

General Description

Vanadium (V) is a bright white ductile metal belonging to group V of the periodic system of elements. It forms compounds mainly in valence states +3, +4 and +5. In the presence of oxygen, air or oxygenated blood, or oxidizing agents, vanadium is always in the +5 oxidation state. In the presence of reducing agents, vanadium compounds are in the +4 oxidation state (1). Vanadium forms both cationic and anionic salts, and can form covalent bonds to yield organometallic compounds which are mostly unstable.

Sources

Vanadium is an ubiquitous metal. The average concentration of vanadium in the earth's crust is 150 µg/g (2); concentrations in soil vary in the range 3-310 µg/g (3) and may reach high values (up to 400 µg/g) in areas polluted by fly ash (4). The concentration of vanadium in water is largely dependent on geographical location and ranges from 0.2 to more than 100 µg/litre in freshwater (2), and from 0.2 to 29 µg/litre in seawater (3). The ocean floor is the main long-term sink of vanadium in the global circulation (4). The concentrations of vanadium in coal and crude petroleum oils vary widely (1-1500 mg/kg) (2).

The world production of vanadium was about 35 000 tonnes in 1981 (5), the major producing countries being Chile, Finland, Namibia, Norway, South Africa, USSR and the United States.

Most of the vanadium produced is used in ferrovanadium and, of this, the majority is used in high-speed and other alloy steel (usually combined with chromium, nickel, manganese, boron and tungsten).

It has been estimated that around 65 000 tonnes of vanadium annually enter the environment from natural sources (crustal weathering and volcanic emissions) and around 200 000 tonnes as a result of man's activities (6). The major anthropogenic point sources of atmospheric emission are metallurgical works (30 kg per tonne of vanadium produced), followed by the burning of crude or residual oil and coal (0.2-2 kg per 1000 tonnes and 30-300 kg per 10⁶ litres) (7).

Global vanadium emissions into the atmosphere from coal combustion in 1968 were estimated to range from 1730 to 3760 tonnes. The contribution of vanadium to the atmosphere from residual-fuel combustion was estimated at 12 400-19 000 tonnes in 1969 and 14 000-22 000 tonnes in 1970. In the production of ferrovanadium for alloy additions in steel-making, vanadium emission to the atmosphere was estimated at 144 tonnes in 1968 (2). The burning of wood, other vegetable matter and solid wastes probably does not result in significant vanadium emission. In 1972, about 94% of all anthropogenic emissions of vanadium to the atmosphere in Canada (2065 tonnes) resulted from the combustion of fuel oil and only 1.2% from metallurgical industries (8).

Occurrence in air

Currently the ambient air levels of vanadium are quite low, but it is suspected that air concentrations will increase in the future as a function of accelerated fossil-fuel combustion in rural areas.

Concentrations vary from tenths of nanograms to a few nanograms. In the air of big cities the annual average was between 50 and 100 ng/m³ (8,9) with significant seasonal variations

(winter averages are more than three times the summer averages). More recent data from a five-year study in four Belgian cities show yearly averages of 41-179 ng/m³ (10). A recent survey of measurements in Member States of the European Community indicates the following concentration ranges: remote areas, 0.001-3 ng/m³; urban areas, 7-200 ng/m³; industrial areas, 10-70 ng/m³ (11). Concentrations up to 2 µg/m³ have been reported in several cities in the northeastern USA. A marked decline of vanadium concentration in ambient air was reported after the introduction of low-sulfur fuels (4). Air pollution by industrial plants may be less than that by power stations and heating equipment. The average concentration in the area surrounding a steel-plant site in Pennsylvania, USA, was 72 ng/m³ (2). Vanadium pentoxide (V₂O₅) was detected near a large metallurgical works in concentrations ranging from 0.98 to 1.49 µg/m³ in 87% of samples and exceeding 2 µg/m³ in 11% of samples (12).

With combustion of the carbonaceous matrix, vanadium is released to the atmosphere as fine fly ash with a long atmospheric residence time. Vanadium concentrates on the surface of particulate matter from coal combustion. The concentration of vanadium in fly ash from residual fuel-oil burning averages around 8% (range 2-18%), representing 56% (39-74%) of the vanadium concentration in fuel. The soluble fraction of fly ash vanadium averages 70% (42-96%). About 80% of the vanadium-containing particles have a mass median aerodynamic diameter smaller than 0.5 µm at the chimney, representing 10-20% of the total aerosol mass of vanadium (13).

