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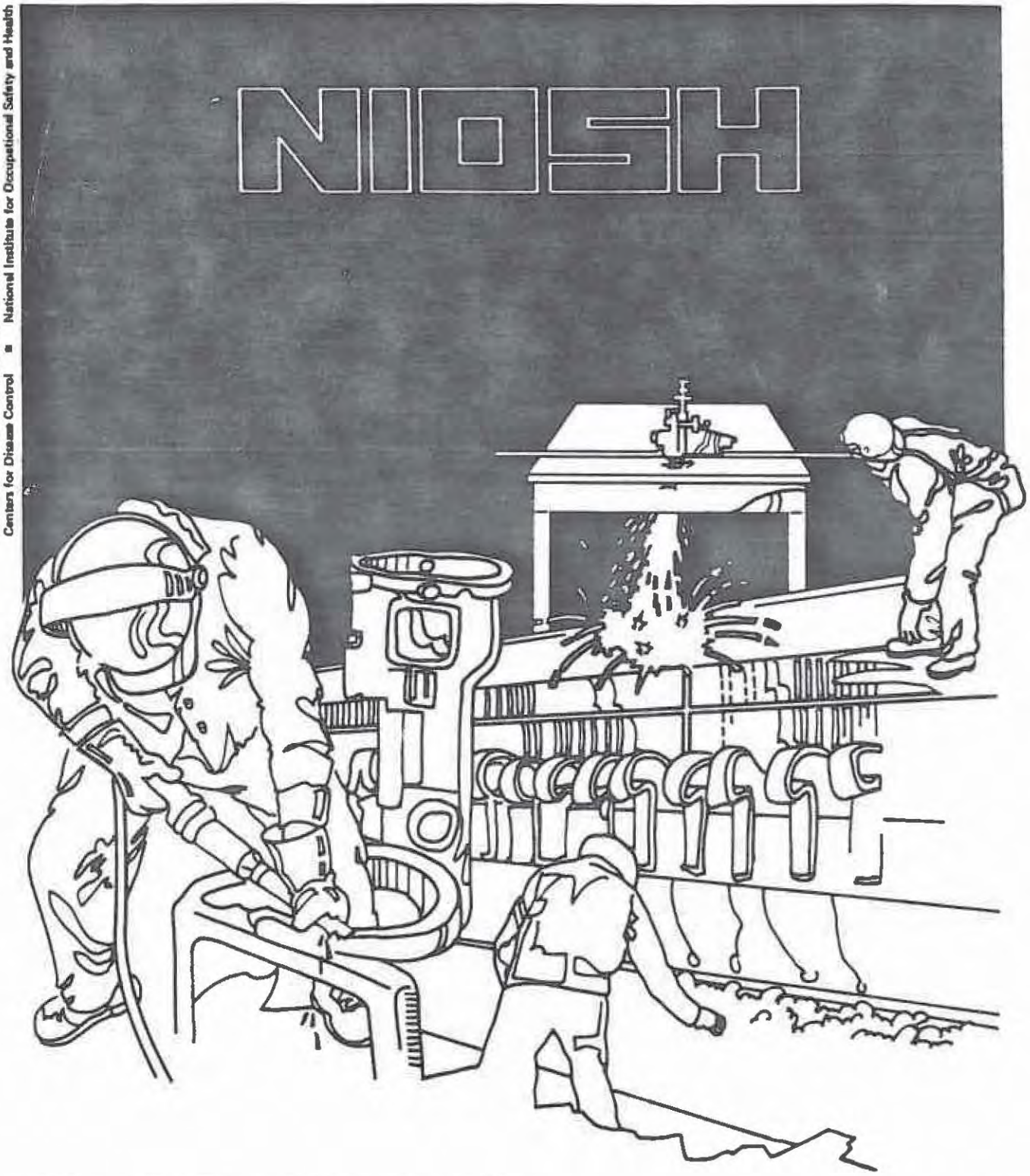

Health Hazard Evaluation Report

HETA 81-055-954
COTTER CORPORATION
CAÑON CITY, COLORADO

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.

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SUMMARY

In October, 1980, the National Institute for Occupational Safety and Health (NIOSH) was asked by the Oil, Chemical and Atomic Workers International Union and its Local 2-844 to evaluate the health effects of exposure to uranium ore dust and to yellowcake at The Cotter Uranium Mill, Canon City, Colorado. The union filed the request after reviewing biological monitoring data which indicated excessive past exposures at the mill.

NIOSH visited the plant on November 12-14 and December 15-17, 1980, and February 17-19, 1981. Investigators reviewed the company industrial hygiene and bioassay records from 1975 to 1981, as well as measurements obtained by the Mine Safety and Health Administration (MSHA) in October 1981. Because of the known nephrotoxicity of soluble uranium compounds, NIOSH also 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.

Company records indicated frequent excessive exposure to uranium in the yellowcake drying and packaging areas of the old mill. Prior to construction of the new mill in 1979, monthly area air samples for total uranium in these areas exceded by as much as 7-8 fold the present occupational standard of $1x10^{-10}$ microcuries per milliliter. Personal air samples during certain operations such as barrel capping were much higher, almost 80 times the present standard. Review of bioassay data for urine uranium indicated that 21.5% of 535 samples measured between 1975 and 1978 exceeded the present limiting value of 30 micrograms per liter which was established to protect against chemical toxicity to the kidney. Both area air uranium monitoring and urine bioassay data indicate that exposures in the new Cotter mill have generally been within the standard.

The medical study found that excretion of the low molecular weight protein, beta-2-microglobulin, was significantly higher in the urine of uranium workers compared to that of control workers. Increased beta-2-microglobulin in the urine is a sensitive marker of renal (kidney) tubular injury, and is consistant 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 exceded 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. A detailed discussion of the medical significance of these findings is included in the report.

Based on these results, NIOSH concluded that there was a health hazard from overexposure to yellowcake at the old mill of the Cotter Corporation. Evidence exists for kidney tubular injury among workers with a history of work in the yellowcake processing area of the old mill. Exposures since 1979 in the new mill have been substantially lower. However, careful industrial hygiene monitoring using personal air samples should be conducted regularly to ensure that exposures in the yellowcake drying and packaging area remain within acceptable limits. Additional medical studies are needed to determine whether sensitive tests of kidney tubular function should be included in the medical surveillance of uranium workers. Further epidemiologic investigation is needed to define the relationship between chronic renal disease and occupational exposure to uranium.

Keywords: SIC 1094 (Uranium mining and milling), uranium, nephrotoxicity, renal effects, chemical toxicity, beta-2-microglobulin

I. INTRODUCTION

In October, 1980, 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 2-844 to conduct a hazard evaluation at the Cotter Corporation uranium mill in Canon City, Colorado. NIOSH was asked to evaluate health effects among the mill workers exposed to uranium ore dust and to yellowcake, a concentrate of natural uranium produced by the mill. The union filed the request after reviewing biological monitoring data which indicated excessive past exposures at the mill.

II. BACKGROUND

The Cotter uranium mill has operated at its present site since 1958. In 1974, Cotter was purchased by Commonwealth Edison Incorporated, a utilities company based in Chicago, Illinois. Cotter receives uranium ore primarily from the Schwartzwalder mine in Jefferson County, Colorado, also a subsidiary of Commonwealth Edison.

The primary function of the mill is to crush, blend, and chemically treat raw ore to extract yellowcake. This product is a chemically complex mixture of diuranates basic uranyl sulfate, and hydrated oxides.[1] The yellowcake is packaged in barrels and shipped to the Allied Chemical Company in Metropolis, Illinois. Before it is usable as nuclear fuel, the uranium must be further enriched by a process that concentrates the U-235.

Prior to 1979 Cotter operated within the original mill buildings, using a carbonate leach extraction process. In 1979, the company rebuilt the entire facility except for the primary crushing area, installed a new sulfuric acid process at the site of the old mill, and added the capability to extract vanadium from the uranium tailings. Although the original workforce expanded from 75 to approximately 200, less than half of the present workers are employed in uranium production.

Cotter is licensed to mill uranium by the Colorado Department of Health, Division of Radiation and Hazardous Wastes. Since 1968 the United States Atomic Energy Commission, now the Nuclear Regulatory Commission (NRC), has delegated regulatory authority to the state. The state's licensing conditions and exposure standards[2] are essentially those of the NRC.[3]

A previous NIOSH hazard evaluation was conducted at the Cotter mill in 1980 by the Division of Respiratory Disease Studies (DRDS). That evaluation, which concerned vanadium exposure in an area of the plant separate from uranium production, could not be completed because of frequent shutdowns in the vanadium process. A report of that evaluation is attached (Appendix A). The present report addresses only the exposures and health effects of the uranium process at Cotter.

