CHAPTER 9

SELENIUM- A NEW ENTRANT TO MEDICAL GEOLOGY

The element selenium could be regarded as a new entrant to the field of medical geology. It is of special importance to the developing countries of the tropical belt where there appears to be an apparent association with some diseases. Since selenium is not an element that is determined in routine investigations, the real impact of selenium excess or deficiency on the population may be hidden. Recently however, the importance of selenium as a medically important element has received increasing attention and many studies, notably in China, have been carried out on the epidemiology of selenium-associated diseases.

Selenium was designated as an essential trace element for humans and animals in the late 1950s. It is a part of the biologically important enzyme glutathione peroxidase (GSH-PX) which acts as an antioxidant preventing tissues degeneration. However, as for some other trace elements, excessive doses could cause ill health. The range between deficiency levels, (<11 μg/g per day) and toxic levels in susceptible people (>900 μg/g per day) is very narrow (Fordyce et al., 2000b; Yang and Xia, 1995). The endemic degenerative heart disease in China, known as Keshan Disease and an endemic osteoarthropathy (Kaschin-Beck Disease) causing deformity of affected joints, are attributed to selenium deficiency.

THE GEOCHEMISTRY OF SELENIUM IN THE ENVIRONMENT

Selenium is classified as a metalloid -an element which has properties of both a metal and a non-metal. It shows chemical similarities to sulphur, which also lies in Group VI of the periodic table. Because of this similarity in form and components they have many interrelations in biology (Adriano, 2001). Selenium exhibits a number of oxidation states, -II, 0, +IV and +VI. The most important oxidation states of selenium in the environment are -II (selenides) 0 (ground state) +IV (selenites) and +VI (selenates). The

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abundance of Se in the earth's crust ranges from 0.05 to 0.09 mg/kg, approximately 1/6000th of sulphur and 1/50th of that of arsenic. Table 9.1 shows the selenium concentrations in some environmental media. Coal and black shales are generally enriched in selenium. Approximately 50 selenium minerals are known and on account of the similarity of Se with S, it is commonly associated with heavy metal (e.g. Ag, Cu, Pb, Hg, Ni) sulphides and occurs as either a selenide or as a substitute ion for S in the crystal lattice (Malisa, 2001).

The mobility and bioavailability of selenium is determined by the chemical form. Table 9.2 shows the common chemical forms of selenium in geological and biological materials. The general geochemistry of selenium is complex since it shows several oxidation states and it has the ability to complex with organic matter (Figure 9.1). Selenium is therefore found in a variety of naturally occurring materials, the main sources being rock weathering, volcanic emissions and metal sulphide deposits.

In sedimentary rocks, Se is associated with the clay size fraction and hence it is more abundant in shales as compared to limestones or sandstones. It is also known that relatively high concentrations (>300 μg/g) of Se are found in some phosphate rocks and in view of the fact that phosphate fertilizers are commonly used, this may be an important source of selenium in the environment (Frankenberger and Engberg, 1998).

The Se concentrations of most soils range from 0.01-2 μg/g (world mean 0.4 μg/g; Fergusson, 1990). In some seleniferous areas, however, Se concentrations up to 1200 μg/g have been reported (Mayland et al., 1989). As shown in Figure 9.2, in acid and neutral soils inorganic Se occurs as very insoluble Se^{4+} compounds of oxides and oxyhydroxides of ferric iron. In neutral and alkaline soils, Se^{6+} is the main oxidation state, being soluble and hence more bioavailable. A notable geochemical feature is that Se^{4+} is adsorbed onto soil particle surfaces, with greater affinity than Se^{6+} (Fugita et al., 2005). The soil geochemistry of selenium is controlled by many factors-among which are:

(a) Se-speciation
(b) soil texture
(c) mineralogy
(d) organic matter
(e) presence of competing ions
(f) iron-oxides
(g) clays.

Table 9.1. Selenium concentrations in selected materials (McNeal and Balistrieri, 1989; Fordyce et al., 1998; Malisa, 2001).

Material	Se (μg/g)
Earth's Crust	0.05
Granite	0.01-0.05
Limestone	0.08
Sandstone	<0.05
Shale	0.06
Phosphate rock	1-300
Granite	0.025
Soil	
USA	<0.1-4.3
England/Wales	<0.01-4.7
Seleniferous	1-80, <1200
Coal	0.46-10.65
Atmospheric dust	0.05-10
River water	
Mississippi	0.00014
Amazon	0.00021
Colorado (alkaline)	0.01-0.4
Lake Michigan	0.0008-0.01
Sea water	0.000009
USA plants	
Grasses	0.01-0.04
Clover and alfalfa	0.03-0.88
Barley	0.2-1.8
Oats	0.15-1
Algae	
Marine	0.04-0.24
Freshwater	<2
Whole fish	
Marine	0.3-2
Freshwater	0.42-0.64
Animal tissue	0.4-4

Table 9.2: Common chemical forms of Se in the environment.