A large number of occupations involve exposure to vanadium. Concentrations in the working environment range from 0.01 to 30 mg/m³ (14), with varying particle distribution patterns and different degrees of solubility for different vanadium compounds. Very high levels of vanadium in the air were reported in boiler cleaning (17-60 mg/m³) (15).

Routes of Exposure

Air

Concentrations of airborne vanadium have increased in recent years, probably because of the increasing direct combustion of crude oil residues in power plants and community-heating systems. The average annual values for urban areas are reported to be in the range of 0.05-0.18 µg/m³. Maximum concentrations of vanadium as high as 2 µg/m³ occur in areas of greatest population density, during the coldest part of the year and during the late evening hours.

Drinking-water

Concentrations of vanadium in drinking-water may range from about 0.2 to more than 100 µg/litre (16); typical values appear to be between 1 and 6 µg/litre (8). The concentration of vanadium in drinking-water depends significantly on geographical location.

Food

The few data on vanadium concentrations in food vary considerably; this may be explained by differences in the food itself and in the analytical methods used. The mean vanadium concentration in the diet was reported to be 32 µg/kg (range 19-50 µg) (17) and the mean daily intake was estimated to be 20 µg/day (18). Byrne & Kosta (19) estimated that the daily dietary intake of vanadium amounts to several tens of micrograms.

The concentration of vanadium in human milk was found to be 0.1-0.2 ng/g (18). An infant drinking one litre of human milk per day would thus have a daily vanadium intake of 0.1-0.2 µg (8).

Relative significance of different routes of exposure

According to a pathway analysis (8), an individual living in a rural environment (assumed air concentration 8 ng vanadium per m³) will have a body-burden of around 100 µg, over 80% of which is derived from the diet; an urban dweller (assumed air concentration 70 ng/m³) will have a total body-burden of around 200 µg (in the latter case, the inhalation route contributes approximately one half of the total burden).

It was estimated that the daily intake by ingestion is about 20 µg and by inhalation 1.5 µg in an urban area and 0.2 µg in a rural area (8).

Kinetics and Metabolism

Absorption

The lungs are a significant site of entry of vanadium in the case of community exposure. The distribution pattern of particles and the solubility of vanadium compounds, as well as alveolar and mucociliar clearance, are important factors that determine the rate of absorption in the respiratory tract. Moreover, the irritative effect of vanadium compounds can significantly modify the absorption of vanadium by the lungs. Vanadium accumulates in the human lung with age, reaching approximately 6.5 µg/g in the over-65 years age group (3). The lung clearance of vanadium pentoxide is relatively rapid in animals after acute exposure, but substantially slower after chronic exposure. The metal is deposited in the lung in relatively insoluble forms. Soluble compounds are partly absorbed, but the extent of absorption in the respiratory tract has not been determined.

The absorption rate of vanadium compounds after ingestion depends on their solubility and chemical nature. Absorption of cationic vanadium is low, not exceeding 0.1-1% (8). Skin is probably a minor route of absorption in man (9).

Distribution

Absorbed vanadium is transported mainly in the plasma, bound to transferrin. The average value for the distribution of blood vanadium between plasma and cells in rats after an intravenous injection of 0.9-30 µg vanadium per kg was found to be 9 : 1 (20). Pentavalent vanadium is reduced in erythrocytes to the tetravalent form. This reduction is a glutathione-dependent process (21).

Byrne & Kosta (19) reported blood vanadium levels in healthy individuals of below 0.5 µg/litre. The analytical method used was neutron activation analysis with pre-separation of vanadium.

Vanadium is widely distributed in body tissues; principle organs of vanadium retention are kidneys, liver, testicles, spleen and bones. A major fraction of vanadium from cellular vanadium was found retained in nuclei (22). In pregnant rats the injected vanadium was found in the fetus (23).

Elimination

Vanadium is excreted mainly in the urine, but also in the faeces. Bile is probably not an important pathway for excretion into the faeces, but the existence of alternative routes for excretion into the gut (salivary excretion or direct transfer across the intestinal wall) has been suggested (20). When 4.5-9.0 mg vanadium as diammonium oxytetravanadate was fed daily to 16 elderly persons, urinary excretion, although quite variable, amounted to a mean of 5.2% of the amount ingested (24).

