PRELIMINARY INVESTIGATION OF EFFECTS ON THE ENVIRONMENT
OF BORON, INDIUM NICKEL, SELENIUM, TIN, VANADIUM AND
THEIR COMPOUNDS
VOLUME VI - VANADIUM

VERSAR, INCORPORATED

PREPARED FOR

ENVIRONMENTAL PROTECTION AGENCY

AUGUST 1975

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PRELIMINARY INVESTIGATION OF

EFFECTS ON THE ENVIRONMENT OF BORON, INDIUM

NICKEL, SELENIUM, TIN, VANADIUM AND THEIR COMPOUNDS

Volume VI

Vanadium

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Project Officer

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August 1975

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Volume VI

Preliminary Investigation of Effects on Environment of Vanadium and Its Compounds

FOREWORD

This is Volume VI of a series of six reports on the environmental effects of boron, indium, nickel, selenium, tin, and vanadium and their compounds. The information is based on literature reviews, direct contact with representatives of companies involved in the production or use of the materials, and consultation with knowledgeable individuals from industry, academic institutions and the Federal Government.

I. VANADIUM INDUSTRY IN THE UNITED STATES

    A. Producers and Sites

        Table 1 lists commercially prepared vanadium compounds and the companies which produce them. Only those chemicals whose production exceeds 1/2 metric ton or $1000 are listed. Other materials may also be mentioned in the discussion because of their unusual properties, such as toxicity, or their anticipated future significance.

Table 1
(1,2)

Chemicals, Producers and Plant Locations

Chemical	Producer	
	Company, subordination	Location
Lithium vanadate	Gulf Resources & Chem. Corp. Lithium Corp. of America	Bessemer City, N.J.
Ammonium vanadate	Kerr-McGee Chem. Corp.	Soda Springs, Idaho
	Union Carbide Corp. Mining and Minerals Div.	Urevan, Colo. Hot Springs, Ark.
	Var-Lac-Oid Chem. Corp.	Elizabeth, N.J.
Sodium vanadate	Atomergic Chemetals Co.	Carlè Place, L.I., N.Y.
	Gallard-Schlesinger Chem. Mfg. Corp.	Los Angeles, Cal.
	Cerac, Inc.	Monomonee Falls, Wis.
	Research Organic/Inorganic Chem. Corp.	Belleville, N.J. Sun Valley, Cal.
	Var-Lac-Oid Chem. Co.	Elizabeth, N.J.
Vanadium metal	Atomergic Chemetals Co.	Carle Place, L.I., N.Y.
	Gallard-Schlesinger Chem. Mfg. Corp.	Los Angeles, Cal.
	Cerac, Inc.	Menomonee Falls, Wis.
	Electronic Space Products, Inc.	Los Angeles, Cal.
	Research Organic/Inorganic Chem. Corp.	Belleville, N.J. Sun Valley, Cal.
	United Mineral & Chem. Corp.	New York, N.Y.
	Var-Lac-Oid Chem. Corp.	Elizabeth, N.J.
	Ventron Corp, Alfa Prod. Div.	Bradford, Pa. Sunnyvale, Cal.

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Table 1. (cont.)

Chemical	Producer Company, subordination	Location
Vanadium acetylacetonate	MacKenzie Chem. Works, Inc.	Central Islip, N.Y.
	The Shepherd Chem. Co.	Cincinnati, Ohio
Vanadium carbonyl	Pressure Chem. Co.	Pittsburgh, Pa.
Vanadium chloride	Foote Mineral Co.	Cambridge, Ohio
Vanadium diboride	U.S. Borax & Chem. Corp. U.S. Borax Research Corp., subsid.	Anaheim, Calif.
	Ventron Corp. Alfa Products Div.	Beverly, Mass.
Vanadium hexacarbonyl	Ventron Corp. Alfa Products Div.	Beverly, Mass.
Vanadium neodecanoate	Mooney Chems., Inc.	Franklin, Pa.
Vanadium oxide	Associated Metals & Minerals Corp. Gulf Chem. & Metallurgical Co., div.	Freeport, Tex. Texas City, Tex.
	Union Carbide Corp. Mining and Minerals Div.	Hot Springs, Ark. Niagara Falls, N.Y.
Vanadium oxytrichloride (vanadyl trichloride)	Foote Mineral Co.	Cambridge, Ohio
	Stauffer Chem. Co. Specialty Chem. Div.	Weston, Mich.
	Union Carbide Corp. Mining and Metals Div.	Niagara Falls, N.Y.
Vanadium oxytrifluoride	Ozark-Mahoning Co.	Tulsa, Okla.
Vanadium pentafluoride	Ozark-Mahoning Co.	Tulsa, Okla.
Vanadium pentoxide (Vanadic anhydride)	Fairmount Chem. Co., Inc.	Newark, N.J.
	Foote Mineral Co.	Cambridge, Ohio
	Kerr-McGee Corp. Kerr-McGee Chem. Corp. subsid.	Soda Springs, Idaho

Table 1. (cont.)

Chemical	Producer	
	Company, subordination	Location
	Stauffer Chem. Co. Indust. Chem. Div.	Manchester, Tex.
	Susquehanna Corp. Susquehanna-Western, Inc., subsid.	Edgemont, S.D.
	Union Carbide Corp. Mining and Metals Div.	Rifle, Colo.
Vanadium sulfate (Vanadic sulfate) (Vanadyl sulfate)	City Chem. Corp.	Jersey City, N.J.
	Fairmount Chem. Co., Inc.	Newark, N.J.
	Foote Mineral Co.	Cambridge, Ohio
Vanadium tetrachloride	Foote Mineral Co.	Cambridge, Ohio
	Stauffer Chem. Co. Specialty Chem. Div.	Weston, Mich.
	Union Carbide Corp. Mining and Minerals Div.	Niagara Falls, N.Y.
Vanadium tetrafluoride	Ozark-Mahoning Co.	Tulsa, Okla.
Vanadium tetraoxide	City Chem. Corp.	Jersey City, N.J.
	Foote Mineral Co.	Cambridge, Ohio
Vanadium trichloride	Foote Mineral Co.	Cambridge, Ohio
	Stauffer Chem. Co. Specialty Chem. Div.	Weston, Mich.
Vanadium trifluoride	Ozark-Mahoning Co.	Tulsa, Okla.
Vanadium trioxide	Foote Mineral Co.	Cambridge, Ohio
Vanadium tris (acetylacetonate)	Foote Mineral Co.	Cambridge, Ohio
	Stauffer Chem. Co. Specialty Chem. Div.	Weston, Mich.
Vanadyl acetate	City Chem. Corp.	Jersey City, N.J.
Vanadyl acetylacetonate	MacKenzie Chem. Works, Inc.	Central Islip, N.Y.
Vanadyl naphthenate	The Shepherd Chem. Co.	Cincinnati, Ohio

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<div align="center">

Table 1

</div>

| Chemical | Producer | |
	Company, subordination	Location
Vanadyl phthal- ocyanine	The Shepherd Chem. Co.	Cincinnati, Ohio
Vanadyl p-tol- uene sulfonate	The Shepherd Chem. Co.	Cincinnati, Ohio

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B. Costs

All prices below are f.o.b. producer:

Table 2

1972 Prices of Vanadium & Compounds
Cost per Kilogram

Ammonium vanadate	$ 7.50
Sodium vanadate	$ 7.18
Vanadium pentoxide	$ 7.08
Vanadium metal (83% V)	$ 8.80 per kilogram contained V
Ferrovanadium (70-75% V)	$10.20 per kilogram contained V

C. Physical Properties

Physical properties of vanadium and important vanadium compounds are tabulated in Table 3.

Table 3
(3)

Physical Properties

Chemical	Specific Gravity	Melting Point $^\circ C$	Boiling Point $^\circ C$	Solubility Water g/100cc $^\circ C$	Comments
Vanadium	5.96	1890 ± 10	∿3000	insoluble	Malleable & ductile (pure)
Vanadium pentoxide	3.357[18]	690	d1750	0.8[20]	Rhombic crystals
Ammonium vandate	2.326	d200		0.52[15] 6.95[96]	Slightly hydro-scopic
Sodium vandate		630		21.1[25] 38.8[75]	Crystalline powder
Vanadium oxytrichloride	1.829	⁻77 ± 2	126.7	s(d)	

II.   PRODUCTION

A.   Quantities

The U.S. vanadium industry (see Section I.A.) is dependent on the extraction of the metal from domestic ores, with the exception of ferrovanadium, which is imported. The available vanadium is recovered as vanadium pentoxide usually with either ammonium or sodium vanadate acting as an intermediate. This activity is given in Table 4.

Table 4

Producers of Vanadium Pentoxide
(4)

Location	Company	Source of ore, type
Rifle, Colo.	Union Carbide	domestic-vanadium-
Soda Springs, Idaho	Kerr-McGee Corp.	uranium from by-product ferrophosphorus
Hot Springs, Ark.	Union Carbide	obtained in elemental phosphorus production
Hot Springs, Ark.	Union Carbide	Ark. vanadium ore
Edgemont, S. Dakota	Susquehanna-Western	domestic ores and/or
Wilmington, Del.	The Pyrites Co.	residues
Texas City, Texas	Gulf Chem. & Metal Corp.	domestic ores and/or residues
Moab, Utah	Atlas Corp.	domestic ores and/or residues
Urevan, Colo.	Union Carbide	taking over operation from Rifle milling operation. V liquor to be processed at Rifle to V_2O_5.

1.   Vanadium and vanadium pentoxide (4)

The United States is the largest producer and consumer of vanadium. The bulk of the output is a by-product of the accelerated uranium production program of 1952 to 1956. Vanadium continues to be a co-product of the carnotite ores mined for their uranium content. With the exception of substantial imports of ferrovanadium, the 1972 U.S. vanadium production statistics are as follows:

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ore and concentrate-recoverable V - 4,430 metric tons V content

V_2O_5 recovered.   - 4,750 metric tons as V

This represents essentially all of the vanadium available to U.S. industry, with the exception of imports of ferrovanadium. In 1972 ferrovanadium amounted to 525 metric tons of contained vanadium.

B. Process

1. Vanadium

Vanadium is not found in the free state; however, it is widely distributed throughout the earth in rather low abundance. It ranks twenty-second among the elements of the earth's crust. The production of the metal involves manufactured compounds as a basis rather than using the naturally occurring ores. Pure vanadium metal is very difficult to prepare because it combines readily with carbon, nitrogen, hydrogen, and oxygen and also forms solid solutions with some of its products.[5] Much of the vanadium produced is, therefore, not highly refined.

There are two commonly used methods for preparing vanadium metal. One involves the reduction of vanadium chloride with hydrogen or magnesium; the other involves the reduction of vanadium oxide with calcium, aluminum, or carbon. The oldest and most widely used method for producing vanadium metal on a commercial scale is the reduction of V_2O_5 with calcium. Recent developments include a two-step process involving the aluminothermic reduction of vanadium oxide combined with electron-beam melting. This method is capable of producing a purer grade of vanadium metal, of the quality required for the nuclear reactor program.

a. Calcium reduction

This method produces vanadium metal of about 99.5 per cent purity. Vanadium pentoxide and calcium are heated in a sealed bomb using calcium chloride as a flux. The calcium oxide formed serves as a thermal booster as well as a flux, resulting in both liquid metal and slage products. (In a modified process, iodine

is used as a flux.) This became the basis for the first large-scale
commercial process for producing vanadium. This process is rela-
tively inefficient with metal yields of only 75 to 80 per cent and
the amount of calcium reductant required 50-60 per cent over that
theoretically needed.

b. Aluminothermic process

Vanadium metal is under consideration as a fuel-
element cladding material for liquid-metal fast-breeder reactors.
To meet the more stringent requirements for this application the
U.S. Atomic Energy Commission developed the aluminothermic process.
This involves reacting vanadium pentoxide with high purity aluminum
in a bomb, forming vanadium-aluminum alloy. The alloyed aluminum and
dissolved oxygen are removed in a high-temperature, high vacuum pro-
cessing step to yield metal of greater than 99.9 per cent purity.

c. Electrotransport technique

The highest purity vanadium prepared to date is
prepared by an electrotransport technique. A high-density current
is passed through a small rod of electrolytically refined metal. At
1700-1850C, the interstitial solute atoms such as carbon, oxygen, and
nitrogen migrate to the negative end of the bar. Using this tech-
nique, vanadium with less than ten ppm of carbon, oxygen, and nitro-
gen has been prepared.