III. MATERIALS AND METHODS

Because of the small, highly mobile workforce at Cotter, a mortality study could not be used to assess the long-term effects of radiation. Rather, the investigators chose to address three areas: a literature review of the medical effects of uranium, a review of industrial hygiene exposure records at the old and new mill, and a cross-sectional medical study of kidney effects among those workers with the longest exposure to soluble uranium.

The data on which to base an assessment of past exposures at the old mill consist entirely of industrial hygiene records collected by Cotter under the federal and state licensing conditions. The extent and content of these records is described under the section 'Review of Exposures'. Limited exposure data collected in the new mill by the Mine Safety and Health Administration (MSHA) in October, 1980 supplement the company records. These industrial hygiene measurements are evaluated in relation to current NRC and state standards, as listed under 'Evaluation Criteria'. The methods of assessing kidney function among current workers are described in detail under 'Medical Study'.

IV. REVIEW OF THE LITERATURE AND EVALUATION CRITERIA

A. REVIEW OF THE LITERATURE

Natural uranium presents both toxic and radiologic hazards. We will discuss first the radiation hazards, and then the chemical toxicity of uranium.

1)Radiation

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. They 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.[4]

Epidemiological studies of uranium mines have shown an excess of lung cancer in miners.[5,6] 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 .33 working

levels* over a 12 month period.[3] 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, the only published U.S. mortality study of mill workers did not show an excess of lung cancer.[7] 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. Animal studies have shown that with prolonged, intense respiratory exposure, these poorly soluble dusts accumulate in the thoracic lymph nodes.[8] Organ deposition of the uranium dust is hazardous because of prolonged internal alpha irradiation. Although data have been published from approximately 28 autopsies of workers dying from non-uranium related causes[9] and although these studies have not found equivalent lymphatic deposits, the controversy is not yet resolved. Of particular concern is the epidemiologic finding by Archer,[7] who observed a nearly four-fold excess of deaths from lymphatic malignancies in a small cohort of uranium millers. All of the cases worked in or near the crushing area of the mill where exposures to insoluble dusts were highest. It remains possible that at high air concentrations of uranium dust, where much of the exposure is respirable, substantial chest deposition may occur. No subsequent mortality studies of mills have been published to clarify this issue.

In mills processing unenriched uranium, external radiation is of less concern than is internal. This is because the predominent emissions of natural uranium are alpha and beta particles, which penetrate the skin poorly, but have high biological activity when emitted internally. In contrast, gamma emissions, which do penetrate the body, are emitted in relatively small amounts.

2) 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.[10] 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.[11-13] Compounds such as uranyl nitrate and uranium acetate are used experimentally to create a model of acute renal failure. Injury

* A working level is defined as any concentration of short-lived radon-222 daughters, in one liter of air, that results in emission of 1.3×10^5 million electron volts (MeV) of alpha particle energy.

following acute exposure affects predominently the proximal tubule. 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. However, the occurrence of chronic effects, particularly in man has been inadequately investigated.

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.[10] In part the present standard was selected because of the absence of reported kidney disease in occupationally exposed workers.[14]

Medical surveillance of renal effects in occupational groups has been less thorough than the original toxicological research. A two year followup of an unspecified number of laboratory workers was reported by Howland.[15] 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 [16] 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[15] and England.[17] However, the transience of the albuminuria has been wrongly interpreted as evidence for the absence of chronic renal injury. No systematic medical or epidemiological studies of renal function in uranium workers have been published, nor have sensitive markers of proximal tubular damage been measured, 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.[7] did not examine renal deaths as a distinct category. The study by Scott [18] did not take into account latency in its followup of 4,500 employees at a gasseous 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[19] 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.

B. EVALUATION CRITERIA

For the purposes of this report, exposures are evaluated in relation to criteria from two sources. These include:

1) Nuclear Regulatory Commission and Colorado State Health Department standards for protection against radiation in restricted areas[2,3]
2) Nuclear Regulatory Commission Regulatory Guide on Bioassay at Uranium Mills[20]

Although only criteria from these sources are used, it should be noted that the standards have changed over time, and that numerous other criteria exist both within and outside of the Nuclear Regulatory Commission literature. For instance, the current Occupational Health and Safety standard for exposure to soluble uranium in air is 50 micrograms per cubic meter of air, one quarter the NRC standard.

The table below shows the NRC standards for uranium and certain radiation exposures, and the maximum allowable levels of uranium in urine and in lung. These bioassay levels have been set to prevent toxicity.

EXPOSURE	Nuclear Regulatory Commission Standard (40 hour workweek)
Natural Uranium In Air	1×10^{-10} microcuries/ml (or 200 ug/m^3)
Radon Daughters	0.33 working levels x 12 months/year
Gamma Radiation	5 rem/year (3 rem/quarter)
Beta Radiation	30 rem/year (7.5 rem/quarter)
LIMITING VALUES FOR BIOASSAY	
Urine Uranium	30 micrograms/liter of urine
Lung Burden (By in vivo test)	16 nanocuries

V. RESULTS

A. Review of Exposures

Environmental records at Cotter consist of three general categories: measurements of air concentrations (both of uranium dust and of radon daughters), measurements of external ionizing radiation, and biological monitoring data. In a uranium mill, the two most important categories are air concentrations, (which reflect the intensity of respiratory exposures), and the biological monitoring data, (which reflect the amount of uranium which has actually passed through the body).

Our purpose will be to summarize and interpret exposure records collected by the company since 1975. Most of the exposure records from the new mill, and all from the old mill, were collected by Cutter, under the federal and state licensing conditions. Although it is imposible to assess the accuracy of these industrial hygiene data from the old mill, the data regarding radon daughter exposures can be compared with that collected by MSHA in October of 1980. These data will be used for comparison.

1. Air Concentrations

The purpose of general air sampling is to measure air concentrations of natural uranium (expressed as microcuries/milliliter of air) and to determine respiratory exposures in different areas of the mill. Since area air sampling is conducted at fixed locations which are usually above the height of a worker's breathing zone, the results may underestimate personal exposures.[20] Tables 1 and 2 present area air sampling data for the crushing and yellowcake drying areas of the mill from 1975-81. The areas were chosen as indicative of the highest exposures to insoluble and to soluble uranium respectively. The data in both tables can be compared with the maximum permissable concentration (MPC) of $1x10^{-10}$ uCi/ml. The crushing shed monthly average air levels were always well below the MPC except for one month in 1978. In contrast, measurements in the yellowcake area prior to 1980 frequently exceeded the MPC. The increase in air levels for both areas prior to 1979 may possibly be attributed to improved measuring procedures. The decrease thereafter reflects substantially lower exposures in the new mill.

The small number of breathing zone measurements available indicate that the highest exposures in yellowcake were associated with specific operations such as repackaging the yellowcake or capping the barrels. For instance, of 11 measurements made between 1975-6 during barrel capping, the average was 78.0 x 10^{-10} microcuries/milliliter. Workers performing these tasks were exposed to very high levels of soluble uranium.

Measurements of radon daughters, although scanty prior to 1980, are uniformly low. Table 3 summarizes measurements collected by the company from 1979-81. All are substantially below the 40 hour workweek standard of .33 Working Levels (WL) per 12 consecutive months. One may compare the company measurements with 14 samples collected by MSHA in October 1980. The MSHA results, which average .018 WL (with a high of .06 WL) are substantially higher than the company data, although all are within acceptable limits.

2). External radiation

Numerous measurements of external beta and gamma radiation have been collected with personal film badges at Cotter. These are presented in Tables 4 and 5 for the years 1975-1979. The gamma exposures are low relative to the current standard of 5 rem (5000 millirem) per year or 3 rem (3000 millirem) per quarter. The standard for skin exposure from beta provides an even wider margin (7.5 rem per quarter or 30 rem per year). It is clear that external beta and gamma exposures, at least as reflected by these data, are well below the standards.

3). Biological Monitoring

The two types of biological monitoring measure the uptake of uranium by the body; in vivo, or 'whole body' testing counts gamma emissions as an indication of the amount of uranium deposited within the lungs and chest; urine bioassay measures the amount of urine uranium which has passed through the body and

been excreted by the kidneys. The first reflects exposure to relatively insoluble uranium ore dust; the latter measures predominantly soluble uranium such as yellowcake. Although in vivo testing is called whole body monitoring, it actually measures only gamma emissions from uranium deposited within the chest. A second limitation is that the test cannot distinguish uranium in the lung from that accumulated in other tissues, such as lymphatics.

The few in vivo results available to NIOSH show very small lung burdens of uranium relative to the maximum permissible lung burden of 16 nanocuries.[14] For example, in 1979, the average measurement for 22 persons was 1.22 nanocuries, and the high was 3.8 nanocuries. Although quality control data are absent for these few measurements, the levels are consistant with the results of the area uranium measurements discussed above.

The urine bioassay data from 1975-81 are presented in Table 6 These results, unlike the in vivo data, show that samples frequently exceeded the limiting value set to prevent chemical toxicity of 30 micrograms/liter.[21] From 1975-78, 21.5% of the samples exceeded this level.