Oxidation state	Chemical forms
Se^0	Elemental selenium
Se^{4+}	Selenite SeO_3^{2-}
	Trimethylselenonium (TMSe) $(CH_3)_3Se^+$
	Selenous acid $H_2Se_3^{3-}$
	Selenium dioxide SeO_2
Se^{6+}	Selenate SeO_4^{2-}
	Selenic acid $H_2SeO_4^{2-}$
Se^{2-}	Selenides Se^{2-}
	Dimethylselenide (DMSe)$(CH_3)Se$
	Dimethyldiselenide (DMdSe)$(CH_3)_2Se_2$
	Hydrogen selenide H_2Se
	Dimethylselenone $(CH_3)_2SeO_2$

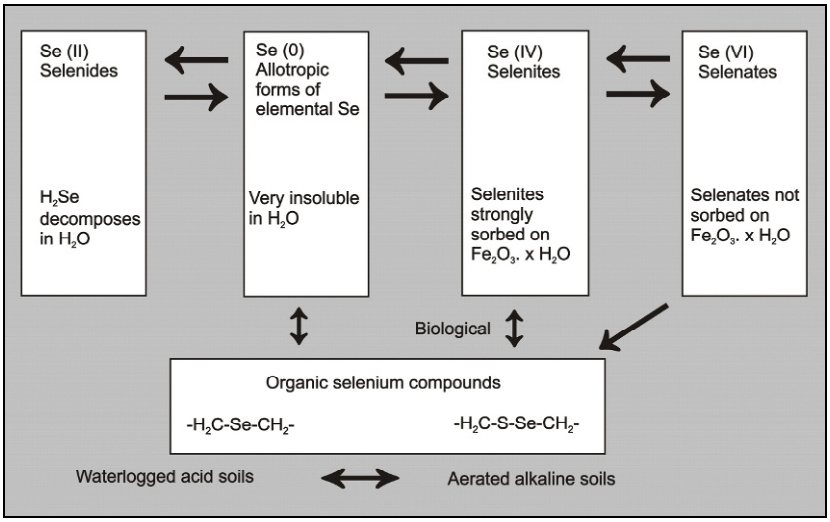

Fig. 9.1. Generalized chemistry of selenium in soils and weathering sediments (Allaway, 1968)

The bioavailability of selenium is particularly influenced by clay and organic matter and these, due to their capacity to trap selenium, lower the bioavailability. The presence of SO_4^{2-} and PO_4^{3-} which compete with Se ions for fixation sites in soils and plants are also known to affect the bioavailability of selenium (Fordyce et al., 1998).

The Se concentrations in water, are generally very low and only rarely exceed the WHO safely limit of 10 µg/L. MacGregor (1998), studied a number of wells in the Amman-Zarqa Basin in Jordan and reported Se concen-

tration up to 12 times the WHO safety range of values being 0.8 µg/L to 112 µg/L. The wells with highest levels were situated extremely close to an abandoned phosphate mine.

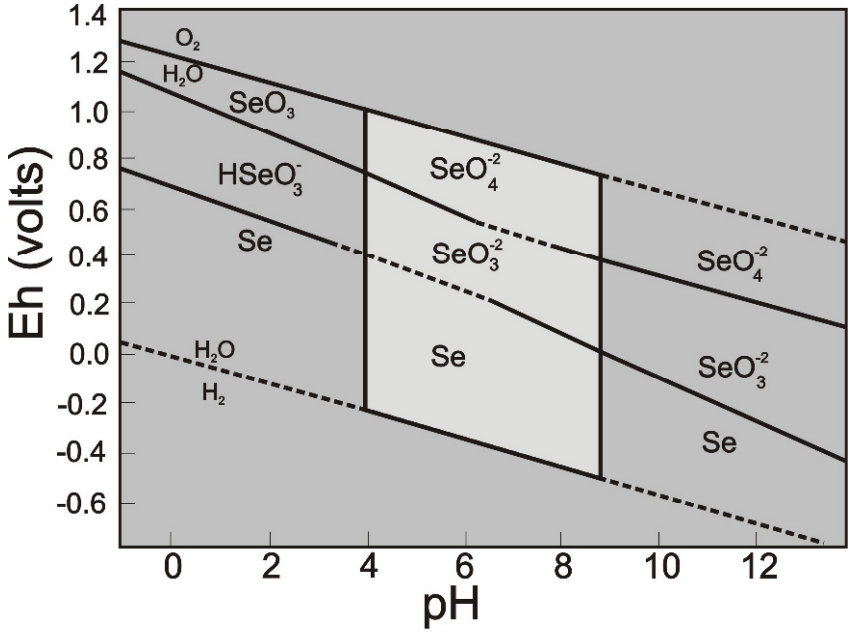

Fig. 9.2. Eh-pH diagram of Se in soils (Mayland et al., 1989)

Selenium is considered non-essential for plants. However, certain plants, termed hyper-accumulators have the ability to absorb and accumulate high concentrations of selenium. Plants absorb selenium in the form of selenate, selenite or organic-Se. Selenate is absorbed by the root system through the binding sites of sulphate. In view of the similarities, S and Se show similar biochemical reactions, though in competition with each other. Selenite, however, is thought to be taken up through different sites. The biochemical similarity of Se and S are seen in their metabolism by the same enzymes and assimilatory pathways (Adriano, 2001). The formation of Se analogues of S compounds that are substrates for S assimilation enzymes therefore, cause Se- toxicity. This is mainly due to the likely replacement of S by Se in the amino acids of proteins, which in turn disrupts the essential catalytic reactions (Figure 9.3).

