDGEMENTS

Evaluation Conducted and Report Prepared by:

Elizabeth M. Ward PhD
Epidemiologist
Industrywide Studies Branch

Alexander B. Smith, MD, MS
Chief, Medical Section
Hazard Evaluation, Technical
Assistance Branch

Michael J. Thun MD, MPH
Chief, Epidemiology II Section
Industrywide Studies Branch

Bruce Hills, MS
Industrial Hygenist
Industrywide Studies Branch

Originating Office

Industrywide Studies Branch
Division of Surveillance, Hazard
Evaluations, and Field Studies
Cincinnati, Ohio

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3. NIOSH, Region VIII

4. MSHA, Region VIII

5. OSHA, Region VIII

For the purpose of informing the "affected employees," the employer shall promptly post the report for a period of 30 calendar days in a prominent place near where the exposed employees work.

TABLE 1
DISTRIBUTION OF URINE URANIUM
RESULTS FROM KERR-MCGEE

Result ug U/L urine	Number of Samples	Percent of Samples
Less than five	438	38.9
5 to 14.99	420	37.3
15 to 29.99	165	14.6
30 to 44.99	39	3.5
45 to 59.99	22	1.9
60 to 99.99	30	2.7
100 to 499.99	10	0.9
500 to 999.99	1	0.1
1000 or greater	2	0.2

23.9% of samples exceeded the NRC lower action level of 15 ug/L and 9.3% of samples exceeded the NRC upper action level of 30 ug/L.

TABLE 2
COMPARISON OF URINE URANIUM BIOASSAY RESULTS
AT THE KERR-MCGEE URANIUM MILL WITH
THOSE AT THE COTTER URANIUM MILL

URINE URANIUM LEVELS AT COTTER: 1975-1981

Year	Number of Samples	Arithmetic Mean	Median	Percentiles 5th-95th	Percent exceeding 30 ug/L
1975	115	65.2	20	7.0-120	39.1%
1976	141	18.9	8	2.5-92	19.9%
1977	154	20.5	7	2.5-70	20.8%
1978	125	19.1	5	2.5-47	8.0%
1979	887	12.2	6	4.0-37	2.7%
1980	904	9.1	5	4.0-15	1.5%
1981	377	7.2	6	4.0-14	0.0%

URINE URANIUM LEVELS AT KERR-MCGEE: 1979-1982

Year	Number of Samples	Arithmetic Mean	Median	Percentiles 5th-95th	Percent exceeding 30 ug/L
1979	22	17.9	10	1-73	18.2%
1980	347	12.8	6	1-50	8.9%
1981	433	11.3	6	1-45	9.2%
1982	320	12.0	7	1-42	8.1%

TABLE 3
DISTRIBUTION OF INDIVIDUALS
BY MEAN AND MEDIAN
URINE URANIUM VALUES

NUMBER OF INDIVIDUALS

	Mean (3 high values included)	(%)	Mean (3 high values excluded)	(%)	Median	(%)
Less than 5	64	41.0%	64	41.0%	71	45.5%
5 to 14.99	66	42.3%	66	42.3%	67	42.9%
15 to 29.99	19	12.2%	21	13.5%	17	10.9%
30 or greater	7	4.5%	5	3.2%	1 (only 1 sample)	0.6%
Total	156		156		156	

TABLE 4
URINE URANIUM LEVELS BY JOB
(IN ORDER OF INCREASING MEAN URINE URANIUM LEVEL)

Job	# of Samples*	# of Individuals**	Mean	Median	Percentiles 5th-95th	% of samples exceeding 30 ug/L
Ore crushing	2	2	1.0	1	1.0-1.0	0
SX (no samples	--	--	--	-	--	--
Supervisor, area unspecified	238	17	4.1	1	1.0-12.0	1.3
Maintenance/ Electrical area unspec.	238	63	7.8	5	1.0-13.0	3.4
Yellowcake packer	7	5	8.0	6	1.0-19	0.0
Laborer, area unspec.	13	14	8.5	5	0-45.0	7.7
Yellowcake general	45	24	11.4	6	1.0-48.6	13.3
Yellowcake drier	201	34	19.0	12	1.0-64.0	16.4
Yellowcake precipitation	179	22	20.5	12	1.0-67.0	19.0