In summary, both the air monitoring and the biological monitoring data indicate that the exposures to soluble uranium at the old Cotter mill frequently exceeded the current standards. Exposures to uranium ore dust, radon daughters, and external beta and gamma radiation, at least as reflected by the available industrial hygiene data, appear to have been less of a problem.

B. MEDICAL STUDY

The cross-sectional medical study was conducted to determine whether employees with prolonged exposure to uranium, particularly to soluble uranium, had detectable renal dysfunction. We focused our evaluation upon the kidney because the kidney is the critical organ for soluble uranium exposure, and because renal effects might be expected from the exposures which occurred in the old mill.

1) METHODS

To select the study group, we used personnel records, seniority lists, and employee committees to compile a list of workers most heavily exposed to soluble uranium. These included workers with one or more years of exposure to the yellowcake area of the old mill (n=27), other senior employees (n=12), and recent employees presently in yellowcake (n=3). Of the 42 identified, 39 agreed to participate. Workers at a nearby cement production plant agreed to serve as controls. We selected the controls by first identifying three potential candidates, matched for race, sex, and age (+ 2 years), for each subject from a complete roster of cement production workers. To contact the potential controls we attended routine safety conferences during the two sequential days of our visit and encouraged participation in the study. Although the safety conferences were mandatory, only about a third of the control candidates were assigned to the groups which met on these days. We contacted and recruited 43 of the 44 candidates attending. Subsequently we

excluded seven who had had prior employment at the uranium mill. Thirty six remained, of whom 33 completed matched pairs, while 3 others were duplicates. Thus, the study populations included 39 uranium exposed workers and 36 controls (unmatched analysis), and 33 pairs (matched pair analysis).

We assessed renal function using markers of both tubular and glomerular function. Urinary beta-2-microglobulin excretion was measured as an indicator of low molecular weight (tubular) proteinuria.[22] To assess glomerular function we measured serum and urine creatinine, and urine volume during an eight hour workday. We computed creatinine clearance, adjusting the values to 1.73 m^2 of body surface area using height and weight information and a standard surface area nomogram.[23]

Since the company did not keep records of individual job histories, we collected questionnaire information about the jobs workers had held at the uranium mill. We also asked both subjects and controls about occupational and non-occupational exposure to uranium, lead, cadmium, and other known nephrotoxins such as phenacetin containing analgesics and aminoglycoside antibiotics. We inquired about known personal or familial renal, prostatic, hypertensive or diabetic disease. We also measured blood pressure, and drew blood samples for lead and cadmium determination.

Urine beta-2-microglobulin measurement was by Phadebas[R] radioimmunoassay on urine refrigerated at a pH above 5.5.[24] Measurement of blood lead and cadmium was by anodic stripping voltametry.[25]

For statistical analyses we used standard chi-square, Student's t test, and product moment correlation coefficient, using software by the Statistical Analytic System (SAS).

2) RESULTS

The study and control populations were similar. Both groups consisted of white male Colorado residents from neighboring towns. The age, work histories, mean blood lead, and mean blood cadmium levels of the two groups are presented in Table 7.

Assessment of nephrotoxic exposures other than uranium showed a small but statistically significant elevation of blood lead in controls, relative to subjects (Table 7). The increase, although statistically significant, was well within the range of many 'unexposed' urban populations elsewhere in the United States. It was not explained by activities involving known lead exposure as reported on the questionaire. Blood cadmium levels were within the normal laboratory range (less than .70 micrograms/100cc) and did not differ significantly between the groups.

We next compared parameters of renal function in the two groups. Table 8 presents the unmatched, and Table 9 the matched analysis. By both analyses beta-2-microglobulin excretion (expressed as mg/g creatinine excreted) was significantly higher among the uranium workers than among controls.

Within the uranium exposed group, the single factor which correlated significantly with beta-2-microglobulin excretion was the years worked in the yellowcake area of the old mill (R=.51, p=.001). Table 10 shows the lack of significant correlation between beta 2-microglobulin and other factors. Clearly not correlated were years worked in the crushing area, age, blood cadmium, blood lead, height, and weight. The job 'shifting', which approached statistical significance (R=.30, p=.075), involves rotation through several areas of the mill. Some or all of the time was spent in yellowcake. Table 11 presents the average levels of beta-2-microglobulin in relation to the years worked in yellowcake. Excluded from this table are five 'shifters' who had never officially been assigned to yellowcake, but who had worked longer than one year rotating through different work areas. The appropriate exposure categories of these men could not be determined.

In Tables 8 and 9 we also present the data on serum creatinine and creatinine clearance for the two groups. The following important points should be noted regarding the results of these two markers of glomerular function. In both groups the serum creatinine levels were normal when compared with published population normals. By contrast, the creatinine clearances of both groups were slightly low as compared to the values of a published reference population of similar age distribution.[23] However, when the exposed and control groups were compared with each other, the controls showed a significantly higher serum creatinine (p=.003), and a significantly lower creatinine clearance (p=.04) than did the exposed. These differences were not explained by diabetic, prostatic, hypertensive, or other renal disease in the control group, raising the possibility that the controls were exposed to some unexpected nephrotoxin. The only variable in the control group for which the correlation with creatinine clearance approached statistical significance was blood lead (R=.32, p=.057). The possible significance of this finding will be discussed below.

VI. DISCUSSION AND INTERPRETATION

In summary, the industrial hygiene and biological monitoring data indicate that excessive exposure to soluble uranium occurred rather frequently in the yellowcake area of the old mill. The medical study shows that the uranium workers excreted more urinary beta-2-microglobulin than did the controls, and that those who had worked for longer periods in the yellowcake ares excreted more than did workers with little or no exposure.

The biological significance of the beta-2-microglobulinuria is uncertain. Beta-2-microglobulin is a small protein which is normally filtered by the kidney and then taken up and catabolized (digested) by cells of the proximal tubules.[22] With damage to these cells, as in chronic cadmium poisoning[27] or gentamycin nephrotoxicity,[28] the protein is excreted whole into the urine. Increased excretion may also occur due to abnormal production of beta-2-microglobulin. This has been reported with the lymphoproliferative disorders multiple myeloma and lymphocytic leukemia.[22] Since we did not measure serum beta-2-microglobulin, increased production cannot be differentiated from excessive secretion.

Although the uranium exposed workers excreted significantly more beta-2-microglobulin than did controls, they did not show the very high levels observed in some cases of chronic cadmium poisoning. This is consistant with the data comparing the relative toxicity of the two metals in animals. Comparative studies have shown that uranium, while highly toxic, is relatively less toxic to the proximal tubules than is cadmium.[29]

An unanswered question is whether the observed level of beta-2-microglobulinuria may precede or accompagny more serious renal disease. The present study did not resolve the issue; a) because we evaluated only present workers (i.e., men able to continue working) b) because field measurements of creatinine clearance are unavoidably imprecise and c) because there is some question of whether the cement control group may have been exposed to some unidentified nephrotoxin. As seen in Tables 7-8, serum creatinine in both groups was normal, whereas the mean creatinine clearances, standardized to 1.73 m^2 body surface area, were below published normal values[9] for groups of similar age distribution. We cannot explain why the control group had a significantly higher creatinine ($p=.003$) and a significantly lower creatinine clearance ($p=.04$) than did the uranium workers. We excluded several possible artifactual explanations for the differences. Urine collection was adequate for both controls and exposed and in fact, was better for the controls (mean excretion of 1.76 ml/min. vs. 1.71 ml./min among exposed). No systematic drift in the laboratory measurements of either serum or urine creatinine was evident within the individual groups. It is possible that the low creatinine clearances for both groups, and particularly for the cement production workers, may have been due to the effect of physical exertion upon renal blood flow, and thus upon clearance of endogenous creatinine. Population "normals" as reported in the literature, are derived from 24 hour collections of urine, frequently in hospital settings. Since we know that factors such as renal blood flow and the production of creatinine by skeletal muscle are affected by posture and physical exertion, resting creatinine clearances may differ from those during physical exertion. In the absence of a different control group, however, we cannot exclude the possibility that the cement workers were exposed to some unidentified nephrotoxic agent. Partially supporting this possibility was the correlation between blood lead and creatinine clearance among the controls. Although it is unlikely that these low levels of blood lead are nephrotoxic, lead may indicate the intensity of exposure to some other nephrotoxic component of cement dust.

In conclusion, the present study demonstrates that a health hazard from excessive exposure to soluble uranium (yellowcake) existed at the old mill of the Cotter Corporation. Evidence exists for kidney tubular injury among workers with a history of work in the yellowcake processing area of the old mill. Exposures since 1979 in the new mill have been substantially lower. However, careful industrial hygiene monitoring using both breathing zone and area air samples should be conducted regularly to ensure that future exposures remain within acceptable limits.