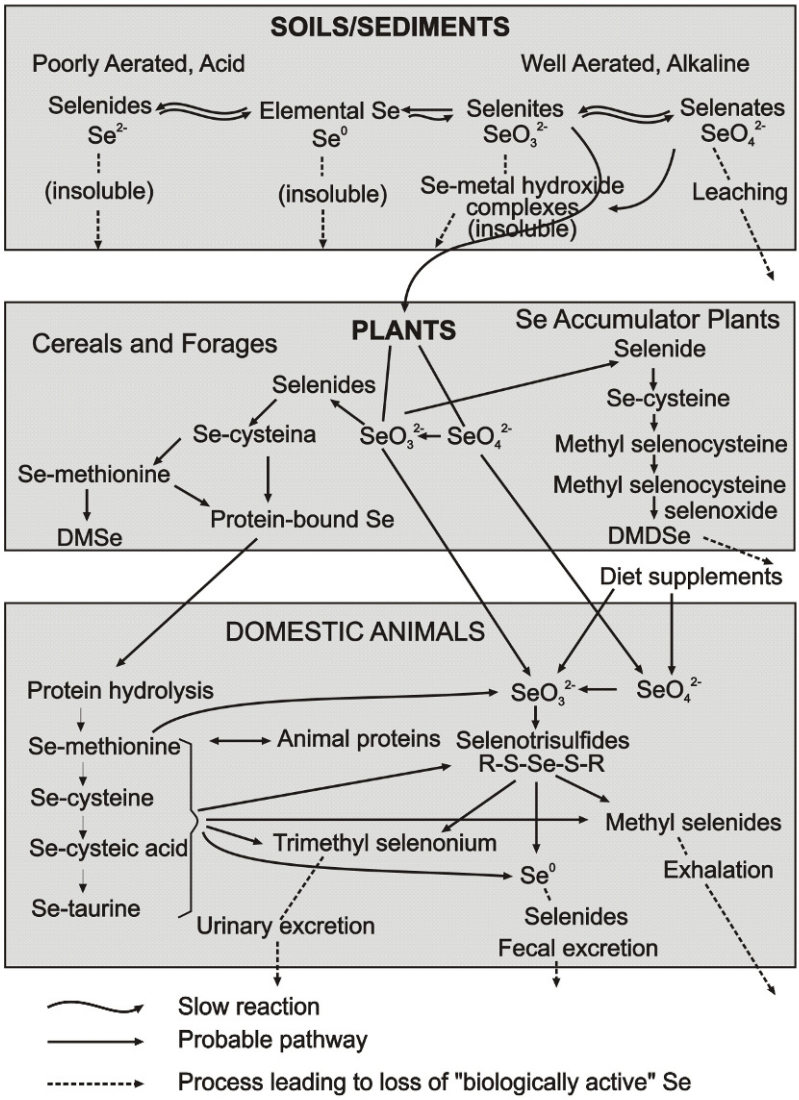

Fig. 9.3. Biogeochemical reactions and pathways of selenium in the soil-plant-animal system (Adriano, 2001)

The hyperaccumulation of selenium by some plants, which in some cases rises to more than 0.5 wt.%, is of special interest. As shown in Figure 9.4, while normal plants (non-adapted) do not separate S from Se biochemically, the hyper-accumulators are able to separate inorganic sulphur (as sulphate) from inorganic selenium (as selenate or selenite) as they enter the plant. Hence Se toxicity is avoided.

