ABOUT THE AUTHOR

James E. Oldfield, Professor emeritus of Animal Nutrition, Department of Animal Sciences, Oregon State University, Corvallis, Oregon, is a leading pioneer in the study and eradication of white muscle disease (WMD). He began publishing on this subject in 1955, showing how the disease can be produced experimentally and as early as 1958 demonstrated (with O.H. Muth, L.F. Remmert, and J.R. Schubert) the protective effects of selenium and vitamin E. The study of WMD and other selenium-responsive conditions in ruminants as well as various aspects of selenium in animal nutrition continued to occupy him during the next four decades. As the Head of the Department of Animal Science at OSU from 1967 to 1983, he helped make it an internationally renowned center of selenium research. James Oldfield was born August 30, 1921, in Victoria, B.C., Canada. He received his early professional training at the University of British Columbia and at Oregon State College (now Oregon State University), where he received his Ph.D. degree in animal science in 1951 and subsequently continued with his research and teaching career. In 1969 he was nominated Distinguished Professor, OSU, and from 1981 to 1983 he was Rosenfield Distinguished Professor of Agriculture. Professional awards include (with O.H. Muth and J.R. Schubert) the Basic Science Award of Oregon Agricultural Experiment Station (1961); the Sigma Xi Research Award, OSU (1964); the Morrison Award, American Society of Animal Science (1972); and the Distinguished Service Award, Western Section, American Society of Animal Science. He also received a Fulbright Research Scholarship to Massey University, New Zealand, in 1974. In 1978 he became a Fellow of the American Society for Animal Science. In 1984 he was elected into the Fur Industry Hall of Fame, and in 1987 he became a Fellow of the American Institute of Nutrition. In addition to his work on selenium, he has studied nutritional requirements and nutritional problems in various species of animals, the autoxidation of lipids (particularly fish oils), growth and reproduction problems, steatitis or “yellow fat”, biological effects of forage control, nutritionally induced fur defects in mink, and the utilization of animal wastes. He is the author of 140 technical papers and journal articles and of 90 reports and non-journal articles, and has organized numerous meetings on selenium and related topics. In 1998 he received the prestigious Klaus Schwarz Medal for his contribution to biological trace element research.

Dedication

This Atlas is affectionately dedicated to the memory of Dr. Douglas V. Frost, an early selenophile, who perhaps more than any other one person was responsible for changing the popular image of selenium from suspected carcinogen to that of an important anticancer agent.

Acknowledgement

Many people have given generously of their knowledge and experience in assembling the text and illustrations included herein and I am grateful to each and all of them. I must add particular appreciation to Conor Reilly, long a chronicler of the selenium scene; to Mike Shirer who applies selenium to New Zealand soils, to Holger Artelt and Yves Palmieri of the Selenium Tellurium Development Association for financial and technical support, and to Cindy Withrow, who understands what I am really trying to say, and who typed the entire text, not once but several times.

James E. Oldfield
Preface

In the year 1265 Marco Polo while traveling the “Silk Road” returning home to Venice, Italy learned of a plant that if eaten by horses caused them to become sick. Illness and ill thrift similarly affected range livestock and horses eating poisonous plants in the western United States in the 1930s’. Death of aquatic birds and malformation of bird embryos in the 1980’s were observed in and about the Kesterson Wildlife Refuge and Reservoir in central California. On the other side of the world at this same period of time, Keshan disease, a human cardiomyopathy was identified in the People’s Republic of China. Diseases of the heart and also muscle tissues had been recognized two decades earlier in animals; sheep, swine and cattle in New Zealand, Australia, Europe and the United States. These disease conditions in animals and humans are the natural consequences of too much or too little of the element selenium in soils. While a known essential trace element since 1957 forward, and now for all animal species as well as humans, selenium is not required by most, if any, plant species. Plants provide forage and food selenium and are the passive conduit of the soil selenium essential for and sometimes toxic to animals and humans. The selenium cycle begins and ends with the soil and its concentration and chemical form in soil determines the need for animal or human supplementation. Selenium supplementation for animals and humans is accomplished by the addition of selenium to fertilizers as in some Scandinavian countries, the coating of seeds with selenium as in Canada or the direct addition of selenium to animal feeds and human supplements.