The study also identifies two important research needs regarding future medical and epidemiologic surveillance of uranium millers. First, additional

research should determine whether sensitive tests of kidney tubular function, such as the measurement of beta-2-microglobulin in urine, should be included in medical surveillance programs. This is important because none of the screening tests currently recommended or required by the NRC are sensitive to the tubular effects which are the predominent lesion of uranium nephrotoxicity. Second, further epidemiologic investigation is needed to define the relationship between chronic renal disease and occupational exposure to uranium.

VII. RECOMMENDATIONS

A. INDUSTRIAL HYGIENE

1) Careful industrial hygiene monitoring using both area air sampling and breathing zone sampling should be conducted regularly with special attention to the yellowcake drying and packaging areas. The frequency and methods of conducting such surveys should conform to the NRC Draft Regulatory Guide on Health Physics Surveys in Uranium Mills.[30] Thus, area air sampling for yellowcake should be performed weekly in airborne radioactivity areas and monthly in areas where yellowcake is present but not in sufficient quantity for the area to be designated an airborne radioactivity area. Samples should be representative of the air inhaled by workers. The volume of air sampled and the method of analysis should allow a lower limit of detection of at least 20 micrograms of uranium per cubic meter of air. Jobs that are likely to involve more than 10 worker hours at or above the maximum permissible concentration should be monitored with breathing zone sampling. Unusual results should be reported promptly to the radiation safety officer and an investigation of the causes should be made.

2) Environmental monitoring of other exposures, such as surveys for Radon 222 daughters and for external radiation should follow the recommendations of the above reference on Health Physics Surveys. A copy of this document should be made available to the worker safety committee, as well as to the radiation safety officer.

B. BIOLOGICAL MONITORING

1) Biological monitoring using urine uranium bioassay and in vivo monitoring should conform to the recommendations of the NRC Regulatory Guide 8.22, Bioassay at Uranium Mills[21]. A copy of this document should be made available to the workers through the local union health and safety committee.

2) Sensitive tests of kidney tubular function, such as measurement of urinary beta-2-microglobulin, should be evaluated further in research settings before they are included in routine medical surveillance of uranium workers.

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X. DISTRIBUTION AND AVAILABILITY OF REPORT

Copies of this report are currently available, upon request, from NIOSH, Division of Technical Services, Publications Dissemination, 4676 Columbia Parkway, Cincinnati, Ohio 45226. After 90 days, the report will be available through the National Technical Information Service (NTIS), Springfield, Virginia, 22161.

Copies of this report have been sent to:

1. Requestors
2. Colorado State Health Department
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 calender days in a prominent place near where the exposed employees work.

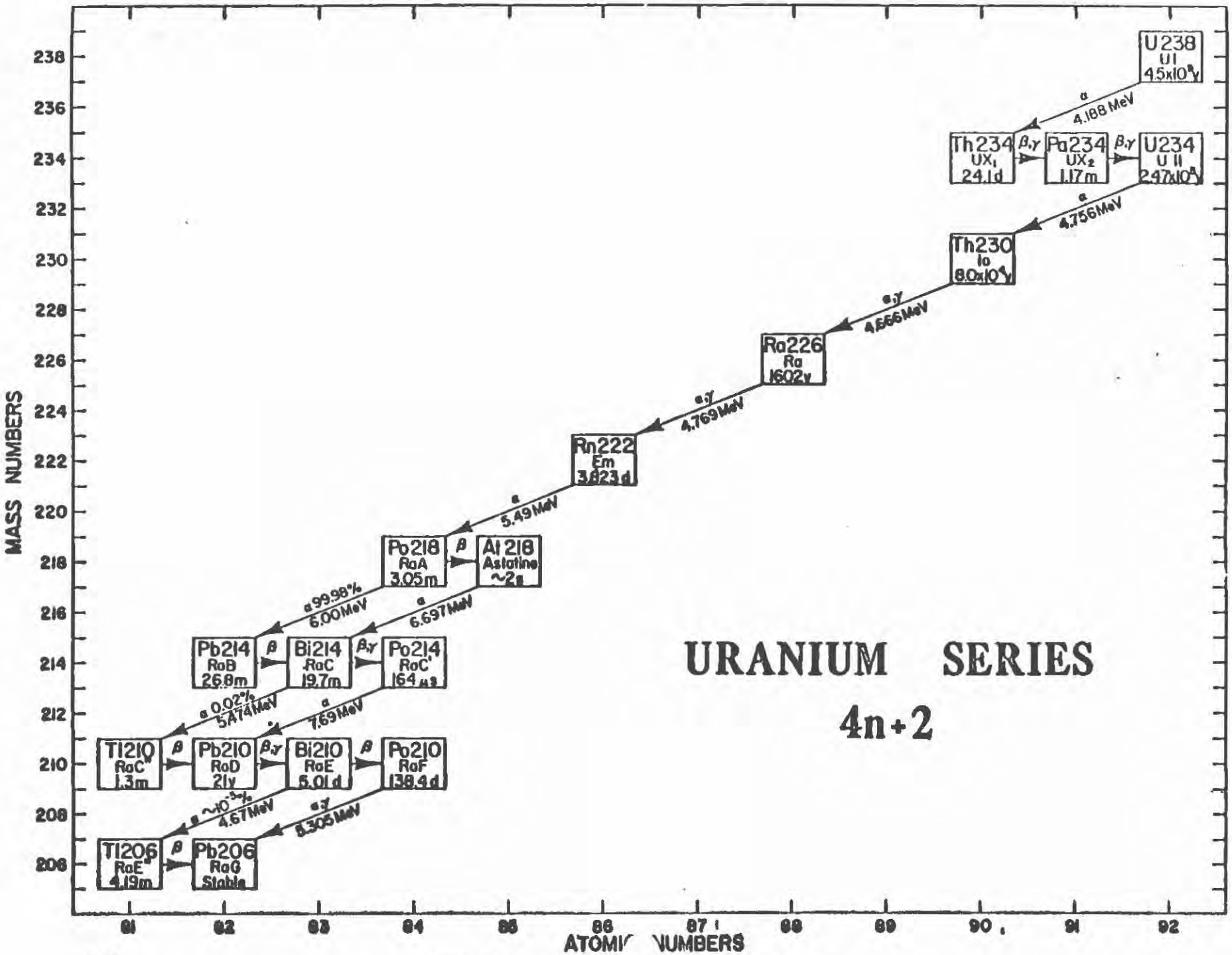

FIGURE 1. URANIUM SERIES DECAY SCHEME

Table 1

Summary of Air Uranium Measurements* In the Crushing Shed 1975-81
Cotter Uranium Mill

Year	Median uCi/ml x10^{-10}	Maximum uCi/ml x 10^{-10}	Percent of Months Exceeding Standard**
1975	.150	.512	0
1976	.154	.527	0
1977	.135	.373	0
1978	.242	2.116	8
1979	.176	.574	0
1980	.064	.485	0
1981	.025	.381	0

* Values are expressed in microcuries per milliliter of air x10^{-10}. They represent averages of the monthly area sampling data collected by Cotter and reported to the Colorado State Health Department. The median is chosen to represent the average because the values are assymetrically distributed.

** The present standard, or maximum permissable concentration of natural uranium in air is 1x10-10 microcuries per milliter of air.

Table 2

Summary of Air Uranium Measurements* In the Yellowcake Dryer Area, Cotter Uranium Mill (1975-81)

Year	Median uCi/ml x10-10	Maximum uCi/ml x 10-10	Percent Exceeding Standard**
1975	.15	1.59	17
1976	.29	.91	0
1977	.44	1.26	17
1978	1.25	5.53	58
1979	.81	7.87	58
1980	.07	.10	0
1981	.07	.40	0

* Values are expressed in microcuries per milliliter of air x 10^{-10}. They represent monthly averages of area sampling for total uranium dust. The data were collected by Cotter and reported to the Colorado State Health Department. The median is chosen to represent the average because the values are assymetriclly distributed.

** The present standard, or maximum permissible concentration in air for natural uranium, is 1×10^{-10} microcuries per milliliter of air.

Table 3

Radon Daughter Measurements* at the Cotter Uranium Mill, (1979-81)

Measurements By Cotter

Year	Number of Samples	Arithmetic Mean (WL)	Maximum (WL)
1979	3	.005	.009
1980	22	.003	.013
1981	75	.015	.394

Measurements by the Mine Safety and Health Administration

1980	14	.018	.06

* Measurements are in working level (WL) units. The current standard permits .33 working levels monthly over a twelve month period.

Table 4

Millirems Per Year of External Beta Radiation*, Cotter, 1975-79

Year	Persons	Arithmetic Mean	Minimum	Maximum
1975	35	1331.4	1789.1	5230
1976	40	836.5	20.0	2200
1977	49	541.2	16.0	1573
1978	52	358.2	12	1772
1979	172	134.4	10	1277

* Values were obtained from personal film badges. None of the results exceed the current standard of 30000 millirems maximum permissable skin exposure to beta radiation per calender year.