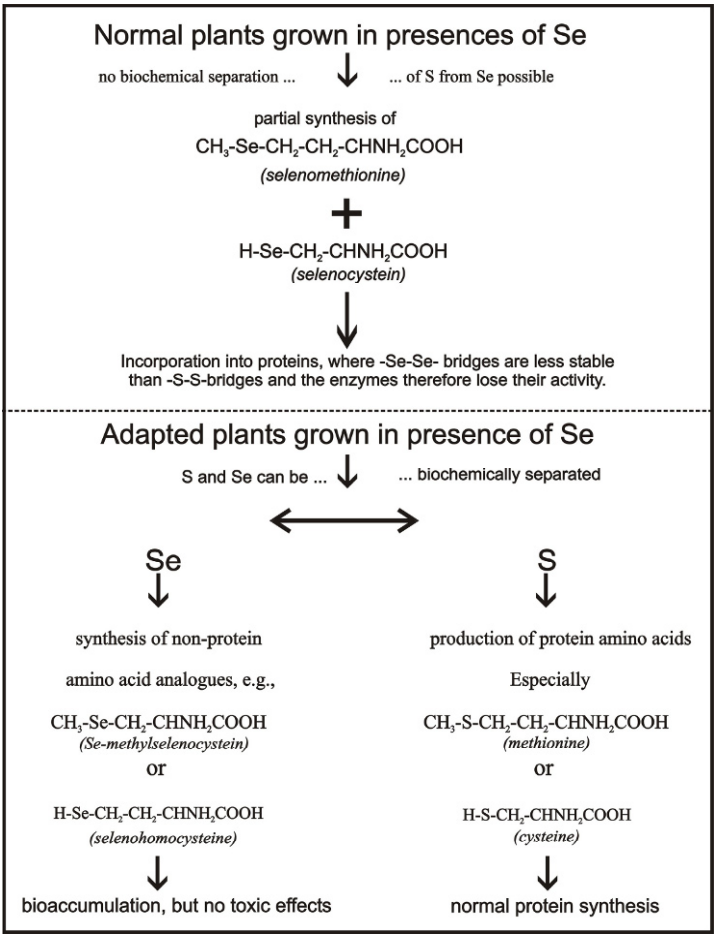

Fig. 9.4. Adaptation and bioaccumulation of selenium in plants (Streit and Stumm, 1993)

Microbial Transformation of Selenium

The oxidation of selenium and its subsequent geochemical pathways in the environment involve to a large extent microbial reduction, oxidation, methylation and demethylation reactions. Biological transformations of toxic Se-oxyanions into less toxic or biologically unavailable forms, such as Se or volatile Se compounds are being investigated thoroughly in view of their potential use in bioremediation (Dungan and Frankenberger, 1999).

Since both selenate [SeO_4^{2-}; Se(VI)] and selenite [SeO_3^{2-};Se (IV)] are toxic and show bioaccumulation, the removal or immobilization of these oxyanions has received greater attention.

The selenium cycle in soil is illustrated in Figure 9.5. As in the case of sulphur, microbial transformations predominate in the cycling of selenium. There are four biological transformations that are of importance. These are (i) reduction (assimilatory and dissimilatory) (ii) oxidation (iii) methylation (iv) demethylation.

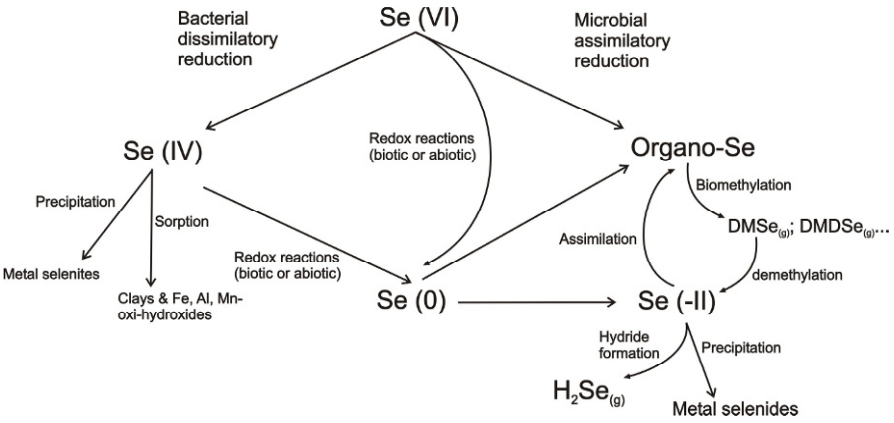

Fig.9.5. Mobility of selenium in the environment

Assimilatory reduction is the reduction and incorporation of selenium into organic compounds and dissimilatory reduction is when microorganisms reduce SeO_4^{2-} as a terminal electron acceptor in energy metabolism. As noted by Dungan and Frankenberger (1999), the focus of attention has been on the dissimilatory reduction of Se-oxyanions in view of their potential application in remediating seleniferous environments by producing the biologically unavailable Se^0. Methylation of Se is considered to be a mechanism used by microorganisms to protect themselves against Se-toxicity in Se-rich environments. The volatilization of Se which follows the methylation reactions then removes the selenium from the toxic environment. Tables 9.3 and 9.4 list the selenium reducing microorganisms.

Dissimilatory Reduction

The reduction of both Se-oxyanions to Se^0 in the soil-sediment-water systems is caused by several types of bacteria, the majority of them being SeO_3^{2-} reducers (Bautista and Alexander, 1972; Doran, 1982). It has been

demonstrated by Oremland et al. (1989) that the bacterial reduction of SeO_4^{2-} to the non-bioavailable Se^0 is a major sink for Se-oxyanions in anoxic sediments.