2. Vanadium pentoxide and sodium and ammonium vanadate[6]

The first stage in processing of most vanadium ores
is the production of an oxide concentrate for all ore sources. The
vanadium-bearing ores are generally crushed, ground, screened and
mixed with a sodium salt such as NaCl or Na_2CO_3. The mixture is
roasted at about 850C, converting the oxides to a water soluble
sodium metavanadate, $NaVO_3$. This is extracted by leaching with
water, the pH adjusted to between two and three with sulfuric acid
which precipitates sodium hexavanadate. $Na_4V_6O_{17}$ (red coke). The
mixture is fused at 700C to a dense black product that is sold as
technical grade vanadium pentoxide containing a minimum of 86 per cent

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V_2O_5. Further purification may be obtained by dissolving the vanadium in an aqueous solution of sodium carbonate and precipitating the iron, aluminum, and silicon impurities by pH adjustment. Then NH_4Cl is added to precipitate ammonium metavanadate, which is calcined to give V_2O_5 greater than 99.8% purity.

In the case of carnotite, a uranium-vanadium ore, sulfuric acid is used directly on the raw ore or the ore may be given an initial roast followed by successive leaching with water and dilute HCl or H_2SO_4. The uranium and vanadium are then separated by liquid-liquid extraction techniques. The pH and oxidation states are carefully controlled during extraction and stripping.

3. Ferrovanadium

The raw materials, either technical-grade V_2O_5, vanadium ore, or slag, are reduced with carbon, ferrosilicon or aluminum to yield a product whose vanadium content may vary from 35 to 80 per cent. The grade of product desired and the consumer use determine the choice of reducing agent.[6]

The vanadium alloy can be purified and consolidated by two different methods. In one procedure the brittle alloy is crushed and heated in a vacuum at 1790C to sublime out most of the aluminum, oxygen, and other impurities. The presence of aluminum facilitates removal of the oxygen, making this process superior to the calcium process. Electron-beam melting of pressed compacts of the vanadium sponge produces further purification and consolidation of the metal.

The other procedure involves direct electron-beam melting of the vanadium aluminum alloy regulus. Two melting steps are required before the desired levels of aluminum and oxygen are reached in the final ingot. These procedures produce comparable purities. Ingots weighing up to 225 kilograms have been prepared by this process, using dried electron-beam melting of the alloy.

Refining of vanadium. Vanadium can be purified by
one of three methods: iodide refining (van Arkel-de Boer process),
electrolytic refining in a fused salt, or electrotransport. The
first method has produced metal of greater than 99.95 per cent pur-
ity. In this process an impure grade of vanadium is reacted with
iodine at 800-900C. to form vanadium diiodide.

> "The volatized iodide is thermally decomposed and depos-
> ited on a hot filament at about 1300C. The refining step
> is carried out in an evacuated and sealed tube. The major
> impurities removed in the process are the gaseous elements
> and those metals that form stable, nonvolatile iodides. Van-
> adium metal containing five ppm nitrogen, 150 ppm carbon,
> and 50 ppm oxygen has been prepared in this way."[6]

The U.S. Bureau of Mines has developed an electro-
lytic process for purifying "crude" vanadium involving the cathodic
deposition of vanadium from an electrolyte consisting of a solution
of VCl_2 in a fused KCL - NiCl eutectic. The vanadium content of
the mixture is between two and five per cent; the operating temper-
ature of the cell is between 650 and 675C. This method has pro-
duced metal crystals or flakes of up to 99.95 per cent purity.

a. Reduction with carbon has been replaced by
other methods in recent years. In fact, vanadium carbide has replaced
ferrovanadium as the vanadium additive in steel. Union Carbide
Corp., for example, markets a product containing 85 per cent vana-
dium, 12 per cent carbon, and 2 per cent iron, called Carvan. Car-
van is produced by the solid state reduction of V_2O_5 with carbon in
a vacuum furnace.

b. The production of ferrovanadium by reduction
with silicon is used to a limited extent. This is a two-stage pro-
cess where technical-grade V_2O_5, ferrosilicon, lime, and fluorspar
are heated in an electric furnace to produce an alloy with about 30
per cent vanadium, but undesirable large amounts of silicon. The
addition of more V_2O_5 and lime extracts most of the silicon or else
forms a vanadium-silicon alloy by reaction of V_2O_5, silica, and coke
in the presence of a flux in an arc furnace. The primary metal is

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then reacted with V_2O_5 to produce ferrovanadium. A silicon process recently developed by Foote Mineral Co. is now used commercially to produce large quantities of ferrovanadium. A vanadium silicide alloy with less than 20 per cent silicon is made in a submerged-arc electric furnace by reacting vanadium-bearing slags with silica, flux and a carbonaceous reductant and then refining it with V_2O_5. This is then reacted with a molten vanadiferous slag in the presence of lime to produce a ferrovanadium alloy called Solvan.

c. Aluminum reduction for making ferrovanadium involves a highly exothermic reaction. A mixture of technical-grade V_2O_5, aluminum, iron scrap, and a flux are charged into an electric furnace and the reaction between Al and V_2O_5 is initiated by the arc. The temperature is controlled by adjusting the size of the particles and the feed rate of the charge, by using reduced material, or by replacing some of the aluminum with a milder reductant such as calcium carbide, silicon or carbon ferrovanadium of up to 80 per cent. The well-known thermite reaction may also be used to produce vanadium. Here, V_2O_5 and iron oxide are reduced by aluminum granules in a magnesite-lined steel vessel or in a water-cooled copper crucible. The reaction is initiated by use of a barium peroxide-aluminum ignition charge.[6]

Ferrovanadium is a general class of alloys, the composition of which depends on the intended application. The compositions of commercial grades of ferrovanadium are listed below.[20]

Product Grade	% vanadium
Iron Foundry	38-42
Grade A, open-hearth	50-55
Grade B, crucible	50-55 and 70-80
Grade C, primos	50-55 and 70-80
High-speed	50-55 and 70-75
Special Grade	50-55 and 70-75
Open-hearth	50-55
Foundry	50-55

C. Estimate of Yearly Vanadium Release

An estimate of the yearly rate of vanadium release to the environment is presented below:

Source	Yearly Rate Of Release (kkg)	Comments
Mining and Processing of Vanadium Ores	50	Mostly (90 per cent) to atmosphere
Combustion of Fuel Oil	22,000	17,000 to atmosphere; 5,000 to landfill
Combustion of Coal	6,635	5,000 to landfill or dump; 1,635 to atmosphere
Metallurgical Usage	1,535	Atmosphere
Other Mineral and Ore Mining and Processing	100	Rough estimate; most available for leaching
Miscellaneous	25	Rough estimate
Total	30,345	Roughly 65 per cent to atmosphere initially; remainder to land; essentially all soluble oxides

Because of the solubility of the vanadium oxide in water, most of the vanadium entering the environment can be expected to enter the waters. This amount cannot be estimated at present due to lack of knowledge concerning lifetimes of vanadium oxides in the air, leaching from landfills and dumps, etc.

More detailed data on vanadium content of the atmosphere, oceans, etc. may be found in Section IX. World-wide data and localized U.S. data on vanadium emissions in Section IX appear to support the estimations presented above.

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III. USES

A. Vanadium and its Compounds

The major use for vanadium and its compounds and alloys is in various types of steels. The end use and consumption of vanadium materials for 1972 are given below. Table 6 presents a detailed summary of current vanadium usage.

Table 5

Vanadium Consumption
(5)

End Use	Vanadium Consumed, 1972 (metric tons)
Steel:	
Carbon	572
Stainless & Heat Resisting	28
Full alloy	800
High-strength low alloy	1,873
Tool	563
Cast irons	55
Superalloys	15
Other alloys:	
Welding & alloy hard-facing rods and materials	10
Nonferrous alloys	321
Others (including magnetic)	18
Chemical & Ceramic Uses:	
Catalysts	134
Miscellaneous and unspecified[1]	171
Total	4,740

[1]includes end uses not listed to avoid disclosing company confidential data.

Table 6   Table of Uses
(4,7)

Compound	Use	Purpose	Comments
Vanadium	Nonferrous alloys	The titanium "6-4" alloy (6% Al-4%V) is becoming increasingly important in supersonic aircraft where strength-weight ratio is important	Vanadium and aluminum impart high temperature strength to titanium
		Vanadium foil can be used as a bonding material in cladding titanium to steel	
	Other	Small amounts used for experimental and special purposes	
Vanadium oxide	Catalyst for organic and inorganic compounds	V_2O_5 is important to the manufacture of sulfuric acid, conversion of naphthalene to phthalic anhydride, propane to acrylonitrite, acetylene to phthalic anhydride	One of the most important commercial compounds of vanadium
			In 1972 consumed 440 metric tons of vanadium
	Potential use in afterburners of automobiles	Will reduce hydrocarbon content of the exhaust	
Vanadium oxide & metavanadates	Manufacture of printing inks	Catalyze the formation of resinous black pigments from tar oils	Some of the quick drying inks use ammonium metavanadate
			Important commercial compound of vanadium
			In 1972 ammonium vanadate consumed 33 metric tons of vanadium

Table **6** Table of Uses
(cont.)

Compound	Use	Purpose	Comments
Vanadium halides	Catalyst in olefin polymerization	$VOCl_3$, VCl_4, and VCl_3 used in the copolymerization of ethylene and propylene	Primary industrial use
	Source of other vanadium compounds		One of the most important commercial vanadium compounds
Ferrovanadium	Steelmaking	Improves certain physical characteristics of steel	Accounts for the bulk of the vanadium compounds use
		Refines the grain structure and increases the hardening range of low-alloy steels	In open-hearth steel, added to ladle
			In electric-furnace steel added before tap or to ladle
			In 1972 contained 85% of total vanadium consumed in U.S., 4,050 metric tons
Other vanadium alloys	Special steels		Ex: vanadium-aluminum alloy (85% V, 15% Al) used in manufacture of titanium metal alloy
	Other alloys		

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B. Consumption Trends and Potential Future Uses

Domestic demand for vanadium in 1972 was almost nine per cent higher than demand in 1971, but still below the peak demand in 1969. Research is underway into the use of vanadium metal as a possible structural material for fast breeder reactors. Also under study is an electrotransport technique for the extraction and purification of vanadium from raw materials. This method may reduce interstitial impurities to less than five ppm by weight. The outcome of research into the use of vanadium pentoxide catalyst in an effort to reduce the smog-contributing components of automobile exhaust fumes may be yet another factor in future vanadium use.[4]

A modest rise is expected in the use of vanadium as a minor additive in various types of steels, cast irons, and special alloys. Substitutes for vanadium may be employed for practically every case, but unless a severe vanadium shortage develops, this is not likely to happen. An increase of greater proportions is predicted for two comparitively new steel-industry uses: high-strength low-alloy steels and the continuous casting of steel alloys and billets. Niobium could be substituted for vanadium, but would not be preferred unless costs or availability became factors. If increased use of these applications reaches the predicted levels, the production of vanadium may be taxed.[8]

A reliable estimate on the use of vanadium alloys as a cladding for fuel elements in fast breeder reactors is not possible. Molybdenum is also under consideration for this application. Neither of these materials is ideal and each must also compete with austenitic stainless steels. The final choice will be based on which has the fewer number of undesirable features. Another factor which must be considered is the position technologically, ecologically, and politically of the breeder reactor itself. The final decision may be more than ten years away. In anticipation of a favorable decision for the use of vanadium, however, government stockpile of vanadium pentoxide should be increased and upgraded to limit the contents of deleterious elements such as hydrogen and boron.[8]

Vanadium sources are expected to change in the future. The largest supplier may be the vanadium-bearing slags, a by-product of the iron industry which uses iron ores with a small percentage of vanadium. Thus the output of vanadium and the economics of iron production could be inter-related. The economically attractive production of vanadium as a co-product of uranium may play a large part in future vanadium supply since increased prospecting activity is expected in the search for new sources of uranium.[8]

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IV. CURRENT PRACTICES

A. Transportation and Handling

1. Vanadium pentoxide and ammonium-metavanadate are packaged in standard 50 kg fiber drums or shipped in bulk. They should be stored in a cool, dry location. A nose or mouth respirator should be used when handling large quantities.

2. Vanadium oxytrichloride is shipped in tank cars, tank trucks and cylinders. It must be stored in a cool, dry location. Skin contact must be avoided and the fumes should be regarded as an acid gas and respiratory system irritant.[9] Vanadium trioxychloride is classified as a corrosive liquid by the Interstate Commerce Commission. This is the only commercially significant vanadium product which requires special handling. In transport, the chemical is identified with a white label, is not accepted on passenger planes and no more than one liter is permitted on cargo planes.[10]

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V. ENVIRONMENTAL CONTAMINATION

A. Overall Appraisal

The potential sources of contamination from use, production, disposal and other sources are discussed below. In most cases the analyses suffer from the scarcity of pertinent information.

The only environmental problem that could be discovered may be produced by the combustion of residual fuel oil, and possibly of coal. Local potential problems around plants and in working areas are mentioned in the course of subsequent discussions. Environmental contamination from vanadium has not appeared to be a serious problem.