Health Effects

Nutritional studies have shown that vanadium is an essential element for the chick and rat (3). Its deficiency may result in growth reduction, impairment of reproduction and disturbances in lipid metabolism. Vanadium is also essential for soil nitrogen-fixing microorganisms (3). It may play a significant role in human nutrition (2).

Biochemical, physiological and pharmacological properties of vanadium compounds have been reviewed (1,25). It has been suggested that vanadium may be a regulatory agent for enzymatic activities in mammalian tissues. Vanadium is a potent inhibitor of many enzymes, while it stimulates adenylate cyclase. It has been shown to inhibit cholesterol biosynthesis and lower plasma cholesterol levels. Vanadium can also directly influence glucose metabolism *in vitro* and may play a role in its regulation. Lipid peroxidation of rat lung extracts, liver microsomes and mitochondria was induced by sulfite and accelerated by the presence of vanadium compounds.

Vanadium may play a physiological role as part of a control on levels of the endogenous antioxidant, glutathione. This may also be important with respect to toxic interactions of chemicals (26).

Effects on experimental animals

The toxicity of vanadium has been found to be high when it is given parenterally, low when it is orally administered, and intermediate in the case of respiratory exposure. Toxicity is related to the valency of the vanadium compound (increasing with increasing valency). Vanadium is toxic both as a cation and as an anion (16,27).

Severe acute exposure (tens of mg/m^3) is responsible for systemic effects. Most frequent findings in animal experiments were in the liver, kidneys, gonads and the nervous, haematological and cardiovascular systems (2,3,16). However, systemic effects at very low levels of exposure were also reported (28). When rats were continuously exposed to an aerosol of vanadium pentoxide at levels of 6 and 27 $\mu\text{g}/\text{m}^3$ (3.4-15 μg vanadium per m^3), neurotoxic effects appeared 30 days from the start of exposure. Measured chronaxy ratio between the flexor and extensor muscles of the tibia returned to normal about 18 days following cessation of exposure. Histopathological changes observed in the liver following the higher level of inhalation exposure (27 $\mu\text{g}/\text{m}^3$ for 70 days) included central vein congestion with scattered small haemorrhages, and granular degeneration of hepatocytes. The kidneys showed marked granular degeneration of the epithelium of the convoluted tubules. In the heart, myocardial vascular congestion was observed, with focal perivascular haemorrhages.

Numerous studies reported acute or chronic respiratory effects in laboratory animals following exposure to different vanadium compounds. Respiratory effects in rats at relatively low levels of vanadium pentoxide aerosol (3-5 mg/m^3) for 2 hours every other day for 3 months, included perivascular oedema, capillary congestion, haemorrhages, and in some cases desquamative bronchitis or pneumonia (29). Similar effects were also observed at much lower levels. Rats exposed to vanadium pentoxide aerosol of 27 $\mu\text{g}/\text{m}^3$ for 70 days developed different respiratory effects, such as marked lung congestion, focal lung haemorrhages and extensive bronchitis (28). Exposure to 2 μg vanadium pentoxide per m^3 was considered to be without effect.

Exposure to vanadium oxides may alter alveolar macrophage integrity and function to the detriment of pulmonary defence. Cytotoxicity, tested on rabbit alveolar macrophages *in vitro*, was directly related to solubility in the order $\text{V}_2\text{O}_5 > \text{V}_2\text{O}_3 > \text{VO}_2$. Dissolved vanadium pentoxide (6 $\mu\text{g}/\text{ml}$) also reduced phagocytosis (3). The effect of vanadium compounds on the function of alveolar macrophages may result in an impairment of the lung's resistance to

secondary bacterial infection.

It has been stated that VOCl_2 is a mutagen for *Escherichia coli* and VOCl_2 , V_2O_5 , NH_4VO_3 are capable of damaging DNA (30). There is no information that vanadium compounds have embryotoxic, teratogenic and carcinogenic effects.