Table 9.3. Selenium reducing microorganisms (Dungan and Frankenberger, 1999)

Organisms	Description of Reaction
Enterobacter cloacae SLD1a-1	Respires SeO_4^{2-} and NO_3^-, and reduces SeO_4^{2-} to Se^0 only in the presence of NO_3^-
Thauera selenatis	Grows anaerobically using SeO_4^{2-}, NO_3^-, and NO_2^-. Reduction of SeO_4^{2-} occurs by way of a SeO_4^{2-} reductase. SeO_4^{2-} is completely reduced to SeO only when NO_3^- is present
Strain SES-3	Respires SeO_4^{2-} and can reduce SeO_3^{2-} to Se^0 in washed-cell suspensions
Pseudomonas stutzeri	Reduction of SeO_4^{2-} and SeO_3^{2-} to Se^0 under anaerobic conditions
Wolinella succinogenes	Adapted cultures able to reduce SeO_4^{2-} and SeO_3^{2-} to Se^0 under anaerobic conditions
Desulfovibrio desulfuricans	SeO_4^{2-} and SeO_3^{2-} were reduced to Se^0 under anaerobic conditions, but both Se oxyanions could not support growth
Salmonella heidelberg	SeO_3^{2-} reduced aerobically to Se^0
Streptococcus faecalis N83 *Streptococcus faecium* K6A	Anaerobic reduction of SeO_3^{2-} to Se^0 by resting-cell suspensions
Clostridium pasteurianum	Reduction of SeO_3^{2-} by hydrogenase (I)
Bacillus subtilis *Pseudomonas fluorescens*	Reduction of SeO_3^{2-} to Se(0) by a NO_2^- and SO_3^{2-} independent enzyme system

Assimilatory Reduction

Both selenate and selenide undergo assimilatory reduction to Se^{2-} which gets incorporated into cellular proteins, similar to the mechanism of sulphur incorporation in amino acids. The products, the selenoamino acids such as selenomethionine and selenocysteine have been produced by some bacteria and yeast. However with excess Se toxicity effects appear and the organisms undergo metabolic deterioration.

Table 9.4. Selenium volatilizing microorganisms (Dungan and Frankenberger, 1999)

Organisms	Se Substrate	Se Product
Fungi		
Scopulariopsis brevicaulis	SeO_4^{2-}, SeO_3^{2-}	DMSe
Penicillium notatum	SeO_4^{2-}, SeO_3^{2-}	DMSe
Penicillium chrysogenum		
Schizopyllum commune	SeO_3^{2-}	DMSe
Aspergillus niger	SeO_4^{2-}	DMSe
Penicillium sp.	SeO_3^{2-}	DMSe
Penicillium sp.	SeO_4^{2-}, SeO_3^{2-}	DMSe
Fusarium sp.		
Cephalosporium sp.		
Scopulariopsis sp.		
Candida humicola	SeO_4^{2-}, SeO_3^{2-}	DMSe
Alternaria alternata	SeO_4^{2-}, SeO_3^{2-}	DMSe
Penicillium citrinum	SeO_3^{2-}-	DMSe, DMDSe
Acremonium falciforme		
Penicillium sp.	SeO_3^{2-}	Unidentified
Bacteria		
Corynebacterium sp.	SeO_4^{2-}, SeO_3^{2-},Se^0	DMSe
Aeromonas sp.	SeO_3^{2-}	DMSe, DMDSe
Flavobacterium sp.		
Pseudomonas sp.		
Pseudomonas fluorescens K27	SeO_4^{2-}	DMSe, DMDSe, DMSeS
Rhodocyclus tenuis	SeO_4^{2-}, SeO_3^{2-}-	DMSe, DMDSe
Rhodospirillum rubrum S1		
Aeromonas veronii	SeO_4^{2-}, SeO_3^{2-} ,Se^0, SeS_2, H_2SeO_3, SeH	DMSe, DMDSe, methylselenol, DMSeS

Oxidation

The oxidation of reduced forms of Se to Se-oxyanions is an important reaction in the environment not only because SeO_4^{2-} and SeO_3^{2-} are soluble and toxic, but also because biomethylation of Se depends mainly on the oxidized forms of Se (Dungan and Frankenberge, 1999). In view of the similar biochemistry of S and Se, it is expected that microbial oxidation of Se occurs in a manner similar to sulphur, both heterotrophic and autotrophic organisms being able to carry out the reaction.

Methylation and Volatilization

In soils, water and sediment enriched in selenium, methylation of Se-compounds from Se-oxyanions and organo-Se compounds can take place (Doran, 1982). This is essentially a mechanism of detoxification of the environment by some bacteria and fungi in soils and by bacteria in the aqueous systems. The methylation of selenite to dimethyl selenide by the soil bacterium Corynebacterium, was proposed by Doran (1982) to follow the mechanism.

SeO_3^{2-}	$\rightarrow$	Se^0	$\rightarrow$	HSeX	$\rightarrow$	CH_3SeH	$\rightarrow$	$(CH_3)_2Se$
Selenite		elemental Se		selenide		monomethanol seleniumhydride		dimethyl-selenide

For the aqueous system, Cooke and Bruland (1987) proposed a pathway for the reduction of dimethylselenide.

$$SeO_3^{2-} \xrightarrow[\text{reduction to (-2)}]{\text{assimilation and}} CH_3Se(CH_2)_2CHNH_2COOH$$

selenomethionine

$$CH_3Se(CH_2)_2CHNH_2COOHCH_3 \xrightarrow{\text{methylation}} (CH_3)_2Se^+(CH_2)_2CHNH_2COOH$$

dimethyl selenomethionine

(dimethyl selenonium ion)

$$(CH_3)_2Se^+(CH_2)_2CHNH_2COOH \xrightarrow{H_2O} CH_3SeCH_3 + CH_3COHCH_2CHNH_2COOH$$

dimethylselenide homoserine

Methyl cobalamin and S-adenosylmethionine are known as the methyl donors in the microbial methylation of Se. Dimethylselenide is considered to be the major volatile species produced by most microorganisms.

