BIOLOGICAL MONITORING OF VANADIUM

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INTRODUCTION

Vanadium is a very common greyish-white metal and nonessential to man. It does not exist in the free state and is difficult to obtain in pure metal form. Carnotite is the most important mineral for vanadium extraction. Vanadium is also found in some petroleum and bitumens. The most widely used compounds are: vanadium dioxide (V$_2$O$_5$), vanadium trioxide (V$_2$O$_3$), vanadium pentoxide (V$_5$O$_7$), sodium metavanadate (NaV$_2$O$_7$), vanadium tetrachloride (VC$_4$) and vanadium sulfide (VS).

Vanadium and its compounds are widely used in industry in the following processes:

- in the production of special vanadium-iron steels used for the manufacture of high speed mechanical tools;
- with other metals (Cu,Cr,Cu,Ti,C) in the composition of corrosion and temperature resistant alloys, e.g., in the aeronautical industry;
- in iron and steel refining and tempering;
- in hard metal production;
- for lining arc welding electrodes;
- in the manufacture of pigments, printing inks and paints;
- in the glass industry;
- in the form of V$_2$O$_5$ as a catalyst in the pharmaceutical industry.

The main risks involved with vanadium exposure occur during the processes of vanadium extraction and preparation from the mineral and to a greater extent during cleaning and repair of petrol containers (Stokinger, 1981). The ACGIH TLV-TWA is 0.05 mg/m$^3$ for dusts and fumes as V$_2$O$_5$, (1985).

METABOLISM

Vanadium is absorbed from the digestive and respiratory tract. The metal is found in the diet, especially in cereals, milk, some vegetables and cooking oils. It has been calculated that daily intake with the diet is
about 2 mg/day (Schroeder et al., 1963). According to some authors, however, the intake is on the order of a few tens of micrograms (Byrne and Kosta, 1978). Absorption of the quantity introduced with the diet is usually low (<1 percent) and thus, ingested vanadium compounds are mainly eliminated with the feces (Byrne and Kosta, 1978).

After intratracheal instillation of radiolabeled vanadium pentoxide in rats, most of the metal was cleared from the lungs by 14 days (Rhoads and Sanders, 1985). After absorption, following fairly uniform early distribution in soft tissues, bone and marrow are the long-term storage sites both in humans and experimental animals (Rhoads and Sanders, 1985; Sharma et al., 1980; Yukawa et al., 1980).

In mice vanadium was deposited in the placenta and in mammary glands. The accumulation of vanadium in the placenta produced irregular development of the fetus (Roshchin and Ordjonikidze, 1978).

Urinary excretion is the predominant route of elimination of absorbed vanadium. In a recent investigation (Sabbioni and Maroni, 1983) the kinetics of urinary elimination of vanadium was studied by evaluating the mean levels of excretion after cessation of exposure in two groups of workers with different degrees of vanadium exposure. Figures 1 and 2 show that in the subjects with "high" exposure (who showed mean urinary vanadium values between 60 and 70 μg/g creatinine at the end of exposure), the half-life was about 20 hours. A period of about 600 hours after cessation of exposure was required to reach urinary vanadium levels of 2 μg/g creatinine (upper value in the control group). In the subjects with "low" exposure (who showed mean urinary vanadium levels of 4.3 μg/g creatinine) the half-time was about 15 hours, and about 18 hours were required after cessation of exposure to reach excretion levels of 2 μg/g creatinine. Urinary elimination of vanadium in humans is therefore rather slow and is similar in behaviour to that observed in experimental animals (Sabbioni and Marafante, 1978). These data suggest the possibility of a slow accumulation of the metal in the body in the course of occupational or environmental exposure.

HUMAN EFFECTS

Exposure to vanadium dusts and fumes causes irritant effects mainly on the respiratory tract. Even brief exposures cause rhinorrhea, epistaxis, dyspnea and acute asthmatic bronchitis with remission usually in a few days. Exposure to vanadium may also result in pneumonia (Kiviluoto et al., 1979; Levy et al., 1984; Wyers, 1946).

A group of volunteers were exposed in an inhalation chamber for 8 hours to vanadium pentoxide dust at a concentration of 1 mg/m³; after about 7 hours persistent cough appeared, lasting about 8 days after cessation of exposure. No alterations of respiratory function parameters or chest X-ray findings were observed. In volunteers exposed to concentrations of 0.5 mg/m³ under identical experimental conditions, coughing occurred the morning after exposure and lasted for about a week. However in subjects exposed to concentrations of 0.1 mg/m³ no irritant phenomena were observed during or immediately after exposure, but rhinorrhea occurred after about 48 hours (Zenz and Berg, 1976).

In a group of 63 workers exposed to vanadium dust concentrations of 0.02-0.04 mg/m³ there was significant increase in polymorphonuclear neutrophils in the nasal mucous and in plasma cells in nasal mucosa biopsies (Kiviluoto et al., 1979).

There is no consensus of opinion in the literature as to bronchitic manifestations becoming chronic, or associated with pulmonary emphysema. A