B. From Use

1. Metallurgical Uses

Small quantities of vanadium are used in steelmaking processes. The content in alloy steels normally ranges from 0.1 to 0.5 per cent, but in some cases may be as much as four or five per cent. The high temperatures involved in these operations cause the emission of some of the vanadium compounds into the atmosphere. The amounts are small and easily controlled. The vanadium pentoxide is melted before its use as additive or alloying agent, producing some pentoxide vapors. The primary source of environmental contamination from vanadium compounds is from emissions to the atmosphere. A recent study estimated emission factors based on observations, stack samples, contacts with operating personnel, and chemical analysis of particulates.[11] The following values were obtained:

Vanadium emissions, kilograms

Steel furnaces

Blast furnace	0.7/1,000 metric tons of pig iron produced
Open-hearth	2.5/1,000 metric tons of steel produced
Basic-oxygen	0.2/1,000 metric tons of steel produced
Cast iron	0.11 per metric ton of charge
Non-ferrous alloy	6.0 per metric ton of vanadium processed

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A 1965 Russian article reports that in "vanadium"-pig iron production a highly disperse aerosol contains among other disperse phases, 0.5-0.6 per cent of trivalent vanadium. Other constituents of the aerosol produce more harmful effects. Similarly, although harmful aerosols are generated in pig-iron converters, the trivalent vanadium present is not the bad actor.[12]

C. From Production and Disposal

1. Mining and processing of ore

Vanadium ore is usually mined in conjunction with the mining of another metal. The primary metal involved has changed over the years. Recent emphasis is on uranium recovery. This has caused some shift in vanadium mining and processing techniques. As a rule, drilling and blasting are required to loosen the ore which is then loaded on trucks and conveyed to the mills. The ores are normally subjected to dry grinding, mixed with lime and salt, and roasted. The product from the roaster, sodium vanadate, is leached out by a variety of methods and the vanadate precipitated out and fused to yield vanadium pentoxide. During various stages of this processing vanadium, compounds do escape into the atmosphere. Records of these are not available, so estimates must be made where possible. Based on observations and consultations with plant personnel, vanadium atmospheric emissions during mining and processing are estimated at abou⁺ .5 кg per metric ton of vanadium handled.[11] Since only overall vanadium production is available, only the total emissions can be calculated. Spot contamination at individual mining and processing sites was estimated at 45 metric tons for 1972.

At various stages in the processing, material is often left uncovered and exposed to the elements. Soluble vanadium compounds leak into the environment as a result of rain or ground water: from the comminuted mineral prior to extractive treatment; from the hot water leaching process to remove sodium metavanate which is about 90 per cent efficient leaving the residue with soluble vanadium compounds; from residues from roasting and leaching

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of carnotite ore, vanadiferous clays and vanadium enriched slags when heaped on the ground or used as landfills.[21]

2. Production of ferrovanadium

In the production of ferrovanadium from vanadium pentoxide, high temperatures are required to melt the pentoxide for either use in the electric reduction furnaces or in chemical reduction processes. A vapor is formed of vanadium metal.[11] This is primarily a problem in the working area rather than as a source of environmental pollution.

3. From inadvertent sources

By far the greatest sources of environmental contamination are inadvertant ones. Specifically, the combustion of oil and coal produce atmospheric emissions in the form of particulates. These constitute the bulk of the vanadium that gets into the environment. The residues from coal and oil furnaces also contain considerable vanadium.

a. Combustion of Oil

The largest single source of environmental vanadium contamination is atmospheric emissions from oil-fired furnaces. The furnace residues contain some vanadium, but may not constitute a significant source of environmental contamination since they are insoluble. This, however, is a potential source of vanadium which the U.S. is not expected to exploit.

Crude petroleum oils contain some vanadium, varying from less than one up to 1,400 ppm depending on the source of the crude oil. For domestic crude oils the extremes are 0.1 ppm vanadium content for New Mexican oil to 78.0 ppm for Montanan crude oil. The vanadium content of Venezuelan crude oil varies from 0.6 ppm for that from San Joaquin in eastern Venezuela to 1400 ppm for Boscan crude from western Venezuela. The vanadium content of crude oil from the Middle East, the other significant source of crude oil used in the U.S., varies from 3 ppm for that from Qatar to 114 ppm for Iranian (Gach Saran) crudes.[11]

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The distillation and refining of the crude oil leaves practically all of the vanadium in the bottoms, where residual fuel oil is obtained. The distillates, which include aviation and motor gasoline, jet fuels, automotive diesel fuels and home heating oil, contain barely detectable amounts of vanadium. The crude and product heavy fuel oils are the only significant petroleum sources of vanadium as potential environmental pollutants. The treatment of the heavy fuel oils to reduce the sulfur content does produce some reduction of vanadium content. A specific case in point is the treatment of a Venezuelan crude oil as indicated below:

	Sulfur Content, %	Vanadium, ppm
untreated	2.6	218
desulfurized	1.0	105
desulfurized	0.5	59

Vanadium emissions from the combustion of residual fuel oils should decrease as fuel oils are desulfurized in accordance with regulations that limit the sulfur content of fuel oils.[21]

A 1965 Soviet article states that workers cleaning out the ash residues at power generating stations where heavy petroleum fuel is burned suffered from "severe poisoning."

Power boilers designed to burn fuel oil are not usually equipped with air pollution control devices; however, combination coal-oil units utilize mechanical collectors and electrostatic precipitators which greatly reduce the atmospheric emission. During 1968 a study was made of the vanadium emission to the atmosphere caused by the combustion of fuel oil. The electric utilities used 28% of the total residual oil using 32% of air pollution control. Emission control for the total residual fuel oil used was estimated as only ten per cent. Based on ten per cent overall control, the vanadium emissions to the atmosphere during 1968 amounted to 17,000 kkg. The situation in 1972 was different in the greater quantities

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of fuel used, more use of air pollution control devices, and different mix of oil crude sources. These would tend to balance out the effects on vanadium emissions from the combustion of residual fuel oil. Consequently, the 1972 vanadium emissions are assumed to be 17,000 tons. This is conservative, considering that the quantity of residual fuel oil used has increased by eight per cent or more every year since 1968. A current estimate would be further complicated by the "energy crisis".

Little is known about the composition or size range of the vanadium compound particles emitted to the atmosphere. According to Davis, the particulates are mixtures of various vanadium oxides and mixed oxides of vanadium and sodium, nickel, and iron with a relatively wide spectrum of particle sizes.[11]

The limited use of pollution control devices in oil-fired furnaces was reported earlier, based on 1970 information. This situation may have changed by now. A 1965 article reported a technique which reduces stack emissions from an oil-fired steam generator through the addition of finely ground magnesium oxide to the fuel oil. By operating at low excess air together with this additive, a significant reduction in stack emissions resulted. The recovered boiler pit ash was rich in vanadium pentoxide. The residual oil used contained 250 to 1,000 ppm vanadium pentoxide and the boiler ash had 32 to 43 per cent vanadium pentoxide. The use of the additive increased boiler efficiency and reliability and reduced maintenance costs. The extent to which the magnesium oxide additive is used has not been determined; however, the technique is apparently well-known. It was discussed again in a paper given at the tenth meeting of the New England Air Pollution Control Association, Hartford, Connecticut, in 1966.[17]

b. Combustion of coal

The combustion of coal presents a significant source of environmental contamination from vanadium. In addition to the atmospheric pollution, considerable vanadium remains in the bottom

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ash. During the combustion of the coal about 35 per cent of the total ash is bottom ash. Tests have shown that the vanadium composition in the fly and bottom ashes are about the same. The vanadium content of domestic coals, as well as that in the resultant ash are given in Table 7.

Table 7
(18)

Concentration of vanadium in domestic coals

Coal source	V in ash, %	V in coal, ppm
Northern Great Plains	0.001-0.058	16
Eastern Interior Region		35
Appalachian Region		21
Texas, Colorado, North & South Dakota	0.01-.01	
West Virginia	0.018-0.039	
Pennsylvania (anthracite)	0.01-0.02	
Back Mountain Bed	0.11	176
Diamond Bed	0.09	92

The consumption of coal in 1972 is shown in Table 8. Using these data an estimate is prepared of the amount of vanadium entering the atmosphere with the fly ash and then remaining with the bottom ash. The following assumptions were made in arriving at this estimate:[11]

> Average vanadium content of coal-22.5 ppm
>
> 90 per cent application of control and 85 per cent efficiency of control

The estimate of vanadium emissions resulting from the burning of coal in the United States in 1972 was 1,635 metric tons.

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Table 8
(9)

1972 U.S. Consumption of Coal (thousand metric tons)

Type	Utilities	Metallurgical	Other Manufac. & Mining	Heating	Misc.	Total
Bitumi- nous & Lignite	314,000	83,300	60,600		8,060	466,000
Anthra- cite	1,430	683		2,650	553	5,330

Modern coal-fired power plants use efficient fly-ash control equipment which reduces considerably the emission of particulates. Cyclones and electrostatic precipitators are the most commonly used control equipment. Cyclones are more economical than electrostatic precipitators when additives which cause the formation of larger ash particles are used. The performances of two coal-fired power plants was compared with different fly-ash control equipment. In one case a cyclone-type separator only was used with resultant fly-ash recovery efficiency of 45 per cent, increased to 82 per cent with fly-ash reinjection. In another case a cyclone-type separator followed by an electrostatic precipitator was used to increase efficiency to 94 per cent.[17]

VI. MONITORING AND ANALYSIS

A. Monitoring

The Quality Assurance and Environmental Monitoring Laboratory of the National Environmental Research Center, state, and local organizations sample air in a systematic manner to obtain information about air quality in the United States. The last comprehensive survey was carried out in 1968-1969. Vanadium present in suspended particulate matter was one of the twelve metals whose concentration was checked. The analysis emphasized urban and rural differences. Spot checks of the air quality have also been conducted in such extreme regions as in highly industrialized areas and very remote regions such as the Artic or Antarctic. Also, many state and local government air pollution control organizations maintain some surveillance of air quality.

Representative information from APTD-1467 is given in Table 9. The data chosen was taken from a comparison of urban and rural vanadium emission characteristics and the effect of the seasons. Examination of the vanadium concentrations in the relative cold and warm seasons indicate that power and heat production with residual oil products and coal are responsible for the increased vanadium concentrations in northeastern cities and also possibly in rural areas. Manufacturing processes do not appear to contribute to the major portion of vanadium emissions.

Analysis of the air over relatively unpopulated areas of the earth, such as the Pacific Ocean area between San Diego and Honolulu, show some vanadium, but only about 1,000th of the amount found over populated areas. The vanadium found over a remote area of northwestern Canada is almost ten times that cited above. This suggests that in addition to vanadium particulates circulated through the air by the wind, some is injected into the atmosphere by wind erosion of rocks and soil.[12]

Monitoring of rivers and other waters for vanadium has not been reported.

Table 9
(19)

Vanadium Concentration in Atmosphere, $\mu g/m^3$

Urban Location	Maximum	Arith. mean	Quarterly Composite				Yearly Average
			1st	2nd	3rd	4th	
New Haven, Conn.	2.500	0.534	1.700	0.530	0.160	1.200	0.897
Wilmington, Del.	1.100	0.237	0.620	0.160	0.230	0.480	0.372
Oakland, Cal.	0.064		0.023	0.033	0.030	0.035	0.030
Mobile, Ala.	0.015		0.032	0.023	0.020	0.023	0.024
Los Angeles, Cal.	0.130		0.034	0.0	0.013	0.013	0.015
Baltimore, Md.	0.720	0.185	0.380	0.240	0.160	0.340	0.280
Nonurban Location							
Orange Co., Ver.	0.180 (highest)	0.041	0.074	0.027	0.020	0.093	0.053
Calvert Co., Md.	0.056	0.019	0.042	0.035	0.035	0.026	o.034

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B. Analysis

1. Vanadium (in air-borne materials)

a. A section of an air filter is rolled up into a cylinder and placed in a hollow graphited electrode, then enclosed in an atmosphere of oxygen. A condensed spark discharges to burn the filter sample and excite the spectra. An internal standard compensates for the small pieces of paper and ash which may be ejected during the process. The spectra are interpreted by using a photographic recording and the line intensities are measured with a microphotometer to provide greater precision. The limits of detection varied from 0.1-1 micrograms; the results agreed favorable with the more conventional technique of ashing the filter, mixing with a spectrographic buffer, and exciting in a D.C. arc.[20]

b. Vanadium in compounds can be measured by irradiating the samples with neutrons and counting the gamma rays emitted by the neutron capture product, 3.8-minute vanadium-52, (with lithium-drifted germanium gamma ray detectors) without prior chemical separation. In some cases, particularly in marine environments, gamma rays from sodium and chlorine may obscure those of vanadium-52, making it necessary to add a chemical step before counting sensitivity of the method. Values as low as 7×10^{-7} $\mu g/m^3$ have been observed in Antartica. Based on the sensitivity of this method of analysis, vanadium can serve as an indicator of wide-scale movement of particulates.[13]

2. Vanadium in solution

a. Atomic absorption spectroscopy in oxy-acetytene or nitric oxide-acetylene flames can be used for the analysis of vanadium in the range of 0.5-100 mg/l. With oxy-acetylene flames, vanadium is extracted as the cupferrate into a mixture of methyl isobutyl ketone and oleic acid and the organic phase aspirated to the flame. The sensitivity is 0.7 mg/l of vanadium in the organic phase. An extraction procedure eliminates potential interferences from a large number of both cations and anions. Excess cupferron must be

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added if Fe^{3+}, Sb^{3+} or Zn^{4+} are present since they are preferentially complexed. When nitrous oxide-acetylene flames are used, an aqueous solution of vanadium salts is aspirated directly and the sensitivity is improved by the use of methyl isobutyl ketone, Al^{3+} and diethylene glycol or diethyl ether.[22]

VII. CHEMICAL REACTIVITY

A. Environmental Reactions

In the massive state, vanadium is not attacked by air. At high temperatures it is oxidized via the lower oxides to form V_2O_5. It also reacts with nitrogen to form VN. Vanadium is quite resistant to corrosion from sea water.[6]

The catalytic property of vanadium compounds to oxidize sulfur dioxide to sulfur trioxide deserves mention. This property could present some problems in the combusion products of high vanadium and high sulfur oils.