Table 5

Millirems Per Year of External Gamma Radiation*, Cotter, 1975-79

Year	Persons	Arithmetic Mean	Minimum	Maximum
1975	33	741.8	10	2200
1976	39	590.8	10	2140
1977	47	422.1	11	1436
1978	48	262.8	10	970
1979	165	93.5	10	474

* Values were obtained from personal film badges. None of the results exceed the current standard of 5000 millirems maximum permissable exposure to gamma radiation per calender year.

Table 6

Urine Uranium Levels at Cotter*: 1975-1981

Year	Number of Samples	Arithmetic Mean	Median	Percentiles 5th-95th	Percent Exceeding Action Level
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%

* Results are in micrograms of uranium per liter of urine. The 'action level is 30 micrograms per liter. In 1979 the lower limit of detection changed from 2.5 to 4.0. The 5th and 95th percentiles are presented to minimize distortion due to contaminated samples.

Data for 1981 include only January through July**

Table 7

Characteristics of the Exposed and Control Groups:
Unmatched Data

Characteristic	Exposed (N=39) Mean (S.D.)	Control (N=36) Mean (S.D.)	Level of Significance*
Age (yrs)	43.7 (10.6)	42.0 (10.2)	ns
Years Employed in Uranium Mill	10.3 (5.8)	0	-
Blood Lead (micrograms/100 cc)	9.4 (2.9)	16.2 (5.6)	.0001
Blood Cadmium (micrograms/100 cc)	0.35 (0.28)	0.28 (0.20)	ns

Level of Significance calculated by Student's t test. NS= p greater than 0.05.

Table 8

Comparison of Renal Function Tests Between the
Exposed and Unexposed Groups: Unmatched Analysis, Cotter 1981

Variable	Group Means		Level of Significance*
	Exposed (N=39)	Unexposed (N=36)	
Urine Beta-2-Microglobulin (mg/g creat.)	0.060	0.036	.004
Serum Creatinine (mg/100 cc)	1.15	1.22	.023
Creatinine Clearance (ml/min)	95.6	88.4	ns

*Level of Significance Calculated By Unmatched t test; ns=p greater than .05.

Table 9

Comparison of Renal Function Between the
Exposed and. Unexposed Groups: Matched Pair Analysis, Cotter, 1981

Variable	Mean Difference Between Pairs	Level of Significance*
Urine Beta-2-Microglobulin (mg/g creat.)	.02	.009
Serum Creatinine (mg/100 cc)	-.10	.003
Creatinine Clearance (ml/min)	8.30	.04

*Level of Significance Calculated By Matched Pair t test ns=p greater than .05.

Table 10

Correlation of Urinary Beta-2-Microglobulin With Single Variables Among the Cotter: Exposed Group Only, 1981

Variable	R Value	Level of Significance
Years in Old Yellowcake	.505	.001
Years in Crushing	-.065	.704
Years as Shifter	.300	.075
Age	.233	.154
Blood Cadmium	-.082	.620
Blood Lead	.180	.274
Height	-.114	.491
Weight	-.268	.099
Systolic Blood Pressure	-.093	.580
Diastolic Blood Pressure	-.228	.589

Table 11

Average Urinary Beta-2-Microglobulin Excretion* Among Uranium Millers In Relation to Years In The Yellowcake Area of the Old Mill: Cotter, 1981

Years in Yellowcake	Persons	Average Urinary Beta-2-Microglobulin
Under 1	8	.035
1-Under 5	14	.051
5-Under 10	9	.067
10 Plus	3	.115

* Units are in mg/g crestinine excreted. The Table excludes five 'shifters' who never worked full time in yellowcake, but worked more than one year at rotating through yellowcake. The appropriate yellowcake category for these men could not be determined.

Table 12

Average Urinary Beta-2-Microglobulin Excretion* Among Uranium Millers In Relation to Years In The Yellowcake Area of the Old Mill, 1981

Years in Yellowcake	Persons	Average Urinary Beta-2-Microglobulin
Under 1	8	.035
1-Under 5	14	.051
5-Under 10	9	.067
Over 10	3	.115

* Units are in mg/g crestinine excreted. The Table excludes five 'shifters' who had less than one year in the yellowcake area, but more than one year of rotating jobs, since the total yellowcake experience of these men is unknown.

APPENDIX A.

CENTER FOR DISEASE CONTROL
NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH

TO : Director, DRDS

DATE: May 6, 1980

FROM : R. S. Bernstein, M.D., Ph.D.
Medical Project Officer

SUBJECT: Interim Report and Recommendations for MSHA TA #80-107: Cotter Corporation and OCAW Vanadium Mill Illnesses

I. SUMMARY

On February 5, 1980, the National Institute for Occupational Safety and Health (NIOSH) received a request to evaluate complaints of nausea, loss of appetite and severe abdominal pains; cough; eye, nose and throat irration; green tongue; difficulty breathing, chest tightness and chest pain; weakness and headache which had resulted in the hospitalization of four Oil, Chemical and Atomic workers employed in the extraction and processing of vanadium by The Cotter Corporation in Cañon City, Colorado.

To evaluate the causes of these symptoms, on February 6- 9, 1980, The Division of Respiratory Disease Studies (DRDS) conducted an industrial hygiene and medical evaluation of the four hospitalized workers, the Cañon City mill and a limited number of other employees who were known to have complained of similar symptoms to a lesser degree of severity.

Area and personal samples for determination of airborne vanadium dusts and fumes were not obtained at the time of the walkthrough but were known to have been out of compliance with present OSHA standards in a number of sites during recent MSHA inspections. Bulk samples were taken for analysis of trace metals. Urine from the hospitalized workers was frozen and later analyzed for trace metals. The process had already been shut down and an epidemiological approach to case confirmation was carried out by interviews, limited physical examinations, and review of hospital records.

The results of bulk sample analyses are still pending at this time. A number of potentially hazardous conditions were observed and pointed out to Cotter Corporation and OCAW officials - eg: The lack of an adequate respiratory protection program; the use and misuse of improper respirator cartridges; the lack of appropriate personal protective equipment; and poor housekeeping methods.

Despite the fact that urinalyses for the four hospitalized workers were negative for evidence of excessive levels of V, As, Pb, Se, or other trace metals, in each case their symptoms and those of other affected workers were strongly clustered in time, place and person-with onset of illness occurring within a short time after the initiation of the vanadium extraction process, recurring on reexposure, and limited to workers regularly or intermittently exposed to these areas. No convincing evidence for an infectious or other etiology was obtained for the hospitalized workers. Of 48 production personnel, 21 (44%) gave histories consistent with work-related vanadium toxicity in the vanadium processing areas. Of 43 maintenance personnel, 17(35%) gave similar histories. The belt filter drying area and solvent extraction area were routinely mentioned by workers giving the above-mentioned symptoms.

On the basis of the observations and data obtained in this study, DRDS/NIOSH determined that potentially hazardous exposures to vanadium and/or other agent(s) occurring in the extraction and processing of vanadium-containing ore existed at The Cotter Corporation mill. Recommendations on ventillation, repirators, work practices and housekeeping were transmitted to Cotter, OCAW, and MSHA officials and are contained in Appendix II.

A proposal to carry out baseline studies of lung function, collect blood and urine for trace metals, administer a standard medical-occupational questionnaire, and collect fingernail clippings for cystine analysis in a subgroup of those workers presumed to be most heavily exposed was not possible because of a lack of cooperation from company and union officials during the shut-down and strike (from 2/7 - 4/10/80). However, a limited survey

of this type could be coupled with an industrial hygiene survey to establish the frequency of acute and sub-clinical health problems once the processes have been back in regular operation for a month or more. No new illnesses have been reported at this time.

II. INTRODUCTION

The Division of Respiratory Disease Studies received a valid request for Technical Assistance from MSHA on 2/5/80 regarding a complaint that the recent onset of work-related acute respiratory and gastro-intestinal illnesses among Oil, Chemical and Atomic Workers (OCAW) employed at the Cotter Corporation in Canon City, Colorado, had resulted in the hospitalization of four employees. It was alleged that the illnesses began to occur shortly after the initiation of a new process of vanadium extraction in December 1979; that the symptomatic employees were those who were assigned to work in the new solvent extraction and product drying areas of the vanadium sides of this uranium-vanadium plant; and that the nature of the alleged symptoms and signs of illnesses were consistent with toxic exposures to vanadium.

An industrial hygiene walk-through survey was carried out (2/6/80) and will be reported separately by Messers, Peach and Wheeler.

Following a brief review of the literature, I conducted a medical/epidemiological site visit (2/8-9/80) in order to confirm cases of affected workers, meet the plant officials and union officials, interview other workers and consult with The Cotter Corporation's local medical doctor. Colorado State Epidemiologist, Dr. Stan Ferguson, was notified of my visit on 2/9/80. Notification of NIOSH and MSHA regional offices that DRDS was conducting a TA in Colorado was provided by the HHE/TA Team Leader, Mr. Peach.