SELENIUM AND HUMAN AND ANIMAL HEALTH

Recent research has shown that the trace element selenium is an essential nutrient and is of fundamental importance to human and animal health. Schwarz and Foltz (1957) first showed the essentiality of selenium when they demonstrated their prevention of liver necrosis in rats by selenium. Subsequently Thompson and Scott (1969) showed that poor growth and higher mortality rates of chicks was caused by Se-poor diets.

More recently (Deverel et al., 1984) selenium poisoning was found to be the major cause of high mortality of wildfowl at the Kesterson Wildlife Refuge in the San Joaquin Valley, California. Selenium from the selenium-rich country rock had been geochemically mobilized and found its way into the Refuge reservoir which was a nesting area for wildfowl. Both fish and wildfowl had very high levels of selenium.

Among the diseases associated with a low intake of Se are cardiomyopathy (Keshan disease), deforming arthritis (Kashin-Beck disease), protein energy malnutrition, haemolytic anaemia, hypertension, ischaemic heart disease, cancer, multiple sclerosis, muscular dystrophy, infertility, cystic fibrosis and alcoholic cirrhosis (Haygarth, 1994; Rotruer et al., 1993).

These case studies clearly demonstrate the essentiality and toxicity of selenium which shows a very narrow threshold window (>900 μg/g per day-toxicity and <11 μg/g per day). Selenium is a component of selenoproteins some of which have important enzymatic functions. Selenocystine is the 21st aminoacid. It has been now recognized that all these enzymes are selenium-dependent, with selenocysteine as the active site (Sunde, 1997). The importance of selenium here is that it functions as a redox centre. Selenium is an important component of glutathione peroxidase (GSHPX), considered as a critical enzyme which prevents oxidative damage at the cellular level. It helps to maintain membrane integrity, protects prostacyclin production and reduces the likelihood of propagation of further oxidative damage to biomolecules such as lipids, lipoproteins and DNA with the associated increased risk of conditions such as atherosclerosis and cancer (Diplock, 1994; Néve, 1996; Rayman, 2000). Selenium deficiency diseases in ani-

mals are mostly seen in livestock and include reproductive impairment, growth depression and white-muscle disease, a myopathy of heart and skeletal muscle affecting mainly lambs and calves (Reilly, 1996). Rayman (2000) reviewed the importance of selenium to human health and discussed the health effects of less overt selenium deficiency. These deficiency-related health effects can be summarized as follows.

Immune function

Deficiency of selenium is thought to be accompanied by loss of immunocompetence. This is perhaps linked to the fact that selenium is normally found in significant amounts in immune tissues such as liver, spleen and lymph nodes. Selenium supplementation is also known to have marked immunostimulating effects.

Viral infections - AIDS

Selenium deficiency is associated with the occurrence, virulence or disease progression of some viral infections. It is a potent inhibitor of HIV replication in vitro. Selenium-deficient HIV patients are nearly 20 times more likely ($p < 0.0001$) to die from HIV-related causes than with sufficient levels (Baum et al., 1997). These authors defined selenium-deficiency as plasma concentrations at or below 85 µg/L. In the case of HIV-infected children, the low levels of plasma selenium were significantly and independently related to mortality (relative risk 5.96; $p = 0.02$) and faster disease progression (Campa et al., 1999).

Reproduction

Selenium is considered essential for male fertility. It is required for testosterone biosynthesis and the formation of and normal development of spermatozoa.

Mood

Rayman (2000) reports some studies where low selenium status had been linked to greater incidence of depression, anxiety, confusion and hostility.

Thyroid function

When selenium deficiency combines with iodine deficiency, hypothyroidism is enhanced and this is manifested as myxoedematous cretinism. This disease is known in the Democratic Republic of Congo (Zaire) where both iodine and selenium are known to be deficient (Vanderpas et al., 1990).

Cardiovascular diseases

Though not proven to any degree of certainty, selenium is thought to be protective against cardiovascular disease (Nève, 1996). Glutathione peroxidase, of which selenium is a component, is known to reduce hydroperoxides of phospholipids and cholesteryl esters associated with lipoproteins. This is expected to reduce the accumulation of oxidized low – density lipoproteins in the artery wall. The status of other antioxidants such as vitamin E however may complicate direct correlations.