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VIII. BIOLOGY
A. Content, Absorption and Excretion
1. Animals

Vanadium levels in human tissues are less than one microgram per gram ash, except in the case of the lungs, in which levels as high as 108 micrograms per gram ashed material have been reported.[23] Vanadium pentoxide is readily absorbed from the lungs into the bloodstream. Seven days after an intratracheal administration of vanadium pentoxide to rats, no vanadium remained in the lung. In a long-term inhalation experiment with rabbits, vanadium was detected not only in the lungs, but also in the liver and kidneys.[23]

The content of vanadium in the enamel and dentin of incisors from horse, fowl, cow, calf, and pig, and canines of pig and dog averaged 29.0 to 41.5 ppm. No significant species differences or differences between enamel and dentin were found.[24]

Subcutaneously injected vanadium-48 pentoxide deposited in higher concentrations in the dentin than in the enamel of developing rat teeth. The highest concentrations were found adjacent to ameloblasts.[25] Topical application of vanadium-48 pentoxide penetrated mainly into the enamel of human teeth, with some radioactivity reaching the cementum and dentin.[26]

Dietary vanadium reduced caries incidence and severity in the Syrian hamster fed a cariogenic diet.[27] A dose of ten ppm vanadium in drinking water had a slight cario-protective effect when administered to mother rats.[28] In another study, vanadium pentoxide in the drinking water not only did not reduce caries incidence in rats, but produced toxic symptoms and inhibition of growth.[29]

Strontium and vanadium fed together decreased caries prevalence in the teeth of rats on a cariogenic diet. Zinc plus vanadium stimulated increased uptake of phosphorus-32 into the teeth of these rats.[30]

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In man, vanadium taken orally in the form of sodium metavanadate was excreted in the feces largely unabsorbed. Within twelve days, 87.6% of the ingested vanadium appeared in the feces, and the remaining 12.4% was recovered in the urine. Rats receiving intragastric radiovanadium absorbed only about 0.5% of the dose.[23]

Humans treated with oral doses of ammonium vanadyl tartrate, 25 to 125 mg daily, excreted vanadium in their urine as long as they were maintained on the drug. Patients continued to excrete vanadium in their urine three weeks after the drug was withdrawn, but four to five weeks later, vanadium was no longer detectable in the urine. Less than 0.5% of a daily dose was excreted in a 24 hour urine specimen, except for one patient receiving 125 mg per day. About 1.2% of the daily dosage was found in his urine. Presumably the rest of the vanadium was not absorbed, and was, therefore, excreted in the feces.[31]

In rats receiving sodium metavanadate intraperitoneally, and in rabbits receiving intravenous injection, most of the vanadium was excreted by the kidneys. The ratio of renal to fecal excretion was 5:1. Sixty-one per cent of the vanadium was excreted by the kidneys within twenty-four hours. In two men injected intravenously with sodium metavanadate daily for six days, 81 per cent of the injected vanadium was eliminated in the urine within seven days after the last injection, nine per cent in the feces; ten per cent was presumably retained in the body.[23]

The rapidity of vanadium excretion should be considered when deciding the best time for the collection of blood samples and urine specimens from industrial workers. The kidney and bowel are apparently the only excretory pathways for vanadium.

The vanadium content of some tunicates of the order Phlebobranchia is shown in Table 10. The oocytes of Ascidia pygmaea are invaded by so-called "test" cells which possess a vanadium chromogen and which are derived from ameboid blood cells. A vanadium pigment is required for the synthesis of cellulose for the test in Ascidiidae species.

Table 10
(32)
Vanadium Content of some Tunicates

Source	%Vanadium	Species
Centrifuged bloodcells	1.45	Ascidia nigra
Eggs	0.017	Phalucia mamillata
Ash	0.19	Phalusia mamillata

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The vanadium organelles in the test cells which invade the embryo may serve as chromogen reserves. When the embryo metamorphoses, large amounts of polysaccharide for the test must be synthesized quickly. The stored chromogen provides the pigment necessary for the cellulose synthesis. [33]

In the tunicate, Ciona intestinalis, the accumulation of vanadium by the blood cells is preceded by a specific active absorption process. The single-cell-layer epithelium is the site of vanadium absorption. The absorption is highly temperature dependent and is inhibited by ouabain, phosphate and arsenate but not chromate, niobate, molybdate or iron. [34]

2. Plants

In plants, vanadium is absorbed by the roots into two compartments: a freely diffusible, exchangeable compartment and a nonexchangeable compartment. In absorption experiments the uptake of vanadium-48 as ammonium vanadate was studied in excised barley roots. Calcium was necessary for absorption, which apparently is not an active process requiring metabolic energy. The rate of absorption of vanadium was highest at pH 4.0, being twice the rate at pH 5-8. At alkaline pH's, less absorption was noted. The predominant ionic species of vanadate present at pH 4.0 are the vanadium (V) dioxide cation (VO_2^+) and vanadic acid (HVO_3). The presence of other anions at ten times the vanadate concentration produced no great inhibition of vanadium uptake. Dihydrogen phosphate, which produced the maximum inhibition, inhibited vanadium uptake by only 27 per cent.

B. Distribution

1. Animals

Ten to twelve per cent of the injected vanadium was retained in rabbits after two weeks and 74 to 84 per cent of that was detected in the skeleton. [23] Small quantities were also present in the kidney, liver, blood, spleen, lungs, adrenals, bone marrow, skin, and muscles.

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In rats injected with radioactive vanadium-48, at the end of four days, the tissues retaining the highest percentages of the injected dose were:

bone (9.9%), liver (6.2%), muscle (5.0%)

kidney '4.4%), and blood (4.4%)

The amount of vanadium is sharply reduced in the livers of patients who have died from tuberculosis.[35] The vanadium content of bone tumors of various types (osteoblastoclastoma, sarcoma, fibrous osteodystrophy, bone cysts, osteochondromas) is higher than in adjacent bone tissue.[36] Brain tumors will also accumulate vanadium.[23]

Vanadium is present in high levels in the blood of some tunicates in green blood cells called vanadocytes. The hemovanadin pigment, which contains nearly ten per cent vanadium in some species is thought to have an oxidation-reduction role, perhaps functioning as an oxygen carrier. The sea cucumber, Sticopus mobii, contains 1200 ppm vanadium as dried matter. A mollusc, Pleurobranchia plumula contained 150 ppm. Vanadium is probably absorbed from the sea water, plankton and from marine silts in these creatures.[23]

In higher plants, the average concentration of vanadium in dried material was one ppm, with the roots containing higher concentrations than the leaves. Root nodules of leguminous plants contained up to four ppm.[23]

The mushroom, Amanita muscaria, contains relatively large amounts of vanadium. Amavadin, a vanadium-containing compound, was isolated from this mushroom.[37]

C.. Growth and Nutrition

Although the precise purpose of vanadium is not known, the essentiality of vanadium for optimum growth in the rat has been demonstrated as shown in Table 11.[38] The addition of 10 to 50 micrograms vanadium as sodium orthovanadate per 100 grams of

Table 11
(38)

Growth Response of Rats to Varying Levels of Vanadium Supplements

(The total increase is given as the percentage
gain over a 21-to 28-day period)

Vanadium (µg/100 g of diet)	Unsupplemented Controls		Supplemented Animals		Total increase (%)	P
	Rats (No.)*	Average daily weight gain	Rats (No.)	Average daily weight gain		
1	7	1.05 ± 0.08	7	1.27 ± 0.10	21	†
5	16	1.04 ± 0.08	16	1.38 ± 0.08	33	∠.01
10	6	1.02 ± 0.14	7	1.38 ± 0.07	35	∠.05
25	14	0.87 ± 0.10	14	1.21 ± 0.09	41	∠.02
50	6	1.02 ± 0.14	7	1.49 ± 0.12	46	∠.02

* A total of 37 rats served as controls in five successive experiments.

† Not significant.

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feed in an "ultraclean", vanadium-free environment enhanced growth of rats by over 40 per cent. Other forms of vanadium had similar, but not as pronounced, effects. The levels of vanadium required for optimum growth effects are those normally found in the environment. In one study, a commercial laboratory ration contained 1.50 ppm fresh weight of vanadium.[39] A rat weighing 75 grams needs one to two micrograms vanadium per day (25 micrograms per 100 grams feed). A human with a weight of 75 kg has been estimated to consume two mg of vanadium per day.[40] Guinea pigs apparently also require vanadium in the diet. In guinea pigs fed a vanadium-free diet, a 50 per cent drop in the calcium and phosphorus contents of bone was observed. Also, an increase in the incidence of dental caries occurred.[23]

No positive effects on the growth of chicks could be demonstrated, but chicks tolerated 20 to 40 micrograms per cent vanadium in their diet.[41]

The requirement for vanadium in plants has been studied in many species, but its essentiality for growth in higher plants has not been conclusively demonstrated.[23]

Vanadium has an essential role in the green algae, Scenedesmus obliquies, where it stimulates photosynthesis and growth.[42]

Vanadium is about 50 per cent as effective as molybdenum in stimulating the growth of cultures of Azotobacter spp.[42] The iron requirement of Streptococcus cremoris can be satisfied with vanadium, but not with molybdenum, cobalt, zinc, copper or manganese.[43]

Vanadium is considered an indispensable trace element in the growth of the fungus, Aspergillus niger.[23]

D. Cytoxicity

Since the most likely route of vanadium exposure is through the lungs, the effects of particulates of various vanadium compounds on laved alveolar macrophages from rabbits were investigated in vitro.[44,45,46,47] Cell viability after a 20 hour exposure was reduced by 50 per cent at the following concentrations.[44]

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Vanadium Compound	LC_{50} (micrograms V per ml)
V_2O_5	13
V_2O_3	21
VO_2	33

Only about nine micrograms vanadium per ml as vanadium pentoxide was required to reduce cell viability by 50 per cent and to decrease total cell number by 70 per cent after 20 hours if the compound was first dissolved in the medium. The phagocytosis of polystyrene-latex spheres was reduced 50 per cent by six micrograms vanadium per ml as vanadium pentoxide.

Exposure of cells or cell-free sonicates to dissolved vanadium pentoxide at levels as high as 50 micrograms vanadium per ml produced only small changes in the specific activities of lysozyme or β-glucuronidase. Acid phosphatase in the cell-free system was 70 per cent inhibited by one microgram per ml.[44]

In another study, cell viability was reduced 50 per cent by 0.1 to 0.2 millimolar ammonium vanadate. Cadmium[+3] and vanadate were found to be 37 to 62 times more toxic for alveolar macrophages than nickel[+2], manganese[+2], or chromium[+3].[45]

The cell surfaces of control alveolar macrophage cultures and cultures exposed to 0.098 millimolar ammonium vanadate (5.0 micrograms vanadium per ml) were examined by electron microscopy.[46] The control cells exhibited intricate surface structure with numerous membrane processes extending upwards as well as attached to the substrate, but the vanadium-treated cells showed severe morphological damage. Most of the cells completely lost their processes, becoming unusually smooth; some developed bleblike structures or appeared to have broken open. When the macrophages were incubated with 0.20 millimolar vanadate (10 micrograms vanadium per ml), cell viability was reduced to 10 per cent, and a great number of cells were so completely destroyed that only remnants of their plasma membrane were seen.