III. BACKGROUND

Three recent publications provide an adequate summary of the occurrence and industrial uses of vanadium and its compounds (1-3). Of the approximately 30,000 tons produced in the world in 1975, about 5,000 tons were produced in the USA. Most of the information on human exposure to vanadium is of occupational origin. Approximately 175,000 American employees have potential exposure to vanadium and its compounds (1). Cotter employs

about 250 hourly workers at Cañon City. Less than half of these employees are routinely exposed to the new vanadium extraction process which was initiated on or about 12/21/79 in this mill which had been extracting uranium for 20 years or so.

Leading American producers of vanadium presently include Union Carbide (Hot Springs, ARK; Uravan, CO; and Rifle, CO), Atlas Minerals (Moab, UT) and Kerr McGee (Soda Springs, ID). A subsidary of the Commonwealth Edison Corporation, The Cotter Corporation has just begun to extract and process vanadium, but officials expect to operate the largest American extraction plant in the near future (projected at 2,000 - 3,000 tons/year).

Details of the Cotter plant's processes will be provided by Messers, Peach and Wheeler. Briefly, uranium-vanadium ores undergo consecutive sulfuric acidification and organic amine chelation processes of extraction in which ammonia and ammonium sulfate are used for pH adjustment and a high grade kerosene is used during the counter-current distribution steps for recovery of organic amines. Vanadium is oxidized to the 5+ state (V_2O_5) with hydrogen peroxide and the product is dried by heat on a moving belt.

IV. PERTINENT LITERATURE AND EVALUATION CRITERIA

Computer-based reviews of NIOSHTIC, TOXLINE, and MEDLINE have provided several more recent case reports or studies of vanadium toxicology than those which have been thoroughly summarized in three recent reviews (1-3).* A search of the NIOSH-related Current Research Activities File provided several additional names of investigators with work in progress including:

Drs. M. Kiviluoto and L. Pyy (Institute of Occupational Health, Helsink, Finland), whose research includes: (1) biologic methods for determining vanadium exposure as well as related diagnostic, preventive, and treatment measures and (2) IH and control technology measures.

Dr. P. Hackett (Battelle Memorial Institute, Richland, WA) whose research concerns placental transfer, teratogenicity, and mutagenicity of lead and vanadium.

*See later under IV., "Review of Studies Published Subsequent to 1977 NIOSH Criteria Document."

Dr. R. G. Cuddihy (Lovelace Inhalation Toxicology Res. Inst., Albuquerque, NM) Metabolism of Inhaled Trace Metals Effluents from Combustion Processes using cultured pulmonary macrophages obtained by lavage after exposure of animals by inhalation.

Dr. M. D. Waters and Dr. Robert Horton (EPA Health Effects Lab, Rearch Triangle Park, NC) whose research includes immunocytotoxic effects of soluble salts of metal pollutants on lavaged rabbit alveolar macrophages. A WHO document on the health effects of vanadium is in preparation by these authors.

Dr. J. D. Shelburne (Duke University, Durham, NC) whose research concerns ultrastructural and X-ray analyses of macrophages exposed to Cd, Ni, Mn, Cr and V.

Present Evaluation Criteria:

The Occupational Safety and Health Administration (OSHA) established the present federal standard for occupational exposures to vanadium and its compounds in 1974 by adoption of the ACGIH TLV of 1968 (4). The scientific evidence on which the present standard is based includes the industrial hygiene literature (5) and experimental studies (6-8). Permissible levels of exposure were established mainly on the basis of the irritative effects of vanadium compounds on mucosal membranes, a property which industrial researchers have ascribed to the acidity of these aqueous solutions in the following order of potency: VCl_3 > V_2O_5 > NH_4VO_3 or $NaVO_3$ > V_2O_3 > alloys of vanadium (9). Exposure standards are as follows:

(1) for vanadium dust (V_2O_5), a ceiling limit of 0.5 mg/m^3

(2) for vanadium fume (V_2O_5) , a ceiling limit of 0.1 mg/m^3

(3) for metallic vanadium, a TWA concentration of 1 mg/m^3 for an 8-hour work shift.

The 1977 NIOSH Criteria Document for a Recommended Standard for Occupational Exposure to Vanadium has not yet been adopted by the Department of Labor (OSHA).

Based on a review of the literature by NIOSH and consultants, two categories of permissible exposure limits were defined:

(1) for vanadium compounds (dusts and fumes), a ceiling limit of 0.05 mg/m^3 based on 15-minute sampling periods.

(2) for metallic vanadium and alloys (dusts and fumes), a TWA concentration limit of 1 mg/m^3 for up to 10 hours/day for a work week not exceeding 40 hours.

There are inadequacies in the number and quality of the industrial hygiene and medical/epidemiological studies of vanadium toxicology which form the scientific basis for the present and proposed workplace standards (1-2). Systemic, pharamacodynamic and metabolic effects (eg the physiological significance, if any, of vanadium's well-documented inhibitory effects on (Na, K)-ATPase); chronic respiratory effects; sensitization by repeated exposures; reproductive effects on males and females as well as measures of mutagenicity, teratogenicity, and carcinogenicity have not been adequately studied. A major criticism of all of the epidemiological studies published through 1977 is the lack of consideration of potentially confounding factors such as cigarette smoking and prior occupational exposures. In this light, the NIOSH recommendations for environmental sampling, medical surveillance and record keeping are very important as is the need for properly designed industry-wide studies in the above mentioned areas of inadequate data. The action level has been defined as equal to the recommended environmental limits for vanadium compounds and metallic vanadium because, when absorbed, they appear to be excreted rapidly and to exhibit low degrees of toxicity.

Evaluation of Acute Overexposure to Vanadium:

While the demonstration of systemic poisoning effects, COPD and sensitization caused by vanadium exposures will require further adequately designed studies, there is no doubt that over-exposure to vanadium produces the following manifestations (1-3):

(1) Mucosal irritation of the eyes, nose and upper airway characterized by discharge from the mucous membranes (rhinitis, conjuctivitis, nose bleeds and

nasal perforation have been reported); cough (productive or dry and hacking, but often persistent after delayed onset and worsened on re-exposure); chest systems (including burning pain in the pharynx, trachea and chest, dyspnea, wheezing, tightness or shortness of breath, congestion and bronchitis); and gastrointestinal symptoms (metallic taste, nausea, anorexia, diarrhea).

(2) The appearance of a green-to-black discoloration of the tongue is an indication of exposure rather than intoxication and may represent the biological reduction of vanadium to the 3+ oxidation state to form the green hexaaquo ion, $[V(H_2O)_6]^{3+}$.

It can be appreciated that the similarity of these manifestations to those of an acute respiratory tract infection makes an unequivocal diagnosis or case confirmation difficult to establish. This is especially true if one accepts some of the reported systemic manifestations as part of the case definition (e.g., malaise, weakness, headache).

Attempts to develop sensitive diagnostic tests which are specific for vanadium toxicity have not yet been successful. The following approaches have been used to document exposure:

(1) Qualitative evidence of acute over-exposure can be demonstrated by determination of the vanadium content of blood and 24-hour urine specimens obtained immediately after a possible toxic exposure and sequentially there after (10)(14)(18). The pharmacodynamyics of vanadium inhalation exposure in humans has not been adequately studied. It appears that absored vanadium is excreted mainly in urine, but also in the feces (17% of an ingested dose is excreted in the urine while 81% appears in the feces). The half-life of an intravenous dose of vanadium is about 96 hours in the rat. Analysis of blood samples should measure the vanadium content of serum and cellular constituents separately since it appears that vanadium is usually transported by the serum transferrin* but may be adsorbed to cells or lipids when present in excess of the usual carrier sites (10). Urine levels should be expressed on a per mg of creatinine basis (14)(15)(18).

*See Sabbioni, E., et al "The Association of Vanadium with the Iron Transport System in Human Blood...," Nuclear Activation Techniques in the Life Sciences, International Atomic Energy Agency, Vienna (in press).

(2) The most sensitive test of chronic over-exposure involves the measurement of a decrement in the cystine content of the fingernails. This reduction in fingernail cystine has been correlated with chronic exposure and has been used as a clinical tool in surveillance programs for vanadium workers, but was not discussed in the NIOSH Criteria Document (11)(15). It is not clear whether this well-documented reduction is the result of a direct inhibition of the synthesis or incorporation of cystine or cysteine in fingernails; whether it is related to vanadium's toxic effects on (Na,K)-ATPase (23)(26); or whether vanadium somehow interferes with cystine absorption or metabolism to produce cystinuria (2)(12). Approximately 3 months are required for the growth of fingernails (i.e. from the time of initial exposure) in order to observe these effects.