James E. Oldfield, Professor Emeritus of Animal Science at Oregon State University at Corvallis, OR, was among the very first scientists in the 1950’s to show a selenium requirement for ruminant animals in areas of Oregon deficient in soil selenium. His 1999 World Selenium Atlas was the first comprehensive effort to assemble in one compendium all of the published information on the worldwide content of selenium in soils, cereals and forages. The “selenium maps” of the World Selenium Atlas have become very useful in assessing the relationships between dietary intake of selenium and animal and human diseases. Particularly interesting in the atlas are the comprehensive maps of selenium and Keshan disease showing the striking correlation of soil selenium and this human disease within the People’s Republic of China. I have used Professor Oldfield’s atlas in my own research only to be disappointed at times to find that several parts of the world have yet to be mapped for selenium and to recognize that there is map work yet to be done.

The importance of researchers continuing to map the soil, plant selenium relationship can be seen in the Serbian Academy of Sciences and Arts regional “conference on Selenium” (volume 78, 1995). Fully 40% of the papers published in the proceedings of that conference
dealt with the selenium content in soils, plants and water of greater Serbia, which is assessed as being selenium deficient with respect to animal and human nutritional requirements. And now Professor Oldfield has revised and updated his World Selenium Atlas in this new Millennium to the benefit of all of us. His revised edition will particularly benefit research scientists, animal scientists, public health officials, toxicologists, policy makers and epidemiologists in their work. All should be grateful for Professor Oldfield’s visionary efforts in producing the first edition and now updating it with the newest information on selenium’s environmental distribution. He is also to be congratulated on his more than 50 years of research and understanding of the importance of selenium to animal and human health.

March 13, 2002

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Foreword

Although selenium has been recognized as an essential micronutrient for some 45 years and has been widely and effectively used as a feed supplement for livestock in areas of deficiency, similar use in the human diet has not been recommended because of the diverse sources of human foods, which make deficiencies less likely.

Now this situation has changed and it has been proven that selenium deficiencies in human diets can occur, and perhaps more significantly, that selenium at slightly higher than “nutritional” levels appears to have a protective effect against certain disease situations including types of cancer and coronary disease. These findings have added urgency to ensuring the adequacy of selenium in the human dietary, on a national basis, and indeed, two countries, Finland and New Zealand have recommended the addition of selenium to fertilizers for croplands. Such decisions require knowledge of the selenium status of soils and crop plants in specific countries or areas within them, and this Atlas is an attempt to assemble in one place what is known about world selenium supply situations.

Some omissions in the first edition of this Atlas have been corrected herein. In the course of gathering the information, the author has been assisted greatly by the friendly and generous sharing of data by selenium investigators, worldwide. It is hoped that the Atlas reflects this knowledge fairly and accurately.
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Readers are cordially invited to submit new, or better quality maps, where they exist, to ensure the continuing up-dating of this Atlas. Mail to: Prof. James Oldfield, Dept. Animal Sciences, Oregon State University, Corvallis, OR 97331, U.S.A. (email: JamesEOldfield@orstedu)

We apologize for the deficient quality of some illustrations.

No suitable alternative material could be located.
Introduction

Selenium is a truly remarkable element. Occurring in only very small quantities - it is ranked 70th among the 98 elements that make up the earth’s crust - it has proven to be extremely useful to humanity in many and diverse ways. In the course of developing these useful applications, it has generated an impressive research effort. Reilly (1996) has estimated that some 100,000 technical papers have been published on it since its discovery in 1817. The Swedish chemist, Jons Jakob Berzelius, isolated selenium from red deposits in the lead chambers of a sulphuric acid plant, where it was apparently involved in worker illnesses. For some years it was regarded as a nuisance: a toxic element with no redeeming features.

But then, in the early years of this century, some industrial applications for selenium began to appear. It was found to impart a brilliant ruby-red color to glass, and in combination with other salts, other colors including brown, pink and yellow. Paradoxically, it would also remove unwanted greenish tints from glass and both glass and ceramic industries became users of significant quantities of it. Even so, the quantities of selenium used were initially small: only about 5,000 kg in 1910. During the years of World War II, selenium demand increased due to the cutoff of German imports of selenium rectifiers and to the discovery that selenium was an excellent substitute for manganese in the manufacture of glass. Then in the 1930’s the xerographic copying process was developed, using selenium, and market demands increased to about 2,300 metric tons annually.