Table 12
(47)

Concentrations of Metallic Ions Causing Reduction
in Viability to 50 Per Cent in Rabbit Alveolar
Macrophages and Human Lung Fibroblasts
(Strain WI-38) at 20 Hours

	Concentration of Metal, mM	
Metallic Ion	Rabbit Alveolar Macrophages	Human Lung Fibroblasts
Cd^{2+}	0.099 (0.062 - 0.151)*	0.126 (0.077 - 0.201)
VO_3^-	0.234 (0.169 - 0.428)	0.620 (0.481 - 0.842)
Ni^{2+}	4.17 (3.74 - 4.71)	2.83 (1.45 - 4.75)
Mn^{2+}	5.29 (4.46 - 6.35)	9.82 (7.37 - 14.7)
Cr^{3+}	5.48 (4.49 - 6.77)	11.3 (Estimated)

* 95% Confidence Limits are Shown in Parentheses.

Table 13
(47)

Concentrations of Metallic Ions Causing Reduction
in Uptake of Thymidine, Uridine,
and Leucine to 50 Per Cent in Human Lung
Fibroblasts (Strain WI-38) at 20 Hours

	Concentration of Metal, mM		
Metallic Ion	TdR-2-^{14}C Uptake	UdR-2-^{14}C Uptake	Leu-1-^{14}C Uptake
Cd^{2+}	0.444	See Note*	0.373
VO_3^-	0.009	0.017	0.044
Ni^{2+}	0.176	0.136	1.27
Mn^{2+}	0.396	0.098	7.56
Cr^{3+}	3.80	3.40	9.49

* A 50 Per Cent Response was not Observed over Concentration Range Studied, (0.004 - 0.089 mM).

In cultures of human lung fibroblasts, vanadium had effects on cell viability similar to the effects on rabbit alveolar macrophages.[47] The LC_{50} for the fibroblast cultures at 20 hours was 0.620 millimolar vanadate. At much lower concentrations, inhibition of the uptake of precursors of DNA, RNA, and protein synthesis were observed in the human lung fibroblasts. Tables 12 and 13 are summaries of viability data and effects on uptake.

E. Metabolic Effects

Vanadium apparently exerts effects on sulfur metabolism, energy metabolism, and lipid metabolism, as well as on various enzymes.[23]

In normal sulfur metabolism, cysteine, cystine, and methionine are the three sulfur-containing amino acids. Normal sulfur excretion occurs as sulfate in the urine, taurine in the bile or keratin in hair and nails. Two cysteines, with free thiol (-SH) groups, are easily and reversibly oxidized to cystine, which can be decarboxylated to beta-mercaptoethylamine, a precursor of coenzyme A. Methionine can be converted to cysteine. Coenzyme A is important in various transacetylation reactions in the body, including those involved in lipid and sterol synthesis.[23]

Rats fed 25 to 1000 ppm vanadium pentoxide exhibit a marked reduction in the cystine content in the hair.[48] Supplementary feeding of methionine somewhat alleviated the effects. The cystine content of the fingernails of vanadium workers was significantly reduced compared with that of healthy people and people suffering from a number of diseases.[23]

Significant reductions in Coenzyme A in the livers of rats injected or fed sodium metavanadate occurred. The mitochondrial fraction of rat liver homogenate contained only 52.5 per cent of the normal amount of coenzyme A and 86 per cent of the normal amount of thioctic acid, a precursor of a coenzyme necessary for pyruvic acid decarboxylation.[23]

Vanadium chloride and sulfate activate pyridoxal phosphate and the enzyme, desulphydrase, which converts cysteine to

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hydrogen sulfide, ammonia and pyruvic acid. Since cysteine is the precursor of cystine and Coenzyme A, the vanadium stimulation of this enzyme may account for these observed effects.[23]

The effects of vanadium on cholesterol and other lipid metabolism are somewhat ambiguous, depending on the amount, compound, and mode of vanadium administration. Vanadium sulfate intraperitoneally injected into rats reduced the incorporation of radioactive acetate into liver cholesterol by more than 50 per cent both *in vivo* and *in vitro*.[49] A decrease in both liver cholesterol and phospholipid content, but not serum cholesterol was observed in rabbits fed 100 ppm vanadium pentoxide.

In rabbits, the addition of 100 ppm vanadium as vanadium pentoxide to the standard diet reduced liver-free cholesterol and phospholipid content by 2/3 and 1/3 respectively. Plasma cholesterol showed no significant changes. The addition of 50 ppm vanadium to a one per cent cholesterol diet restricted the hypercholesterolemia observed in the absence of vanadium. Vanadium feeding after dietary hypercholestorolemia promoted a faster return to normal cholesterol levels compared with rabbits fed no vanadium.[50]

In rabbits fed high-cholesterol diets, 50 per cent less cholesterol was found in the aortas of rabbits subsequently fed a diet containing 0.05% vanadium sulfate compared with rabbits maintained on standard laboratory rations. The serum cholesterol levels in the two groups showed little difference. The almost complete mobilization of cholesterol from tissues other than the aorta occurred in both groups, which raised serum cholesterol levels and probably reduced hepatic cholesterol synthesis. Cholesterol synthesis in the controls probably returned to normal activity when serum cholesterol levels approximated normal after non-aortic tissue clearance was completed, resulting in only very slow mobilization of aortic cholesterol. Since hepatic cholesterol synthesis is apparently inhibited by vanadium, the aortic cholesterol in the vanadium-treated rats would be mobilized more rapidly. Similar effects have been observed in chickens.[23]

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Vanadium inhibits cholesterol synthesis at several steps in the pathway. These steps involve Coenzyme A transfer reactions.[23]

Serum cholesterol levels in a group of middle-aged male vanadium workers were significantly lower than those of a control population, 205 mg per 100 ml compared with 228 mg per 100 ml. The vanadium workers excreted four times more vanadium in the urine as did the controls.[23]

Oral administration of vanadium as diammonium oxytartrato-vanadate to five male medical students at a dosage of 100 to 125 mg daily resulted in reduced fecal sterol excretion, 20 per cent lower free and total cholesterol, and a rise in serum triglycerides.[51] These effects returned to normal when vanadium was removed.

Somewhat different effects were observed in rabbits which received an intragastric dose of cholesterol of 1 g per kg as a five per cent solution in cholesterol together with vanadium sulfate injections.[52] Rabbits receiving a dose of 1 mg per kg vanadium sulfate had much lower blood cholesterol levels at various intervals after treatment than did animals receiving solely cholesterol. With repeated daily injections of vanadium sulfate for five days, a slight retardation in cholesterol level occurred after injection, but the degree of hypercholesterolemia subsequently reached the control levels, and by 240 hours, when control cholesterols had returned to normal, the cholesterol level in vanadium-injected animals was twice the control level. The injection of 2.5 mg per kg of vanadium twice a day did not cause an initial decrease in blood cholesterol. Rather, an increase began at 96 hours after injection, peaking at 120 hours, and falling to normal after 14 days.

Several effects on lipids and metabolism were observed after subcutaneous injection of 5 to 30 mg vanadium as ammonium vanadate per kg.[53] Liver and serum triglycerides increased prominently 4 to 48 hours after injection, the increase depending upon the dose. Serum total cholesterol fell, reaching a minimum 24 hours

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after injection. Serum free fatty acids decreased gradually from
4 to 72 hours after injection. The decreases in serum cholesterol
and free fatty acids did not correlate with dose of vanadium. The
activities of both serum glutamate oxalcacetate transaminase and
serum glutamate pyruvate transaminase both increased according to the
dose of vanadium. No distinct changes in serum total cholesterol,
phospholipids or triglycerides could be detected in patients receiving
25 to 125 mg of ammonium vanadyl tartrate daily for several weeks.[31]

Vanadium exerts effects on various sites of energy meta-
bolism. Hyperglycemia has been observed in dogs intravenously per-
fused with lethal amounts of sodium metavanadate. In yeast vana-
dium increases the hydrolysis of hexose diphosphates. In male rats
given carbon-14-labelled glucose orally and ammonium metavanadate
intraperitoneally, significantly reduced amounts of carbon-14 diox-
ide were collected compared with controls, reflecting reduced oxi-
dation of glucose.[54] The addition of sodium metavanadate to guinea
pig or rat liver suspensions increased the rate of oxygen consumption.

Vanadate apparently uncouples the oxidative phosphoryl-
ation by which ATP is produced via aerobic respiration and the elec-
tron transport system.[55] In the liver mitochondria of chicks fed
25 ppm vanadium as ammonium metavanadate, although the amount of
oxygen consumed increased slightly, the rate of ATP synthesis dropped
by 22 per cent and the P/O ration dropped by 31 per cent. *In vitro*
addition of vanadate (one millimolar) to liver mitochondria pro-
duced more pronounced decreases in ATP production and P/O ratio. The
use of intermediate substrates, succinate or β-hydroxybutyrate, did
not prevent the uncoupling. ATPase activity was at the least twenty
times the control activity in the presence of vanadate. Both ATP-[14]
C-ADP exchange and ATP-[32]phosphorus exchange were inhibited by vana-
date, the latter being more sensitive.

Vanadium salts influence the oxidation of various tyrosine and tryptophan derivatives, including 5-hydroxytryptamine, or serotonin.[23] Serotonin has a variable effect on blood pressure when administered to animals. Vanadium salts have been shown to activate the oxidation of serotonin to 5-hydroxyindole acetic acid (5-HIAA) by monoamine oxidase of guinea pig liver. In hypertensive patients, excretion of vanadium was three times the average normal level of 0.6 micrograms per liter. If serotonin and similar amines are supposed to have a pressor effect, vanadium deficiency, resulting in decreased monoamino oxidase activity, may result in occasional vasospasms being sustained.

The enzymatic oxidation of tyrosine to dopa is accelerated by vanadium, copper, cobalt, and nickel. A quantitative relationship between metal concentration and tyrosine oxidation has been found.[56]

Vanadium plays a definite, but obscure role in hemopoiesis.[23] Anemic rats return to normal in one-third to one-half the usual six weeks time after initiation of iron supplementation when 0.05 mg of vanadium was also given. Normal young rats fed 0.25 mg iron and 0.05 mg vanadium daily developed reticulocytosis and a faster increase in red blood cells and hemoglobin occurred than with iron alone. Vanadium gluconate given to rabbits intravenously for 40 days also produced significant reticulocytosis and slowly developing polycythemia. The stimulatory effect of vanadium on human hemopoiesis has not been demonstrated. Vanadium may function through the activation of pyridoxal phosphate since pyridoxine deficiency may lead to a reduction in red blood cells and hemoglobin.

Vanadium compounds inhibit the enzymes which catabolize acetylcholine.[23] One hundred ml of ten millomolar metavanadate weakly inhibited the pseudocholinesterase of horse serum.[57] Vanadyl sulfate significantly decreased cholinesterase activity in rat brain tissue preparations. No accompanying changes in free or bound acetylcholine occurred at the same time.[58]

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Radioactive vanadium-48 uptake was studied in selected organs and liver subcellular particles of rats.[23] No significant difference was seen in the rate or amount of uptake by liver particles of the three oxidation states of vanadium during the first four hours. This amount of radioactivity was retained by the particles up to 96 hours, at which time other organs contained 10 to 84 per cent of their ten minute contents. At 96 hours, 46 per cent of the vanadium-48 had been excreted in the urine, and nine per cent in the feces. The amount of radioactivity in the liver supernatant fraction had decreased from 57 per cent to eleven per cent, but the mitochondrial and nuclear fractions increased from approximately 14 to 40 per cent. The microsomal fraction changed little.

Doses of 0.03 to 10 mg per kg vanadyl sulfate did not affect the liver's ability to acetylate sulfanilamide, but doses of 15 to 20 mg per kg did. Phenylacetic, phenylethylacetic and diphenylacetic acids inhibit sulfanilamide acetylation to different degrees. Small doses of vanadyl sulfate did not potentiate their effects, but high doses strengthened the effects of phenylacetic and diphenylacetic only.[59]

In plants, vanadium appears to participate in the absorption and reduction of nitrogen.[23] The addition of vanadium (40 ppm) to the nutritive solution on sand cultures enhanced the absorption of nitrate nitrogen.[60] When vanadium and molybdenum were present together, nitrate reduction was increased, as was the free amino acid content of the plants.[61] Vanadium, but not molybdenum, enhanced nitrite reduction, thereby resulting in increased protein synthesis.[62] In aquatic cultures of the cucumber, Cucumis sativus, vanadium caused rapid reduction of nitrate in the leaves and also stimulated the accumulation of chlorophyll and carotene.[63]

In experiments conducted with the ribosomal fractions of pea cotyledons, vanadium and molybdenum reduced ribonuclease activity.[64] Stabilization of the ribosome structure may be

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accomplished by these metal ions. The ribonucleic acid component of the ribosomes contained most of the ions accumulated by the ribonucleoprotein particles.