Review of Studies Published Subsequent to 1977 NIOSH Criteria Document (13-26):

Three of these studies are in Japanese or German and I am awaiting translations (15-17) . The validity of blood and urine analyses for correlation with levels of exposure has been confirmed for groups, but not for individuals in three of these studies (14)(15)(18). There has been confirmation of the earlier reports of a significant reduction of fingernail cystine in chronically exposed workers compared to controls (15). Since it takes 3-3 1/2 months from the time of initial exposure until an effect is manifest at the tip of the fingernail, it is not surprising that acute serum and urine levels of vanadium do not seem to correlate well with fingernail cystine levels (11). In addition numerous basic science reports from reputable investigators published in high quality refereed journals have confirmed earlier reports of toxic effects of vanadium in a variety of oxidation states and physical-chemical forms on pulmonary macrophage function, on Na-K ATPase activity in a variety of tissues, on myocardial function and on sperm motility (19-29).

Kiviluoto, etal (13), have provided the first study of macroscopic, cytological and histological changes in the nasopharynx and sputum of 63 workers exposed for an average of 11 years to monitored levels of vanadium dust and matched by age and smoking history with neighborhood controls exposed to "inert" dusts. Air sampling between 1970 and 1975 indicated average vanadium

levels of 0.2 - 0.5 mg/m^3 in the "total dust gathered on filter paper" (there was no indication of the particle size distribution nor the volume of air sampled). At the end of the men's two- or four-week vacation in the summer of 1975, a longitudinal study began with a base line otological examination of the upper respiratory tract. At the same time a cross-sectional study was carried out to evaluate sputum cytology, nasal secretion smears, nasal mucosal biopsies from the mid-inferior turbinates, and ventilatory function tests. However, at the beginning of 1976, the company introduced industrial hygiene measures which resulted in a decrease of breathing zone and area exposures to 0.01-0.04 mg of vanadium per cubic meter. In March-June of 1976, the exposed group was re-examined by the same otologist and thirty-one of them underwent cytological re-evaluations. The results of matched-pair analyses demonstrated: (a). No significant differences in the prevalence of common cold nor in the findings of either anterior or posterior macroscopic rhinoscopy between the two groups at the baseline exam, and no significant changes in the exposed group at the follow-up exam; (b). A significant increase in the number of plasma and round cells as well as mucosal papillarity was noted in the biopsies of exposed workers, in addition to an increase in the number of neutrophils in their nasal secretions; (c). There were no increased numbers of secretion eosinophils nor other signs of allergic inflammation; and (d). There were no changes suggestive of a carcinogenic effect.

A major criticism of this work is the unknown effects of the dramatic change in exposure levels during the study and the lack of follow-up histological data on the effects of the reduced exposure after the baseline examinations. Also, there is no data on biological absorption of vanadium from blood, urine or other tissues and the results of lung function studies were not reported.

In the paper by Gylseth, etal (14), the technique of neutron activation analysis* has been employed to examine whether variations in exposure to vanadium fumes and ferrovanadium dust produce correlated detectable

*See Zoller, W.H. and Gordon, G.E., Anal. Chem. 42 (1970) 257-265. and Allen, R.O. and Steinnes, E., Anal, Chem. 50 (1978) 1553-1555.

changes in urine and blood vanadium levels. Six persons were designated as low- or non-exposed on the basis of their job descriptions and eleven persons were considered moderately- or highly-exposed on the basis of personal air sampling. For the latter group, air sampling in the breathing zone was carried out using Casella personal air samples at 2l/min (? liters/min) and 37 mm, 0.8μm Millipore filters. Exposure levels of vanadium for the moderately- or highly-exposed group ranged from about 0.04 to 4 mg/m^3 during four successive workdays of sampling. About 2/3 of these samples were between 0.05 and 0.1mg/m^3. Blood and urine samples were collected from both groups before and after the workshift on the first day as well as after the workshift on the fourth day. The vanadium on the filters was analyzed by atomic absoption spectrometry. Neuton activation, following a "difficult and expensive" analytical technique with a "chemical yield of 50 - 70%, was used to analyze urine and blood samples. The latter were not separated into serum and cells as suggested by Schroeder, etal (10). The results gave no consistent correlation between exposure level and blood- or urine-vanadium concentration for individual workers. Within each exposure group there was no statistically significant difference in blood or urine vanadium concentration between the before and after-shift mean values on the first day, or between the before-shift mean values on the first day and the after-shift mean values on the fourth day. However, when the blood- and urine-vanadium concentration data for each group were pooled (disregarding the day and time of collection) there was statistically significant differences between the two groups for blood-vanadium and for urine-vanadium corrected for the creatinine concentration. A regression analysis of n mol of vanadium per liter of blood vs n mol of vanadium per mg of creatinine in urine samples gave a correlation coefficient, r=0.50. The authors do not recommend this method of surveillance for the routine control of vanadium dust exposures.

A major criticism of this work is the lack of exposure data for the low- or non-exposed group. Considering the "difficult and expensive" nature of the neutron activation analytical method, it is of interest that the authors noted a 50-fold difference in the blood-vanadium concentration measured by this technique in non-exposed Italian vs Belgian persons as reported in Nuclear Activation Techniques in the Life Sciences, IAEA, Vienna (in press).

V. RESULTS OF WALK-THROUGH SURVEYS

Environmental Observations (See separate trip report by Messers, Peach and Wheeler):

The following chemicals are known to be present in the ore: fluorides, manganese, magnesium, arsenic, selenium, aluminum, molybdenum and phosphorous. The solvent extraction of vanadium (decribed briefly under THE INDUSTRY) involves the use of: "alamine 33" to chelate the vanadium, a high grade kerosene (nepoleum H-70B), isodecyl alcohol, ammonia, ammonium sulphate, sulfuric acid and hydrogen peroxide.

I met with plant officials in a conference on 2/8/80. In attendance were Bill Badger (Assistant Manager), Bob Maixner (Health Physics), Jerry Powers (Health and Safety), and Dale Delaney (Personnel). Mr. Miles Fixman (Plant Manager) was unavailable. Corporate headquarters are in Chicago (Mr. George Rifakes is President of Cotter which is a subsidiary of Commonwealth Edison). The corporate medical director is Dr. Mehn (312-294-4003) and his associate is Dr. Haley. Regional headquarters are in Lakewood, CO. (Mr. McCloskey is Executive Vice President and General Manager and Mr. Rick Schwartz is Production Manager). Following several recent MSHA citations of Cotter for incorrect respirators and short-term over-exposures under existing standards, the company contracted with engineering (Tom Moyer Assoc) and industrial hygiene consultants (Terry Howard of Hager Labs, Denver) to obtain advice for appropriate control technology modifications. The company attorney (Mr. Winston Duke) is presently contesting MSHA's authority to oversee milling operations on jurisdictional grounds.

The vanadium processing was temporarily discontinued on 2/7/80 pending the substitution of soda ash for ammonium sulfate and other engineering and IH measures including ordering the appropriate respirators. When Mr. Krauth and I asked for respirators to walk through the vanadium drying area on 2/9/80 after notifying Mr. Badger of our intentions, the warehouse had still not ordered the NIOSH/MSHA recommended cannisters. The OCAW went on strike 3/1/80 and operations have only recently begun again, although at this writing the vanadium extraction has not yet reached the product drying operation.

Medical Case Confirmation:

On 2/8/80 I met with Dr. Darvin Ritchie who is a local family practitioner acting as the Cotter Corporation plant physician. We called the plant and informed them that I would be in town for two days to speak with management and union officials as well as affected employees and to visit the plant in order to confirm cases and assess the situation.

At St. Thomas More Hospital, Dr. Ritchie and I reviewed the records and examined the four hospitalized employees who had been admitted on 1/28, 2/3 (two men), and 2/6/80 for diagnostic studies and management of severe abdominal pains in addition to other symptoms typical of vanadium toxicity: persistent dry cough worsened on re-exposure, eye irritation, nose bleeds, dyspnea and substernal burning pain, weakness, anorexia, and headache. Only one of the four had a transient fever (100.2°F) on admission; but his pulse was 70, his chest clear to auscultation and roentgenography and he had no adenopathy or other abnormality on physical exam or laboratory testing. Another man had been treated 10 days prior to admission with erythromycin for tracheitis (a common complaint consistent with vanadium irritation of the upper airway in this non-smoking, non-drinking Mormon gentleman). He had developed an increased total (indirect) bilirubin of 5.7 which was decreasing to normal limits by the time of admission after the erythromycin had been discontinued. In my opinion, all other findings were within normal limits for these four men except that one of them had a positive mononucleosis test and a subsequent heterophile test was negative. All of the men were managed with symptomatic therapy and discharged after several days of extensive tests.