Oxidative-stress or inflammatory conditions

Selenium is known to exhibit properties of an antioxidant and anti- inflammatory agent. In the case of pancreatitis, asthma and rheumatoid arthritis, selenium levels are considered to be important. In a study in Germany, intravenous administration of selenium to patients with necrotising pancreatitis reduced mortality from 89% in controls to zero in the treatment group (Kuklinsky and Schweder, 1996).

Cancer

An inverse relation between selenium intake and cancer mortality has been proposed in several epidemiological studies. The dietary intake of selenium in 27 countries was studied by Schrauzer et al. (1977). In the Harvard based Health Professionals' Cohort study (Yoshizawa et al., 1998) which involved the investigation of selenium intake and prostate cancer in 34000 men, it was observed that those in the lowest quintile of selenium status had 3 times the chances of developing cancer as against the highest quintile. It should however be emphasised again that correlation does not necessarily mean causation and further detailed studies are very necessary to attribute causal effects of selenium deficiency or toxicity to health.

SELENIUM DEFECIENCY DISEASES IN CHINA

The two diseases, Keshans's Disease (KD) and Kashin-Beck Disease (KBD) are particularly prevalent in some parts of China, and given China's population of over 1.3 billion(~20% global population) global Se-deficiency in China is of special interest in medical geology. Apart from KD and KBD, the impact of selenium-deficiency on human longevity is also an interesting study in China (Moffat, 1990; Foster and Zhang, 1995) bearing in mind that there is a relative lack of migration of people from village to village and the reliance of locally grown food which is influenced heavily by the soil chemistry.

Figure 9.6 illustrates the distribution of KSD and KBD in China. The low Se-belt (Figure 9.7) also known as the 'disease belt' stretches from Heilongjiang Province in the NE to Yunnan Province on the SW. The average abundance of Se in the earth's crust of China is 0.058 mg/kg and is lower than that in other parts of the world (Xia and Tang, 1990). In the different rock types, Se decreased in the order slate > clay rocks > basic and ultrabasic rocks > alkaline rocks > basalt > granite > hypersthene sandstones > limestones.

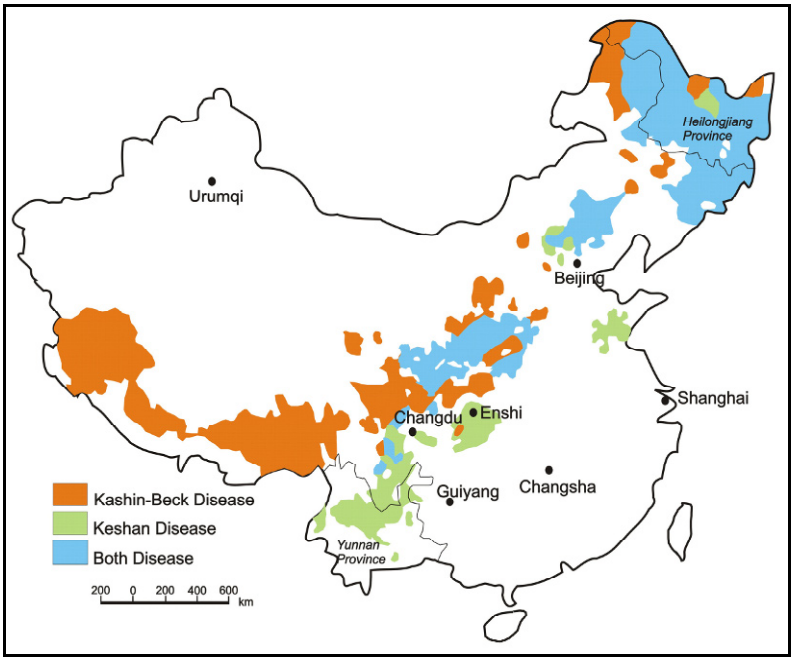

Fig. 9.6. Distribution of KSD and KBD in China (Tan et al., 2002)