The mushroom,[43] Amanita muscaria, accumulates vanadium and produces a vanadium-containing compound named amavadin.[37]

The ability of vanadium to partially replace molybdenum in stimulating nitrogen-fixation by Azotobacter strains may be due to vanadium incorporation into the nitrogenase complex promoting increased stability and more efficient utilization of molybdenum-starved cells.[42]

Induction of the penicillinase of Bacillus licheniformis can apparently be achieved with vanadate, as well as molybdate and tungstate.[65]

Commercial RNA from yeast was examined by electron spin resonance, and some of the spectral absorptions could be identified as vanadium and iron.[65] Iron complexes tended to form with the RNA molecules easily precipitable by ethyl alcohol, whereas the vanadium complexes formed more readily with smaller RNA molecules and surface groups on the macromolecules.

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IX. ENVIRONMENTAL EFFECTS

A. Environmental Content, Transportation, and Contamination

Natural and manmade sources contribute to the vanadium content of the atmosphere.[13] Natural sources of atmospheric vanadium are marine aerosols, produced by the bursting of bubbles at the sea surface, continental dust from wind erosion, and a small contribution from volcanic ash. The ash from the burning of fuel oil and crude petroleum is the primary source of manmade vanadium atmospheric pollution.

Assuming that no specific mechanism for vanadium enrichment in marine aerosols or continental dust is operating, the amounts of atmospheric vanadium arising from natural sources may be estimated relative to the amounts of other elements known to arise from those sources.[13] Thus, to estimate the upper limit of the marine aerosol contribution of vanadium in a given location, one can multiply the observed sodium and chlorine concentrations in the atmospheric particulate by the vanadium to sodium and vanadium to chlorine ratios in sea water. Likewise, the upper limit of vanadium from dust and volcanoes could be estimated by assuming all of the observed iron arises from those sources, and multiplying the iron concentrations by the vanadium to iron ratio. Other elements, such as magnesium, can be used to check results obtained in this way.

Using this method for predicting vanadium in the air of windward Hawaii, the conclusion reached was that only 31% of the airborne vanadium arose from natural sources.[13] The predicted vanadium from natural sources for rural Canada amounted to 100 per cent of the vanadium present, as would be expected in an area far removed from man's activities. Tables 14 and 15 illustrate the calculations.

This technique has been applied to the atmospheric vanadium in several U.S. cities using the continental dust component only.[13] The results are summarized in Table 16.

Table 14
(13)

Predicted Vanadium Concentrations for
Windward Hawaii from Natural Sources

Source	Element Used as Basis	Concn of Element, ng/meter^3	Predicted V Concn, ng/meter^3	
Marine aerosols	Na	5300	0.0010 }	0.0010
	Mg	670	0.0010	
Continental dust	Fe	38	0.099 }	
	Mn	0.86	0.103 }	0.114
	Al	101	0.141	
			Total 0.11	

Observed	Acid soluble	0.24 + 0.10
	Insoluble	0.11 $\mp$ 0.08
	Total	0.35 + 0.13
Predicted natural/observed		31%

Table 15
(13)

Predicted Vanadium Concentrations for
Rural Canada from Natural Sources

Source	Element Used as Basis	Concn of Element, ng/meter^3	Predicted V Concn, ng/meter^3	
Marine aerosols	Na	44	0.0000085 ⎫	0.000005
	Cl	13	0.0000019 ⎭	
Continental dust	Fe	210	0.55 ⎫	
	Mn	6.7	0.80 ⎪	
	Al	186	0.26 ⎬	0.73
	Sc	0.11	1.57 ⎪	
	La	0.20	0.50 ⎭	
	Zn	15.4	31 ⎫	Anomalous
	Sb	0.25	25 ⎭	
			Total	≈ 0.73

Observed   0.72 + 0.50
Predicted natural/observed ≈ 100%

Table 16
(13)

Predicted Levels of Atmospheric Vanadium
Originating from Natural Sources

Locality	Number of Samples	V Concentration (nanograms per m^3)		% from Natural Sources
		Predicted	Observed	
Honolulu	12	1.64	3.4 ± 2.3	47%
Los Angeles	18	5.2	12.5 ± 8	42%
San Francisco	9	2.9	6.2 ± 2.1	47%
Northwest Indiana	25	9.4	8.2 ± 2.8	114%
New York City	270	3.9	170	2%
New York City	150	2.0	1190	2%
Boston	numerous	0.4-3.0	600	0.07-0.5%

In the highly industrialized cities of the northeastern
United States, the apparent large contribution of man's activities
to atmospheric vanadium can be explained by the combustion of resid-
ual fuel oil high in vanadium content. The contribution of coal and
residual fuel oil to atmospheric vanadium have been estimated using
the elemental ratios. Table 17 shows that the predicted ranges of
vanadium in the oil easily account for the observed concentration
of airborne vanadium in the Boston area.

An examination of the particle size distribution of vana-
dium in the Boston area supports the contention that most of the
atmospheric vanadium originates from fuel combustion.[13] Vanadium
is preferentially associated with the smallest particles collected,
as would be expected for the high temperature of oil combustion fol-
lowed by condensation of the vaporized material. Elements such as
iron or aluminum arising principally from coal combustion and conti-
nent dust are found in highest concentrations on the largest partic-
ulates in air. In the Boston area, although 342,500 metric tons of
coal were burned in 1966, this represented only twelve metric tons
of vanadium. The 1386 million gallons of residual oil burned con-
tained 4100 metric tons of vanadium.

The estimated annual rates of global injection of vanadium
into the atmosphere from petroleum and natural sources are summar-
ized in Table 18.

The amount of vanadium in sea water is about 5×10^{-8} per
cent.[14] Industrial effluents may provide sources of vanadium for
water pollution.[15]

The amounts of vanadium in some water samples from various
parts of the world are shown in Table 19.

Vanadium is present in many foods. Values are given in
Tables 20 and 21.

Table 17
(13)

Predicted Vanadium Contributions from
Various Sources in the Boston Area

Source	Element Used as Basis	Concn of Element, ng/meter^3	Predicted V Concn, ng/meter^3	
Continental	Fe	1400	3.6	
	Mn	30	0.4	
	Al	1430	2.0	0.4-3.0
	Sc	0.29	4.1	
	Co	2.4	30	
Coal	Fe	1400	3.5	
	Mn	30	2.3	
	Al	1430	2.6	2-9
	Co	2.4	8.6	

			Venezuelan Crude	Residual Oil
Residual oil	Co	2.4	1,400	7,000
	Zn	380	16,300	550,000
	Sb	9	3,650	157,000
	Se	4	1,210	11,600
Range of Predictions			1200-16,300	7,000-550,000

Observed Range	90-2400
Average	600

Table 18
(13)

Estimated Annual Rates of Global Injection of Vanadium
into the Atmosphere from Petroleum and Natural Sources

Source	Total Amount Injected or Consumed Annually, 10^6 metric tons	Assumed V Concn, ppm	Total V Injected, metric tons
A. Natural			
Soil and rock dust	200	135	2.7×10^4
Volcanic debris	50	200	10^4
Sea salt	80	0.15	12
Total natural			3.7×10^4
B. Petroleum	2000	50	2×10^4

Table 19
(16)

Vanadium in Some Water Samples

| | Vanadium in water samples | |
	Range	Average
	ppm	ppm
Water samples from:		
Atlantic Ocean	5.0×10^{-3} - 4.4×10^{-3}	4.6×10^{-3}
Baltic Sea	3.5×10^{-3} - 5.0×10^{-3}	4.0×10^{-3}
Lake Maelar (Sweden)	0.12×10^{-3} - 0.24×10^{-3}	0.1×10^{-3}
Small Lake (Stockholm, Sweden)	0.45×10^{-3} - 0.48×10^{-3}	0.46×10^{-3}
Drinking water from:		
Boston	0.9×10^{-4} - 1.3×10^{-4}	1.1×10^{-4}
New York	0.3×10^{-4} - 0.5×10^{-4}	0.4×10^{-4}
Chicago	0.8×10^{-4} - 1.9×10^{-4}	1.4×10^{-4}
Oslo (Norway)	0.4×10^{-4} - 0.7×10^{-4}	0.6×10^{-4}
Stockholm (Sweden)	4.9×10^{-4} - 5.0×10^{-4}	5.0×10^{-4}
Kiruna (Sweden)	0.7×10^{-4} - 0.9×10^{-4}	0.8×10^{-4}
Trosa (Sweden)	8.3×10^{-4} - 8.6×10^{-4}	8.5×10^{-4}

Table 20
(16)

Vanadium in Some Animal Specimens

Specimen	Ash wt. Mean	Wet wt	
		Range	Average
	ppm	ppm	ppm
Calf liver, Stockholm	0.51	0.83×10^{-2}-1.18×10^{-2}	1.0×10^{-2}
Calf liver, Boston	0.11	0.18×10^{-2}-0.30×10^{-2}	0.24×10^{-2}
Calf flesh, Stockholm	$< 10^{-4}$		
Calf teeth, Stockholm	$< 10^{-4}$		
Calf bone, Stockholm	$< 10^{-4}$		
Pork, Stockholm	$< 10^{-4}$		
Fresh trout, soft tissues	0.06	0.03×10^{-2}-0.07×10^{-2}	0.04×10^{-2}
Fresh mackerel, soft tissues (North Sea)	0.20	0.15×10^{-2}-0.36×10^{-2}	0.26×10^{-2}
Fresh mackerel, bone	2.9	1.1 -4.1	2.0
Sardines, Sweden	0.28	0.40×10^{-2}-1.32×10^{-2}	0.86×10^{-2}
Sardines, Norway	0.20	0.42×10^{-2}-1.0×10^{-2}	0.70×10^{-2}
Sardines, Portugal	0.46	0.81×10^{-2}-1.8×10^{-2}	1.3×10^{-2}
Fresh milk, Boston	0.24×10^{-3}	0.71×10^{-4}-0.94×10^{-4}	0.84×10^{-4}
Fresh milk, Chicago	0.16×10^{-3}	0.66×10^{-4}-0.88×10^{-4}	0.77×10^{-4}
Fresh milk, New York	0.13×10^{-3}	0.52×10^{-4}-0.96×10^{-4}	0.74×10^{-4}
Fresh milk, Stockholm	0.48×10^{-3}	0.76×10^{-4}-1.45×10^{-4}	1.1×10^{-4}
Fresh milk, Oslo (Norway)	0.20×10^{-3}	0.70×10^{-4}-0.92×10^{-4}	0.80×10^{-4}
Fresh milk, Goteborg (Sweden)	$< 10^{-4}$		

Table 20
(16) (cont.)
Vanadium in Some Animal Specimens[1]

Specimen	Ash wt. Mean	Wet wt	
		Range	Average
	ppm	ppm	ppm
Dried skim milk:			
Carnation[2] (U.S.A.)	0.50×10^{-3}	1.8×10^{-4}-2.8×10^{-4}	2.3×10^{-4}
Starlac[3] (U.S.A.)	0.48×10^{-3}	1.3×10^{-4}-2.3×10^{-4}	1.9×10^{-4}
Famos[4] (Sweden)	$< 10^{-4}$		
Semper[4] (Sweden)	$< 10^{-4}$		
Lobster, meat (North Sea)	16.1	3.2×10^{-2}-5.3×10^{-2}	4.3×10^{-2}
Gelatin (Sweden)	2.5	3.9×10^{-2}-5.0×10^{-2}	4.4×10^{-2}

[1]Values represent ten samples of each of the specimens listed.

[2]Carnation Food Company, Los Angeles.

[3]Borden's Food Products, New York.

[4]Semper Company, Stockholm, Sweden.