In summary only one of these four men was briefly febrile at the time of admission; all of the men gave me similar histories of onset of classical symptoms of vanadium toxicity; in each case their symptoms were correlated in time and place with work in the new vanadium processing areas and not experienced prior to the institution of this process nor in other areas of the plant; and no convincing evidence was obtained

during their hospitalization to support an infectious etiology for their complaints. Therefore, I believe that their illnesses were caused or aggravated by vanadium and/or other agents present in those new processes. Urines collected early in their hospitalizations had been frozen by the hospital pathologist, Dr. Benzmiller. To document exposure I requested that these specimens be sent to NIOSH/DPSE in Cincinnati for qualitative analysis of trace metals and quantitative analysis of vanadium, lead and arsenic. Only one of the four men was noted to have a green tongue on admission. The results of the urinalyses were negative for evidence of excessive trace metals (including V, Pb, As, Se, Fe, Zn, Al, Be, Cd, Co, Cr, Cu, Mn, and Ni.)

With the help of Mr. Mike Krauth, the OCAW local president, I obtained information from a number of other hourly employees and observed several individuals with green discoloration of their tongue. Out of a total of 43 maintenance personnel, 17 men had complained of eye irritation and cough (plus or minus other chest symptoms) which they had experienced when assigned to work in the new vanadium processing areas* of the plant--especially the belt filter ammonium vanadate drying area which was mentioned by 9 of the 17. Out of a total of 48 men who work in the uranium vanadium plant (I don't know how many of them are regularly located on the vanadium side) 21 men had complained of some or all of the following recurring vanadium process-related symptoms: cough (13); eye, nose and/or throat irritation (14); chest symptoms (14); and gastrointestional symptoms (11).

In summary 35% of maintenance workers and at least 45% of process workers gave histories of symptoms which were consistent with acute overexposure to vanadium.

Medical Surveillance Program (See Appendix I):

The pre-employment history and physical exam includes a lumbo-sacral X-ray, chest roentgenogram, and other routine blood and urine laboratory procedures. There are routine periodic follow-up exams every 2 years, but

*These symptoms were reported as having their initial onset within 10 days to 2 weeks after the new vanadium extraction process began in the last week of December,

no routine exams on job transfer or termination. The next routine exam is scheduled for 5/9/80 but the corporate medical director expressed no interest in obtaining baseline urine and blood data on trace metals at this time. Records of terminated employees are maintained--but are kept in boxes without adequate organization. The state of Colorado recently conducted a mortality study of the county in which the mill is located in connection with a license renewal. (Dr. Stan Ferguson, State Epidemiologist, has the details which do not seem relevant to this problem.)

Union-Management Issues:

Contract negotiations between Cotter and OCAW began at about the same time as the onset of illnesses associated with the new vanadium extraction process. There have been anecdotal reports of falsification of health and safety records by Cotter as well as multiple MSHA citations and our own observations of their lack of response to NIOSH IH recommendations. In general, an adversary relationship exists between OCAW and Cotter. The OCAW went on strike against Cotter on 3/1/80 and the plant began partial operations again on 4/10/80.

VI. RECOMMENDATIONS

On 2/15/80, a meeting was held with the Director, DRDS, to discuss the IH and medical/epidemiological findings of the Cotter Plant walk through surveys. It was noted that the vanadium process had been temporarily discontinued and that OCAW and Cotter were requested to notify us when the modified process was started up again which they failed to do. An MSHA inspection is also being planned when the process is back in full operation. It was also noted that NIOSH research objectives concerning inadequately documented areas of vanadium toxicology might not be accomplished in a medical/epidemiological investigation of the Cotter plant which has only been processing vanadium for several months and is now engaged in modifying the process to be used. However, a NIOSH environmental/IH survey for Technical Assistance to MSHA would document whether process changes will bring Cotter into compliance with present OSHA standards.

A list of eight recommendations for the correction of potentially hazardous conditions was sent to the Cañon City plant management, OCAW and MSHA officials on 2/25/80 over the signature of the Director, DRDS, NIOSH (these are attached as Appendix II).

Although an industry-wide study would be required to address the research needs regarding systemic effects, COPD, sensitization, and diagnostic/surveillance methods, it might be appropriate to carry out a limited medical/epidemiological survey at the time of environmental sampling a month or so after the modified Cotter process is reinstituted. Such a survey could include a questionnaire regarding medical/occupational history, respiratory and gastrointestinal symptoms and smoking habits for all Cotter employees, as well as a shift study of PFT's and biological measures of exposure (urine and blood for vanadium content and fingernails for cystine content) for a group of highly exposed individuals. It would be necessary to identify an appropriate control group in Canon City to adjust for bias related to lung function and dietary or drinking water sources of vanadium.

A limited medical/epidemiological study would also be helpful to the Union and the Department of Labor in making a decision regarding the need for promulgation of a new vanadium standard based on an updated version of the 1977 NIOSH Criteria Document.

Because of a lack of cooperation by the Union and Management during the strike it was not possible to carry out baseline studies of blood and urine levels of vanadium as well as pulmonary function studies prior to the return to work under the modified vanadium process conditions. Fingernail cystine levels at this time might have reflected the effects of exposures in January during the early phases of the onset of illnesses.

We have asked both OCAW and Cotter officials to request an HHE to obviate the jurisdictional dispute and will continue to be available to MSHA for technical assistance if illnesses recur or if it seems appropriate to carry out the IH/Medical-Epidemiology Survey which I mentioned above.

Robert S. Bernstein, M. D.

cc: Chiefs, CIB, EIB
Team Leader, MHETAP

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PUBLIC HEALTH SERVICE
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February 25, 1980

re MSHA TA #80-107

President
Cotter Corporation
Box 751
Canon City, Colorado 81212

Dear Sir:

This letter is a follow-up to the visit Mr. Michael J. Peach III and Mr. Robert W. Wheeler, P.E. made to your facility on February 6, 1980. At the time of the visit, Mr. Peach and Mr. Wheeler made several recommendations to your staff safety officer, Mr. Jerry Powers, regarding potentially hazardous conditions that were observed. They are as follows:

1. We recommend that a comprehensive respiratory protection program be implemented immediately to include, but not limited to, the proper selection, wearing and cleaning of NIOSH/MSHA approved respirators. The elements of a comprehensive respiratory protection program are contained in the NIOSH publication, "A Guide to Industrial Respiratory Protection" which has been provided to Mr. Jerry Powers under separate cover.

2. Special attention must be given to the selection of respirator cartridges in that workers in selected operations may be exposed to multiple contaminants as in the solvent extraction area where the workers are exposed to mists, vanadium oxide fumes, ammonia gases, and organic vapors. Ambient airborne concentrations of ammonia measured to date by MSHA reflect concentrations to be well below the current MSHA standard. Therefore, a combination high efficiency filter chemical cartridge is required to protect against mists, vanadium oxide fumes, and organic vapors.

3. As recommended by NIOSH in the NIOSH/OSHA "Pocket Guide to Chemical Hazards", also sent to Mr. Powers under separate cover, a full face piece should be worn in the vanadium product line and solvent extraction areas.

4. Individual respirators should be assigned to each worker requiring their use. The practice of workers using the same respirator in subsequent work shifts is poor sanitary practice and should be discouraged.

Workers required to enter and work in confined spaces such as the product bin in the vanadium product operation should be provided with and required to wear a type C Supplied Air (SA) respirator with a full face piece operated in pressure-demand or positive

Appendix III

pressure mode or with full face piece, helmet, or hood operated in continuous-flow mode (ref. pages 22, 108, and 109, NIOSH/OSHA "Pocket Guide to Chemical Hazards"). Requirements for the quality of the air supply is given on page 93, par. 1910.134 of the "Guide to Industrial Respiratory Protection" previously mentioned. In addition, information on maintenance of air supply compressors which should be considered are being provided for your information under separate cover.

6. Proper eye protection, aprons, and boots should be routinely worn by all workers handling liquid chemicals. In addition, ladles with extended handles should be used when dipping liquid samples from tanks. It would be prudent to wear protective clothing over their work clothes when working on solvent exchange and product line and this clothing should be removed prior to entering the control room.

7. Consideration should be given to installing a hand washing facility in the control rooms, both in the extraction area and product line, so that hands can be washed before eating lunch, ingesting any food products, or smoking.

8. Workers dirty clothes should <u>not</u> be stored in the same room with clean clothes. Separate change rooms should be set up so that work clothes laden with dust and chemicals do not come into contact with clothes that are worn to the home. A good practice would be to wash all work clothes of workers on the vanadium product line.

The above recommendations made are based on observations made of potentially hazardous conditions observed by NIOSH industrial hygienists during a walk-through survey of your facility and are intended to assist you in protecting the health of your employees.

Thank you for the help and courtesy you extended to our industrial hygienists during their visit to your facility.

Sincerely yours,

James A. Merchant, M.D., Dr. P.H.
Director
Division of Respiratory Disease Studies

cc:
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HKrauth, OCAW Local 2-644
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Appendix II

DEPARTMENT OF HEALTH AND HUMAN SERVICES
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