* Excluding samples with missing results
**Including individuals with missing results

TABLE 5

MEAN AIR CONCENTRATIONS OF URANIUM IN THE YELLOWCAKE AREA OF THE KERR-MCGEE URANIUM MILL (1970, 1975 and 1980)

	Year	u Ci/ml x 10^{-11}	%Current MPC*
Drier area	1970	2.42	24.2
	1975	3.24	32.4
	1980	--**	36.0
Precipitator Operators	1970	2.26	22.6
	1975	2.60	26.0
	1980	--**	38.0

* MPC = 1.0 x 10^{-10} u Ci/ml.
** The 1980 measurements were in units of disintegrations per liter of air per minute gross alpha activity and should be compared to an MPC of 0.222 DPM/L.

TABLE 6

DURATION OF WORK AND CURRENT STATUS AMONG
WORKERS EMPLOYED IN THE YELLOWCAKE AREA
1979-1982

Bioassay ID	Total years at mill	Total years in YC area*	Job	Currently employed?
122	4	1 1/2	Precipitator Operator/ Drier Operator	No
114	5	2	Drier Operator/ Precipitator Operator	No
132	4	3	YC Operator/ Drier Operator/ YC Maintenance Sub.	Yes
121	4	3	Drier Operator	No
116	5	3	Drier Operator	No
102	6	3	Precipitator Operator	Yes
111	5	4	Drier Operator/ Precipitator Operator	Yes
117	6	4	Precipitator Operator	Yes
129	8	4	Drier Operator/ Drier Substitute	Yes
110	6	6	Precipitator Operator/ Drier Operator	Yes
107	7	7	Precipitator Operator	Yes

*These individuals were selected from the bioassay records by examining work histories of individuals with either mean urine uranium levels over 15 or more than 25 bioassay samples.

APPENDIX I
Grouping of Jobs of Kerr-McGee Workers

Area	Job Titles Included
Ore Crushing	Crusher, 2nd class operator
	Crusher, utility operator
SX	SX operator
Yellowcake Drier	Drier relief
	YC Drier sub/trainee
	YC Drier substitute
	YC Drier trainee
	YC Operator - drier
	YC Operator - drier (sub)
	Elec. drier/dismantle wires
	Maintenance drier carpentry work
	Elect. drier/dismantle controls
	Elect. drier/dismantle wires & controls
	Maint. drier overhall
	Maint. sub (YC oper. drier)
Yellowcake packer	Packaging
	Temp. packaging
Yellowcake precipitation	Temp Precip.
	YC operator - precip.
	YC precip. oper. transferred to labor
	Precip. Misc. cleanup work
	Precip. oper. (was drier operator)
	Maint. Precip. misc. EMICO rakes
	Maint. precip. overhall
Yellowcake general	YC substitute
	YC sub. cleanup/washdown
	YC Maint. helper (cleanup)
	YC Maint. and sub.
	Maint. drier/precip. overhall
	Maint. drier/precip. foreman
Supervisory area unspecified	Area foreman
	Foreman
	Maintenance - shift boss
	Shift foreman-milling
	Shift - mill foreman
	Shift boss - milling
	Relief foreman

Maintenance/electrical unspecified	Electrician Elect. sub. (student) Maintenance Maintenance - carpenter shop Maintenance - paint shop Maintenance - rubber shop Student summer help - Maint. Maint. sub. laborer Maint. sub. (paint shop-student) Maint. sub. (rubber shop-student) Maint. sub. (utility oper.)
Laborer, unspecified	Laborer Laborer/misc. duties Student summer help