Table 21
(16)
Vanadium in Some Fruits and Vegetables[1,2]

	Ash wt Mean	Dry wt Mean	Wet wt	
			Range	Average
	ppm	ppm	ppm	ppm
Dill	4.6	0.84	$0.12 \quad -0.15$	0.14
Lettuce	2.8	0.58	$1.9 \times 10^{-2}-2.3 \times 10^{-2}$	2.1×10^{-2}
Parsley[3]	29.5	4.52	$0.50 \quad -1.11$	0.79
Cucumbers	0.38	5.6×10^{-2}	$1.6 \times 10^{-3}-2.3 \times 10^{-3}$	2.1×10^{-3}
Radishes[3]	7.9	1.26	$5.22 \times 10^{-2}-5.21 \times 10^{-2}$	5.21×10^{-2}
Strawberries[3]	0.66	3.1×10^{-2}		
Wild strawberries	0.72	4.1×10^{-2}		
Red whortleberries	0.54	1.02×10^{-2}	$1.5 \times 10^{-3}-1.7 \times 10^{-3}$	1.6×10^{-3}
Apples[3]	0.33	0.86×10^{-2}	$1.89 \times 10^{-3}-1.25 \times 10^{-3}$	1.10×10^{-3}
Tomatoes[3]	0.041	0.53×10^{-3}	$0.16 \times 10^{-4}-0.38 \times 10^{-4}$	0.27×10^{-4}
Cauliflower	0.093	1.09×10^{-3}	$0.72 \times 10^{-4}-0.83 \times 10^{-4}$	0.77×10^{-4}
Potatoes[3]	0.093×10^{-2}	0.64×10^{-2}	$0.75 \times 10^{-3}-0.89 \times 10^{-3}$	0.82×10^{-3}
Pears	$<10^{-4}$			
Carrots[3]	$<10^{-4}$			
Common beets	$<10^{-4}$			
Peas, frozen	$<10^{-4}$			

[1]Values represent 10 samples of each item.

[2]All samples were taken from the Stockholm area.

[3]Of these species five samples of each grown in New Hampshire (U.S.A.) and five samples grown in Rhode Island (U.S.A.) were also analyzed. The results from these 70 samples were, in general, somewhat lower than those tabulated for the fruits and vegetables grown in Sweden.

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B. Bioaccumulation

Vanadium is readily absorbed through the lungs, and to a small extent, by the small intestine.[23] Animals do not retain vanadium for long periods of time. Doses of vanadium are rapidly excreted in the urine and the feces, and complete clearance of the vanadium from the animal usually occurs two to three weeks after vanadium administration has ceased. Vanadium tends to accumulate in the bones, perhaps substituting for phosphorus. In the adult red tail swordfish, Xiphosphorus helleri, vanadium-48 accumulated in the bones, fins, skin, and liver after a three day exposure to the radio-active element.[39]

Some marine organisms, notably the tunicates, accumulate vanadium. Apparently they can concentrate vanadium from sea water with the pharyngeal mucous sheath. Vanadium serves as the metallic element in the tunicate blood, existing complexed to pyrrole rings in a structure similar to bile salts. Small amounts of vanadium have been found in other phyla of marine organisms as well.[14] The percentage of vanadium of the dry weight is summarized in Table 22. Assuming the dry weight is only one per cent of the weight, many of the organisms still contain 10 to 100 times the vanadium concentration as that reported for sea water (5×10^{-8}%).

Plants take up vanadium through the roots especially at acidic pH's. Toxic amounts can be accumulated if insufficient iron is present. Some plants such as Cleomes spp. appear to be vanadium accumulators.[67]

Radioactive vanadium-48 was taken up by some vegetables grown in soil boxes containing vanadium-48 trichloride and cold sodium vanadate. Vanadium-48 was taken up into both leaf and root vegetables and into the leaves of root vegetables.[39]

Table 22
(14)
The Vanadium Content of Some Marine Organisms

Group	Species	Vanadium ($\%$ in dry matter)
Thallophyta	Marine algae	5×10^{-3} (dry ash)
Porifera	Halichondria sp.	3×10^{-3} (dry ash)
	Choanites ficus	1.7×10^{-4}
Coelenterata	Cyanea capilluta	5×10^{-4}
	Metridium dianthus	4×10^{-3}
	Anemonia sulcata	2.3×10^{-4}
Bryozoa	Plumatella fungosa	1.68×10^{-3}
Echinodermata	Stichopus mobii	0.123
	S. japonicus var. armatus	trace
	S. tremulus	5.7×10^{-3}
	Cucumaria lefevrei	1×10^{-4}
	Asterias glacialis	3×10^{-4}
	A. rubens	9×10^{-4}
	Paracentrus lividus	8×10^{-5}
	Brissopis lyrifera (shell)	9×10^{-4}
Mollusca	Sepia officinalis	trace
	Helix sp.	5×10^{-5}
	Loligo sp.	4×10^{-5}
	Mytilus edulis	1.2×10^{-4}
	Crassostrea sp.	1.3×10^{-4}
	Patella vulgata	1×10^{-5}
Crustacea	Carcinus moenas	4×10^{-5}
	Lepas anatifera	1.2×10^{-4}

Table 22
(14) (cont.)

The Vanadium Content of Some Marine Organisms

Group	Species	Vanadium (% in dry matter)
Tunicata	Ascidia mammillata (England)	0.17
	Ascidia mentula (England)	0.186
	Ascidia mentula (Atlantic)	0.0982
	Ascidia mentula var. rudis (Eng.)	0.145
	Ascidia aspersa scabra (England)	0.112
	Ciona intestinalis (Sweden)	0.062
	Ciona intestinalis (England)	0.040
	Ciona intestinalis (Atlantic)	0.0166
	Botrylloides schlosseri (Atlantic)	8×10^{-4}
	Pyura savigni (Atlantic)	7×10^{-4}
	Dendrodoa grossularia (Atlantic)	1×10^{-3}
	Distomus varialosus (Atlantic)	6×10^{-3}
	Molgula manhattensis (Atlantic)	3.2×10^{-3}
	Didemnum candidum (Atlantic)	2.6×10^{-4}
	Didemnum maculosum (Atlantic)	3×10^{-3}
	Morchellium argus (Atlantic)	3.7×10^{-4}
	Parascidia turbinata (Atlantic)	8.3×10^{-4}
	Parascidia aureolata (Atlantic)	3.9×10^{-4}
	Aplidium pallidum (Atlantic)	4.3×10^{-4}
	Clavelina lepadiformis (Atlantic)	7×10^{-4}
Pisces	Scyllium caniculata	4×10^{-6}
	Gadus merlangus	1.4×10^{-5}
	Scomber sp.	2.2×10^{-5}
	Trigla sp.	1×10^{-5}

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X. TOXICITY

A. Humans

1. Occupational exposure

Many occupations involve exposure to aerosols and dusts of vanadium compounds.[15,23] Mining and milling of vanadium-containing ores such as patronite involve health risks from handling of the "inactive" ore before roasting, the "active" ore after the roast, and in the final processing. Atmospheres in a Peruvian operation contained 0.18 to 58.82 mg vanadium per cubic meter of air in the active processing, the highest concentrations of 12.77 mg per cubic meter occurring in the final stages of processing.

Dust concentrations in the production of vanadium pentoxide and vanadates at one time might have amounted to 6.5 mg vanadium pentoxide per cubic meter.[23]

The production of ferrovanadium and other vanadium alloys, vanadium catalysts, and pure vanadium compounds such as vanadium trioxide, vanadium chloride, and vanadium carbide, also involve high vanadium atmospheres.

Occupational exposure to vanadium is not limited to production of vanadium compounds.[23] Since most crude oils contain up to 0.072 per cent vanadium by weight, occupations involving exposure to ash and fumes derived from these fuels may involve hazardous vanadium levels. The ash of these oils is about 65 per cent vanadium, with some ship's boiler ash containing 82 per cent vanadium. Men who clean such oil-fired burners are likely to be exposed to rather high concentrations of vanadium dust containing all valences of vanadium compounds.

The symptoms of exposure to vanadium-laden dusts are generally acute; relief occurs promptly when the irritant is removed.[23] Upper respiratory irritation characterized by bronchospasm, paroxysmal cough, expectoration, chest constriction, and shortness of breath is the most frequent symptom. Accompanying conjunctivitis, rhinitis, soreness of pharynx, and purulent eye discharges often occur.

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Persons suffering from these symptoms are particularly susceptible to respiratory diseases such as pneumonia. Other symptoms reported include a feeling of weakness, elevated blood pressure, palpitation, skin pallor, tremor of fingers and arms, and irritation of face and arms. Hypersensitivity to vanadium has been induced after acute exposures. A greenish-black tongue, with septic teeth and lingual furring, is indicative of vanadium exposure, but not necessarily poisoning. The green color is believed to be due to the deposition of quadrivalent vanadium in the tongue and gums. Systemic poisoning due to overexposure to vanadium dusts can occur. The symptoms include nausea, vomiting and diarrhea, central nervous system disturbances, cardiovascular disturbances, anemia, hysteria, melancholia, dimness of vision, and retinitis.

Recommended limits for maximum permissible concentrations adopted at the American Conference of Governmental Industrial Hygienists in 1961 are:[23]

Contaminant	Maximum Permissible Concentration (mg per m^3)
Vanadium pentoxide (dust)	0.5
Vanadium pentoxide (fume)	0.1
Ferrovanadium (dust)	1.0

Exposures lower than 0.5 mg per cubic meter of vanadium pentoxide may produce eye, nose and throat irritation.

2. Other studies

Human volunteers were exposed to vanadium pentoxide dusts for eight hours.[68] The dust concentrations were 0.1 to 0.25 mg vanadium pentoxide per cubic meter of air, with 98 per cent of the particles less than five microns in diameter. Considerable mucus was formed in the airways within 24 hours, followed by coughing lasting from 48 to 72 hours. A concentration of 1 mg per cubic meter induced coughing after only five to seven hours exposure, which persisted for eight days. No pathological changes were found in the lungs, in blood chemistry or morphology, or in the cystine content of hair and nail clippings.

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Intravenous injection of sodium tetravanadate (20 mg as vanadium pentoxide) into men produced, at various times, salivation, lacrimation, disappearance of the pulse, cessation of breathing, diarrhea, and a fall of up to three degrees in body temperature.[23] It is believed that a 30 mg dose would kill a 70 kg man. A well-tolerated dose of the hexavanadate was 60 mg vanadium pentoxide injected by any route, although intramuscular injection produced some pain and swelling. At a toxic intravenous dose of this salt (100 to 125 mg vanadium pentoxide), nausea and vomiting, with albumin and cylindrical casts in the urine were produced.

In a study in which patients were treated with daily oral doses of ammonium vanadyl tartrate, 25 to 125 mg, the only signs of toxicity were diarrhea and cramping when the large doses were given. The small amounts of vanadium absorbed by the intestine probably account for this, as well as man's tolerance to vanadium and rapid excretion.[31]

B. Mammals

1. Acute toxicity

The degree of exposure and the valence of the vanadium compounds affect the severity of symptoms.[15] The median lethal doses of vanadium compounds in relation to valency as determined in rats are shown in Table 23. Toxicity of some vanadium compounds is shown in Table 24.

The LD_{50} for a single intraperitoneal injection of sodium metavanadate was 0.29 mg per kg in the mouse, and 0.22 mg per kg in the rat.[69]

The lethal doses of various vanadium compounds in various animals are summarized in Table 25.

Symptoms of acute toxicity manifest two distinct modes of action: first, a central effect on the nervous system causing drowsiness with convulsions, followed by gradual paralysis of respiration and motion; second, an effect on the alimentary tract causing abdominal pain, with diarrhea and bloody stools.[23]

Table 23
(15)

Effect of Valence on Vanadium Toxicity to Rats

Vanadium Compound	Valence	Median Lethal Dose (mg V/kg)
ammonium vanadate (NH_4VO_3)	5	10
vanadium trichloride (VCl_3)	3	23
vanadium diiodide (VI_2)	2	68

Table 24
(70)
Acute Toxicity of Some Vanadium Compounds

Vanadium Compound	Animal	Mode of Administration	Toxic dose (mg per kg)
vanadic acid, monosodium-salt	rat	intraperitoneal	4 (LDLo)
vanadium dichloride	rat	oral	540 (LD_{50})
vanadium (II) oxide	mouse	intraperitoneal	9 (LD_{50})
vanadium pentoxide	human	inhalation	$0.1 mg/m^3/8$ hr (TCLo
vanadium tetrachloride	rat	oral	160 (LD_{50})
vanadium trichloride	rat	oral	350 (LD_{50})
trichlorooxo vanadium (vanadyltrichloride)	rat	oral	140 (LD_{50})
vanadyl chloride	rat	oral	140 (LD_{50})

LD_{50}: lethal dose to 50 per cent of animals tested

LDLo: minimum lethal dose

TCLo: lowest toxic concentration

Table 25
(23)

Lethal Doses, in mg V_2O/kg

Rabbits were injected intravenously, other animals subcutaneously

	Rabbit	Guinea-pig	Rat	Mouse
Colloidal V_2O_5	1-2	20-28		87.5-117.5
Ammonium metavanadate	1.5-2.0	1-2	20-30	25-30
Sodium orthovanadate	2-3	1-2	50-60	50-100
Sodium pyrovanadate	3-4	1-2	40-50	50-100
Sodium tetravanadate	6-8	18-20	30-40	25-50
Sodium hexavanadate	30-40	40-50	40-50	100-150
Vanadyl sulphate	18-20	35-45	158-190	125-150
Sodium vanadate		30-40	10-20	100-150

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Peripheral vasoconstriction in the spleen, kidney, and intestine occurs after sublethal doses of metavanadate. This occurred in the intact animal, after cervical cord section or decapitation, and in isolated perfused organs illustrating that this was a direct effect of the vanadium.[23]

Hyperperistalsis, bronchospasm, diuresis, hypothermia, and hemorrhagic gastroenteritis are other acute symptoms.