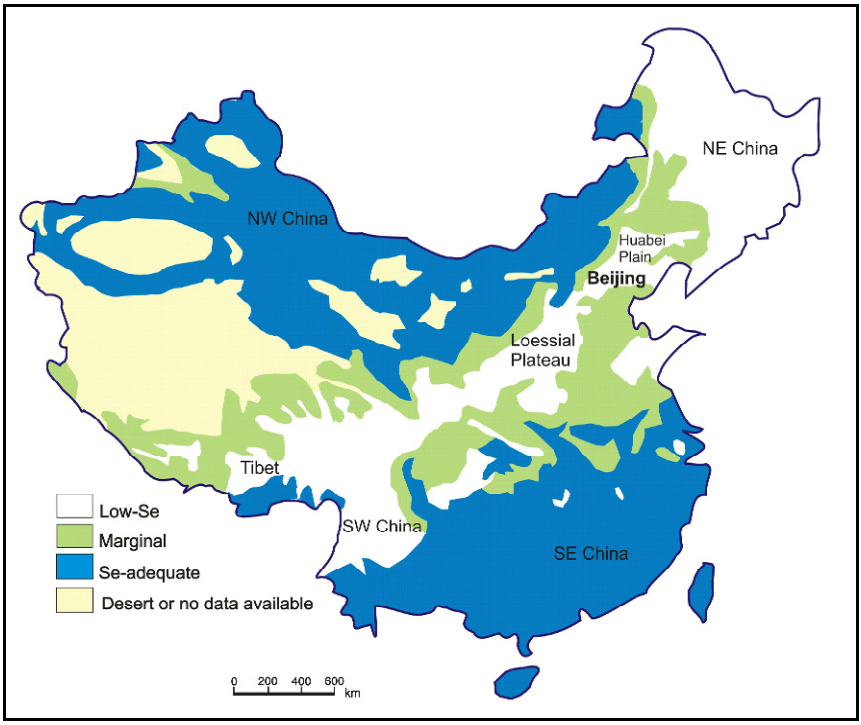

Fig. 9.7. Geographical distribution of the Chinese Low-Se Belt (Tan, 1989)

Wang and Gao (2001) report the occurrence of two notable Se-excessive or seleniferous regions in China, namely the Exi region in Hubei Province and the Ziyang region in Shanxi Province. The bedrock in the Exi seleniferous region consisted of carbonaceous shale and high Se coal.

Whereas the concentration of Se in fresh water around the world is 0.2 µg/L in the KD and KBD areas in China, in the low Se belt, Se concentration in well water is sometimes as low as 0.11 µg/L. In most Chinese cities and urban areas it is 0.65 µg/L. On the other hand, in the selenium-rich areas, two well water samples had 8.4 and 72 mg/L selenium (Wang, 1991).

Tan et al. (2002) studied the selenium contents in soil in the endemic disease areas in China and noted that the concentration of selenium in soil mainly depends on two groups of factors. These were (i) geographically azonal factors such as parent rocks and landforms which determine the source of Se in soil (ii) zonal factors such as biological and climatic factors, which influences the migration, maintenance, existing forms and availability of Se in e.g. soils, plants.

As shown in Table 9.5 in the KD and KBD areas, the concentrations of total-Se and water soluble Se both in cultivated and natural soils are lower than that in non-affected areas. The natural soils in the affected areas included dark brown soil, brown earth, drab soil, yellow-brown soil, red drab soil, loessial soil, purple soil and black soil. In disease-free areas, laterites, red soils, yellow soils, desert soils, chernozem, chestnut soil and calcic brown soils were found.

Table 9.5. Se contents of soils in the areas with and without KSD/KBD (Note: N: number of samples; X: arithmetic mean; G: geometric mean; S.D.: arithmetic standard deviation; S_{lg}: geometric standard deviation) (Tan et al., 2002)

Type	Area with KBD / KSD					Area without KBD / KSD				
	X	SD	G	S_{lg}	N	X	SD	G	S_{lg}	N
Total Se (mg/kg)										
Cultivated	0.112	0.057	0.100	0.1816	35	0.224	0.134	0.219	0.3297161	
Natural	0.119	0.075	0.105	0.2177	69	0.227	0.141	0.211	0.319286	
Water soluble Se (µg/kg)										
Cultivated	2.5	1.0	2.5	0.3914	25	6.8	9.1	4.7	0.3106151	
Natural	2.8	2.2	2.2	0.2651	22	6.7	13.2	4.7	0.445271	

Tan et al. (2002) concluded that the distribution of Se in top soil in China and its relationship to Keshan Disease and Kashin-Beck Disease is indicative of <0.125 mg/kg total selenium. The Se content in seleniferous areas was >3 mg/kg.

Fordyce et al. (2000b) studied the soil, grain and water chemistry in relation to human selenium responsive diseases in Enshi District China and noted that the majority of samples in the low selenium villages are deficient or marginal in Se and that Se availability to plants is inhibited by adsorption onto organic matter and Fe oxyhydroxides in soil. They also observed that in high Se-toxicity villages, the Se-bioavailability is controlled by the total soil Se concentration and pH. Johnson et al. (2000) were of the view that the organic content of the soils is a major factor in controlling the availability of Se and that the high incidence KD villages have the most organic-rich soils. Although higher in total Se, the organic-rich soils had little bioavailable Se resulting in a Se-deficient food chain.

SELENIUM AND IODINE DEFICIENCY DISEASES (IDD)

Recent research has shown that Se deficiency may be an important factor in the onset of IDD. It is known that the selenoenzyme, type-I iodothyronine deiodinase (IDI), is responsible for the conversion of the prohormone T_4 to the active hormone T_3 which exerts a major influence on cellular differentiation, growth and development, especially in the foetus, neonate and child. The conversion of T_4 to T_3 is inhibited by Se-deficiency and this affects thyroid hormone metabolism (Arthur and Beckett, 1994; Arthur et al., 1999).

Fordyce et al. (2000a) studied the relationship of IDD in Sri Lanka and Se deficiency and observed that it is unlikely that Se deficiency is the main controlling factor in IDD. However they were of the opinion that it could contribute to the onset of goitre along with iodine deficiency and other factors such as poor nutritional status and the presence of goitrogenic substances in the diet.