Effects on humans

Both acute and chronic poisonings have been described in workers engaged in the industrial production and use of vanadium. Most of the reported clinical symptoms reflect irritative effects of vanadium on the respiratory

There is insufficient evidence that vanadium causes generalized systemic effects except at extremely high concentrations (3). Several observers described only vague, general signs or symptoms and reported nervous disturbances, neurasthenic or vegetative symptoms, occasionally tremors, palpitation of the heart, high incidence of extrasystoles, changes in the blood picture (anaemia, leukopenia, punctatebasophilia of the erythrocytes), reduced level of cholesterol in the blood, etc. (16).

Numerous studies have reported acute and chronic respiratory effects mainly due to exposure to vanadium pentoxide (2,3,16). Most of the reported clinical symptoms reflect irritative effects of vanadium on the upper respiratory tract. Only at high concentrations (above 1 mg vanadium per m^3) were more serious effects of the lower respiratory tract observed, such as bronchitis and pneumonitis. Respiratory effects after acute or chronic exposure to low levels of vanadium are summarized in Table 1.

Acute changes in lung function in workers exposed to vanadium compounds during cleaning of boilers were reported (time-weighted average concentration of respirable dust 523 $\mu\text{g}/\text{m}^3$, 15.3% of vanadium, giving a value of 80 μg vanadium per m^3) (31). In spite of the fact that the workers wore respirators, the changes in lung function developed within 24 hours and pre-exposure levels had not returned by the eighth day; however, the efficacy of the respirators was low (about 9% leakage).

Respiratory effects after acute exposure to vanadium pentoxide dust were also observed in a clinical study in which healthy volunteers were exposed to levels of 100, 200 and 1000 μg vanadium pentoxide per m^3 for 8 hours (32). It was found that 98% of the particles were smaller than 5 μm . Coughing, which persisted for several days, was the most characteristic symptom at all three levels of exposure.

Chronic inhalation of vanadium pentoxide dusts in industry has resulted in rhinitis, pharyngitis, bronchitis, chronic productive cough, wheezing, shortness of breath and fatigue. Pneumonitis and bronchopneumonitis have also been observed (2,3,16). It has been reported that vanadium workers are more susceptible to colds and other respiratory illnesses than others.

Respiratory effects after chronic exposure to vanadium pentoxide were described in refinery workers (33) at an average level of 300 μg vanadium per m^3 and at a maximum concentration of about 1 mg vanadium per m^3 (92% of the particles were smaller than 0.5 μm). In comparison to controls, a statistically significant higher incidence of symptoms, described as cough with sputum production, eye, nose and throat irritation, injected pharynx and green tongue were reported.

In workers exposed for several months to vanadium-containing dust in the range of 10-40 μg vanadium per m^3 (concentrations were previously higher, 200-500 μg vanadium per m^3), a macroscopic and microscopic survey of the upper respiratory tract of 63 males was performed (34). The results were compared with a control group of workers exposed to inert dust only, and matched for age and smoking habits. Microscopic examination of nasal smears revealed a statistically significant increase in neutrophils, and biopsies of nasal mucosa showed significantly elevated numbers of plasma and round cells. The histological picture

was characteristic for an irritating effect of vanadium dust on the mucous membranes seen earlier in exposed laboratory animals.

One study described a significant incidence (58%) of injected pharynx in workers engaged in the refining of vanadium pentoxide from soot generated by the combustion of heavy fuel-oil (35). Concentrations of vanadium in the air at various locations in the work environment were all less than $400 \mu\text{g}/\text{m}^3$, with mean values of $1.2\text{-}12.0 \mu\text{g}/\text{m}^3$.

Vanadium compounds appear to be capable of inducing asthma bronchiale in previously nonatopic subjects and continuing manifestations of asthmatic symptoms were reported 8 weeks after subjects had left the industry (vanadium pentoxide refinery) (36).

Some epidemiological data have shown positive correlations between the vanadium content of urban air and mortality from bronchitis, pneumonia, nephritis, cancer (other than lung cancer in males) (37) and "heart disease" (38). However, these analyses did not take into account important, probably relevant, factors such as exposure to other chemicals, smoking habits, etc. (16). In addition, most of these causes of excess mortality have not been reported in workers occupationally exposed to vanadium compounds (3).