Both kidney and liver damage are evident in vanadium poisoning. Glomerular hyperemia and necrosis of the convoluted tubules were observed; congested and fatty degeneration of the liver occurred. Cortical and medullary hemorrhaging in the suprarenals were seen. The nuclei of the cortical pyramidal cells in the brain showed less distinctly on staining, indicating that the tigroid substance was diminished.[23]

Death in rabbits was due to respiratory failure, thought to be due to the direct action of vanadium on the central respiratory center.[23]

Toxic effects of ingested vanadium occur in rats fed 25 ppm vanadium as sodium metavanadate. Animals receiving eleven and 22.5 ppm for twelve weeks appeared normal throughout the test period. A dose of 92 ppm was quite toxic and 368 ppm usually caused death within 10 weeks. A dose of 6 mg of vanadium as meta-or ortho-vanadate, administered by stomach tube as 2 mg per day, was lethal to rats weighing less than 100 g. Bleeding from the nose and intestine, marked diarrhea, dyspnea, and paralysis of the hind legs occurred before death.[23]

Vanadium apparently has no cumulative action, and daily doses can be given indefinitely as long as the tolerance level is not exceeded. Typical symptoms of acute vanadium poisoning were observed in the autopsy of poisoned animals.

A lethal inhalation dose of 500 mg per cubic meter vanadium pentoxide to cats caused death in 23 minutes. Approximately 205 mg per cubic meter vanadium pentoxide for seven hours was lethal to rabbits. Seventy mg per cubic meter was lethal to rats if

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prolonged over 20 hours. Typical symptoms were gastroenteritis, pneumonitis and pulmonary edema.[23]

One of the mechanisms for detoxification of vanadium compounds by the body may be acidification. The LD_{85} dose (9.1 mg vanadium per kg) of sodium vanadate for mice at pH 7 was reduced to essentially zero when the pH of the injection was 1.8. Increasing the injection pH to 12.5 did not increase the 85 per cent toxicity level.[71]

Alterations of bicarbonate reserve by injection of ammonium chloride to cause acidosis, or sodium bicarbonate to produce alkalosis also affected vanadium toxicity. In ammonium chloride treated mice, the lethality of a sodium vanadate injection was reduced from 60 per cent to 25 per cent. The mortality was increased from 40 per cent to 80 per cent in sodium-bicarbonate-treated mice.[71]

Ascorbic acid and calcium disodium EDTA injections have been demonstrated to antagonize the toxic actions of vanadium compounds.[72]

2. Chronic toxicity

Daily subdermal injections (0.6 to 1.0 mg per kg for 300 days) or oral doses (6 to 11 mg per kg) to rats produced changes in many organs.[73] The lungs presented the picture of pneumonia and numerous macrophages appeared. In the liver, fatty degeneration, vacuolization, and reticular necrosis of liver cells were observed. Vacuolization of the kidney tubules and thyroid epithelial cells of the follicle was observed. The follicles of the spleen were atrophied, with macrophages occurring in the organ. Fatty degeneration of the adrenals and degeneration of the pancreatic parenchyma occurred in some animals. No changes occurred in the ovaries, but the canal of the epididymis was dilated, with some flattening of the epithelial cells.

Chronic poisoning by inhalation of trivalent vanadium (40 to 70 mg per m^3 for two hours daily) resulted in a fall in hemoglobin level from 75 per cent to 67 per cent, a 33 per cent drop in white blood cells, a drop in albumin and increase in globulin, resulting in a halving of the albumin: globulin ratio, an increase in

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amino acids (cysteine, arginine, histidine) in the blood, a ten per cent rise in nucleic acid and a 30 per cent decrease in serum sulfhydryl groups over the eleven-month experimental period.[15] A 50 per cent fall in vitamin C in the blood also occurred. The symptoms described above were evident.

Vanadium trichloride proved to be more toxic than vanadium trioxide. Vanadium pentoxide was three to five times as toxic.[15]

3.   Teratogenicity, carcinogenicity and mutagenicity

No evidence of carcinogenicity, mutagenicity, or teratogenicity of vanadium compounds has been found. Some cases of apparent allergic response after occupational exposures have been noted.[23]

C.   Birds

Vanadium administered as calcium vanadate at a level of 30 ppm vanadium significantly depressed the growth of young chicks.[74] In one study, ten ppm vanadium in the feed reduced feed consumption, but no differences in blood phosphorus or bone ash levels were observed.[75] No mortality occurred with 20 to 120 ppm vanadium as calcium vanadate in the diet, but 30 per cent and 100 per cent mortality occurred with 200 and 400 ppm, respectively.[74]

In white Leghorn laying hens, a level of 30 ppm vanadium as ammonium vanadate depressed egg production.[76] Egg hatchability was depressed at levels of 50 ppm. Body weight tended to decrease as the level of vanadium increased, and albumin quality in the eggs was markedly depressed by 15 to 20 ppm in the diet.

A reduction in the toxic effects of 20 ppm vanadium as ammonium vanadate fed to chicks was accomplished by the replacement of five per cent sucrose in a sucrose-fish diet with degossypolized cottonseed meal or the addition of five per cent dehydrated grass.[77] The addition of 0.25-0.50 % ascorbic acid to the ration also reduced the vanadium toxicity. Since vanadyl chloride ($VOCl_2$) with a valence of four was as toxic as ammonium vanadate, the ascorbic acid effect is not likely to be a reduction in the oxidation state of vanadium.[74]

D. Plants

Little information has been found on the toxicity of vanadium to plants. Vanadium in sand culture of <u>Medicago sativa</u> at a level of 500 ppm reduced plant growth. The effect on the roots was particularly severe.[78]

Toxicity symptoms appear in soybean, flax and peas with 2.5 to 5 ppm vanadium in the nutrient solution. Vanadium did not counteract the symptoms of iron-deficiency chlorosis. The reduction of the standard iron concentration by 1/3 to 1/2 accentuated the toxicity of vanadium. Raising the iron supply to 20 or 30 ppm reduced the vanadium contents of the shoot and counteracted the vanadium toxicity.

When the iron supply was low, vanadium reduced the phosphorus content of the shoot in soybean and flax, but increased the phosphorus content in peas.[79]

E. Microorganisms

The growth of <u>Mycobacterium tuberculosis</u> diminished when the concentration of vanadium pentoxide in the medium was three micrograms per ml. An increase in vanadium from three to five micrograms per ml reduced growth even more, and growth ceased with concentrations over five micrograms per ml.[35,80]

Vanadyl sulfate has demonstrated therapeutic usefulness in the treatment of tuberculosis in white mice and guinea pigs.[81]

F. Results of Personal Contacts with Medical Personnel

A total of 74 toxicologists and medical examiners throughout the United States were contacted by telephone or letter with regard to their professional acquaintance with incidences of accidental poisoning attributed to vanadium or its compounds. None of the 31 responses cited such an incident.

XI. STANDARDS AND CURRENT REGULATIONS

Threshold Limit Values (TLV): [82] (recommended)

Vanadium pentoxide dust: 0.5 mg/m^3 as V_2O_5
Vanadium pentoxide fume: 0.1 mg/m^3 as V_2O_5

Based on German experience the recommended maximum concentration in work areas are: [11]

V dust (probably as V_2O_5) - 0.5 mg/m^3
V smoke (probably as V_2O_5) - 0.1 mg/m^3
ferrovanadium dust - 1 mg/m^3

In the USSR, the maximum allowable concentrations adopted for occupational exposure to industrial hazards are: [83]

V_2O_5 "condensation" aerosol - 0.1 mg/m^3
V_2O_5 "comminution" aerosol - 0.5 mg/m^3
Vanadates and vanadium chlorides - 0.5 mg/m^3
Ferrovanadium and vanadium-aluminum alloys - 1.0 mg/m^3
Vanadium carbide - 4.0 mg/m^3

There is some difficulty, especially in foreign articles, in distinguishing between "regulation" and "standard". The foreign regulations presented below must therefore be interpreted with reservations.

One German article[84] states that " the maximum allowable working place concentration of vanadium in air is uniform for all industrial nations." Maximum allowable emission concentrations, however, differ. In the USSR the maximum is 0.002 mg/m^3; in the Federal Republic of Germany a maximum allowable emission concentration of 0.001 mg vanadium/m^3 is under discussion.

Only one article, a 1960 Russian piece, expressed concern with the vanadium content of water. The writer suggested a vanadium concentration of 0.1 mg/1 as a maximum permissible limit for water basins.

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XIII.   SUMMARY AND CONCLUSIONS

A.   Summary

The United States is the largest producer and consumer of vanadium. The material is produced at present mainly as a by-product of the urenium industry. However, this could change in the future as vanadium is available from several other sources.

The release of vanadium to the environment from man's activities is estimated to be over 30,000 metric tons per year, of which about two-thirds arises from the combustion of residual fuel oil. The amount entering the environment from processing and use of vanadium material is small in comparison to the above. In view of the solubility of vanadium oxide, much of the vanadium in wastes can be considered to enter the waters over a finite period of time. Natural sources of vanadium include wind erosion of rocks and transport from soils.

Toxicity of vanadium and its compounds to humans varies from moderate to acute. There has been little apparent adverse effect of the vanadium in the environment, but occupational hazards exist and are well-documented. Also, residue from oil-fired furnaces often contains dangerous levels of vanadium compounds. The Threshold Limiting Value for concentration of vanadium compounds has been set as follows: vanadium pentoxide dust, 1.5 mg/m^3; vanadium pentoxide fume, 0.1 mg/m^3; and ferrovanadium dust, 1.0 mg/m^3.

Vanadium has marked effects on human metabolism in that it reduces chloresterol production and affects production of various enzymes and sulfur-containing amino acids. Vanadium ingested by humans appears to be excreted largely unabsorbed. No evidence was found of teratogenicity, carcinogenicity or mutagenicity occasioned by vanadium. Some apparent allergic response was observed to develop after occupational exposures.

Vanadium compounds may be absorbed through the lungs and, to a small extent, by the intestine. Most of the orally ingested vanadium is excreted in the feces. Elevated urinary vanadium levels reflect vanadium exposure, and systemic vanadium is rapidly eliminated from the body by the kidneys. Vanadium interferes with sulfhydryl group metabolism and reduces hypercholesterolemia.

Toxicity varies with valence state and mode of administration.

The effects of vanadium on higher animals are quite similar to those on humans. In the case of rats, the need for vanadium for optimum growth has been shown. The effects of vanadium on animal metabolism are also similar to that in humans. Toxicity in animals is greater than in humans and greatly dependent on exposure and the valence of the vanadium ion. For the latter, the order of toxicity is 5 + > 3 + > 2 +. The green blood pigment of the tunicates, a group of marine lower chordates, contains vanadium, and these organisms effectively accumulate the element from sea water and silts. Some holothurians (sea cucumbers) also contain high levels of vanadium.

The essentiality of vanadium for growth in higher plants has not be conclusively demonstrated. It has been shown that vanadium plays a role in the absorption and reduction of nitrogen by plants. Information on the toxicity of plants to vanadium is relatively limited, particularly information on long-term toxicity effects.

Plants accumulate and translocate vanadium, particularly at acidic pH. Phytotoxicity may involve interference with iron uptake by plants.

B. Conclusions

The following conclusions are based on the information contained in this report:

(1) Although a relatively large amount of vanadium (on the order of 30,000 metric tons per year) enters the environment from man's activities, no widespread detrimental effects have been identified to date. Presumably, man and the higher animals do not accumulate vanadium in hazardous amounts.

(2) Relatively little information is available concerning effects of vanadium on plants, lower animals, and microorganisms. Since some of these species accumulate vanadium and some exhibit detrimental effects for acute exposure, potential environmental hazards from vanadium may exist if environmental vanadium levels increase.

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(3) Use of vanadium will probably continue to increase, particularly its use in steels and other alloys, but there is no present indication that such usage increases will be rapid. Potential supplies are available to meet significant increases in demand.

C. Recommendations

The following recommendations of further work are based on the summary and conclusions presented above:

(1) It is recommended that further study of effects of vanadium, specifically soluble vanadium oxides upon plants (and possibly lower animals and microorganisms as well) be performed. Areas of high soil vanadium perhaps could be useful to such a study.

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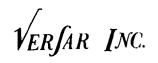
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