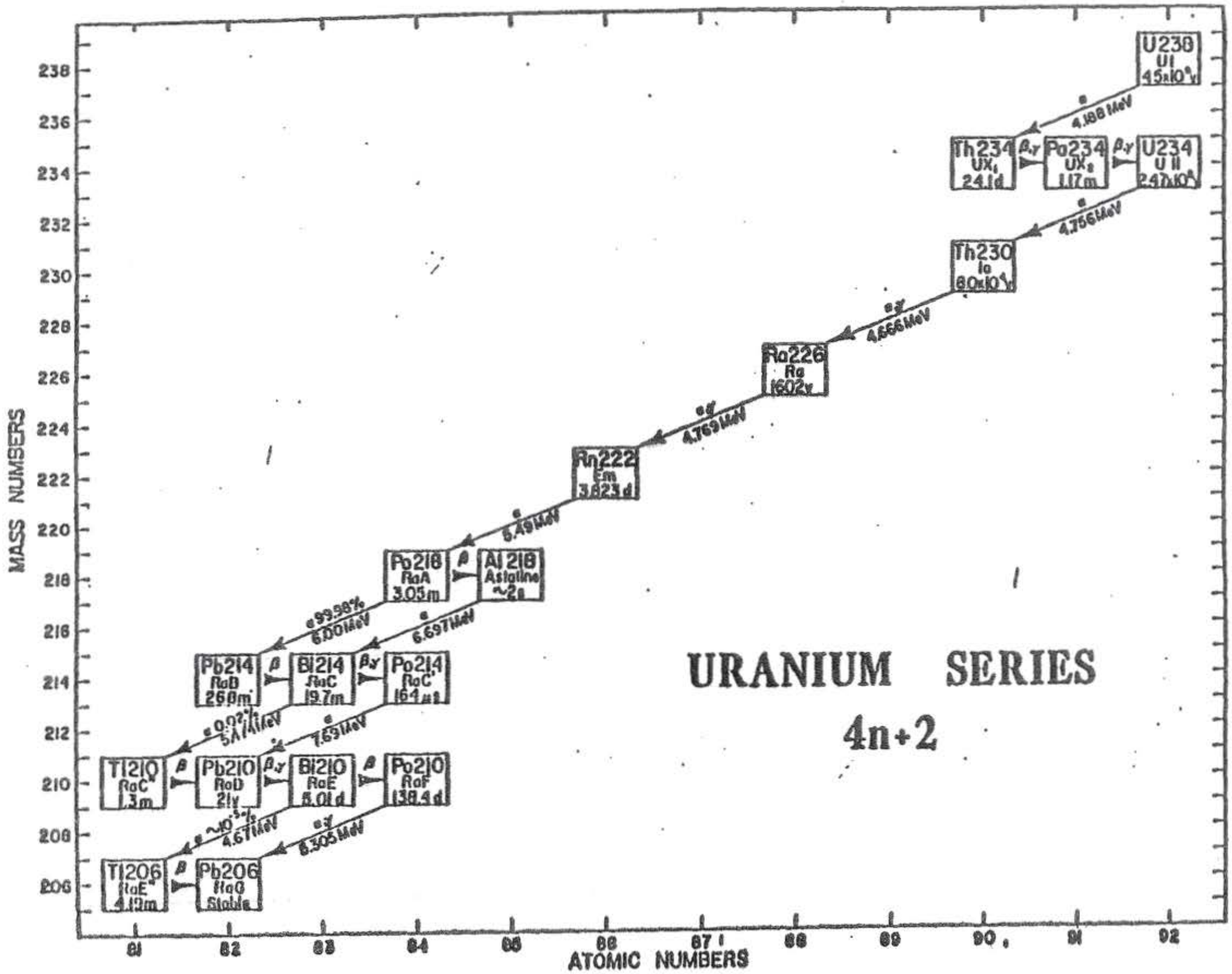
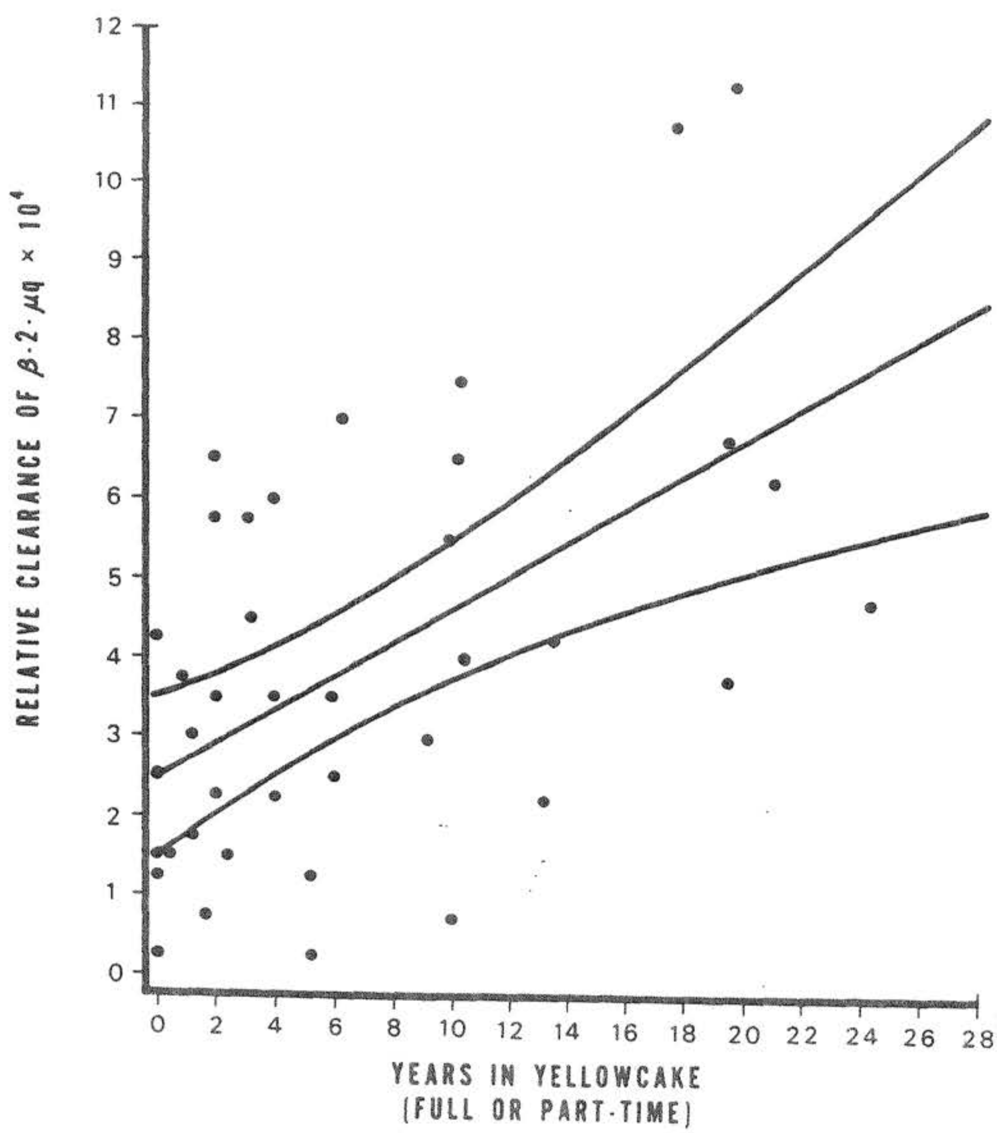


FIGURE 1. URANIUM SERIES DECAY SCHEME

Figure 1

Figure 3


RELATIVE CLEARANCE OF β·2·μg × 10⁴
12
11
10
9
8
7
6
5
4
3
2
1
0
0 2 4 6 8 10 12 14 16 18 20 22 24 26 28
YEARS IN YELLOWCAKE
(FULL OR PART-TIME)

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