Evaluation of Human Health Risks

Exposure

The natural background level of vanadium in air has been reported to range from 0.02 to $1.9 \text{ ng}/\text{m}^3$ in Canada (2). Vanadium concentrations recorded in rural areas varied from a few nanograms to tenths of a nanogram per m^3 , and in urban areas from 50 to $200 \text{ ng}/\text{m}^3$. In big cities during winter, when high-vanadium fuel-oil was used for heating purposes, concentrations as high as $2000 \text{ ng}/\text{m}^3$ have been reported. Air pollution by industrial plants may be less than that caused by power stations and heating equipment.

The concentrations of vanadium in workplace air ($0.01\text{-}60 \text{ mg}/\text{m}^3$) are much higher than in the general environment.

Health risk evaluation

The acute and chronic effects of vanadium exposure on the respiratory system of occupationally exposed workers should be regarded as the most significant factors when establishing air quality guidelines. Most of the clinical symptoms reported reflect irritative effects of vanadium on the upper respiratory tract, except at higher concentrations (above $1 \text{ mg vanadium per m}^3$), when more serious effects on the lower respiratory tract are observed. Clinical symptoms of acute exposure are reported (31) in workers exposed to concentrations ranging from $80 \mu\text{g}/\text{m}^3$ to several $\text{mg vanadium per m}^3$, and in healthy volunteers (32) exposed to concentrations of $56\text{-}560 \mu\text{g vanadium per m}^3$ (Table 1).

A study of occupationally exposed groups provides data reasonably consistent with those obtained from controlled acute human exposure experiments, suggesting that the lowest-observed-adverse-effect level for acute exposure can be considered to be $60 \mu\text{g vanadium per m}^3$.

Chronic exposure to vanadium compounds revealed a continuum in the respiratory effects, ranging from slight changes in the upper respiratory tract, with irritation, coughing and injection of pharynx, detectable at $20 \mu\text{g vanadium per m}^3$, to more serious effects such as chronic bronchitis and pneumonitis, which occurred at levels above $1 \text{ mg}/\text{m}^3$. Occupational studies illustrate the concentration-effect relationship at low levels of exposure (33-35), showing increased prevalence of irritative symptoms of the upper respiratory tract; this suggests that $20 \mu\text{g vanadium per m}^3$ can be regarded as the lowest-observed-adverse-effect level for chronic exposure (Table 1). There are no conclusive data on the health effects of

exposure to airborne vanadium at present concentrations in the general population, and a susceptible subpopulation is not known. However, vanadium is a potent respiratory irritant, which would suggest that asthmatics should be considered a special group at risk.

There are no well documented animal data to support findings in human studies, although one study reported systemic and local respiratory effects in rats at levels of 3.4-15 $\mu\text{g}/\text{m}^3$ (28).

Guidelines

Available data from occupational studies suggest that the lowest-observed-adverse-effect level of vanadium can be assumed to be 20 $\mu\text{g}/\text{m}^3$, based on chronic upper respiratory tract symptoms. Since the adverse nature of the observed effects on the upper respiratory tract were minimal at this concentration and a susceptible subpopulation has not been identified, a protection factor of 20 was selected. It is believed that below 1 $\mu\text{g}/\text{m}^3$ (averaging time 24 hours) environmental exposure to vanadium is not likely to have adverse effects on health.

The available evidence indicates that the current vanadium levels generally found in industrialized countries are not in the range associated with potentially harmful effects.

Table 1. Respiratory effects after acute and chronic exposures to low levels of vanadium

Type of exposure	Vanadium compound	Concentration in $\mu\text{g}/\text{m}^3$		Symptoms	Reference
		Compound	Vanadium		
Acute					
Boiler cleaning	V_2O_5	523	80	Changes in parameters of lung functions	(31)
Clinical study (experimental 8-hour exposure)	V_2O_3	1000	560	Respiratory irritation: persistent and frequent cough, expiratory wheezes	(32)
	V_2O_5				
	V_2O_5	200	112	Persistent cough (7-10 days)	
	V_2O_5	100	56	Slight cough for 4 days	
Chronic					
Vanadium refinery	V_2O_5	536	300	Respiratory irritation: cough, sputum, nose and throat irritation, injected pharynx	(33)
Vanadium refinery	V_2O_5	18-71	10-40	Irritative changes of mucous membranes of upper respiratory tract	(34)
Vanadium processing	V_2O_5	—	1.2-12.0	Respiratory irritation: injected pharynx	(35)
	V_2O_3				

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