But selenium was found to have important biological uses, too. About mid-20th century, a German biochemist, Klaus Schwarz, changed forever the concept that selenium was merely an environmental toxicant, when he showed that, in extremely small quantities, it performed as an essential nutrient (Schwarz and Foltz, 1950). Schwarz’s studies were done with laboratory rats but in short order they stimulated a surprising amount of research with domestic, food-producing animals which showed that a selenium deficiency was the underlying cause of a number of previously untreatable metabolic disease problems. In quick succession, exudative diathesis in chicks (Patterson, Milstreys and Stokstad, 1957), white muscle disease in calves and lambs (Muth et al., 1958), ill-thrift in calves and lambs (Drake, Grant and Hartley, 1960) and hepatosis dietetica in pigs (Moir and Masters, 1970) were found to be selenium-responsive. These observations, along with evidence that selenium could improve rates of growth (Oldfield et al., 1960) and of reproduction (Andrews et al., 1968) have made selenium supplementation an accepted and valued livestock production practice, virtually worldwide. The resulting widespread usage in both agricultural and industrial applications significantly increased the market demand for selenium, which became an item in commercial trade.

Selenium’s successful application in domestic animal production naturally raised questions about similar involvements with humans and these were emphasized by the discovery that two conditions occurring in China and eastern Russia - Keshan Disease, a cardiomyopathy, and Kaschin-Beck disease, an ankylosis (“big-joint disease”) resulted from selenium deficiency. Then, more recently, interest has intensified, with demonstrations that supra-nutritional levels of dietary selenium may protect against coronary disease and certain types of cancer.
Where Selenium Comes From

The ultimate source of all selenium is the rocks and soils of our terrestrial environment in which it is ubiquitous but by no means evenly distributed. Most soils contain from 0.1 to 2 ppm (parts per million) of selenium (Swaine, 1955) but it can vary from practically none to 100 ppm (Rosenfeld and Beath, 1964). Selenium can cause problems at both ends of its supply spectrum. At dietary levels of less than 0.1 ppm, various deficiency symptoms may appear in animals and the incidence of these increases sharply at levels below 0.05 ppm (NRC, 1983). On the other hand, plants or plant products containing more than 5 ppm selenium lead to easily-recognizable toxic symptoms (Williams et al., 1941), which suggests a tolerance range of about 100-fold (0.05 - 5.00 ppm). It is important, of course, to be able to recognize areas of selenium toxicity, as well as of deficiency. Some toxic levels of selenium occur from natural sources, where high selenium soils have had their selenium concentrated, by agricultural practices or by selenium accumulation in plants; others originate from industrial operations, including combustion of fossil fuels, or sulfide ore mining.

Methods of Determining Selenium Status

The assessment of selenium status in specific, localized areas can be approached in several ways. The total selenium in soils can be measured by chemical analysis and used as an indicator of selenium levels in indigenous plants, or animals consuming them, but it has some inherent problems. The biological availability of selenium to plants and subsequently to animals depends on a number of things, including the chemical form in which the selenium occurs, the acidity/alkalinity of the soil (pH) and the presence or absence of other elements, which may enhance or inhibit its absorption. Plant species are another consideration: some types of plants being much more efficient at removing selenium from the soil than others.

In alkaline, well-drained soils, selenium tends to form selenates (Se^{+6}) which are highly available to plants and may sometimes lead to their accumulation at toxic levels. But in acid and more poorly-drained soils, a ferric iron-selenite complex may form, which is only slightly available to plants. Such soils rarely, if ever, support the accumulation of toxic levels of selenium in plants grown on them (NRC, 1971). Elemental selenium (Se^0) is quite stable when it occurs in soils and is generally biologically unavailable. Apart from some slight microbial action, it seems to be not oxidized in the soil to forms that are more readily available to plants and animals (Watkinson and Davies, 1967).

Selenium Fertilization

The concept of adding selenium to fertilizer mixes to improve selenium status of deficient soils is not new. Although the first attempts at controlling selenium deficiencies in animals was by direct treatment of the animals themselves, either orally or by injection, it was not long before trials were run on the feasibility of correcting deficiencies by soil amendment. In 1973 investigators in New York state added sodium selenite (Na_2SeO_3) to soils to provide either 2.24 or 4.48 kg Se per...
hectare (Cary and Allaway, 1973). Corn, oats and various forage crops were grown and their selenium contents were generally above 0.1 ppm, which is sufficient to protect animals against selenium-deficiency disease, and the higher level of application produced selenium concentrations below those generally considered toxic. It was concluded that addition of about 2 kg Se/hectare as sodium selenite would be sufficient to produce forage crops that would protect animals from selenium-deficiency disease for at least 4 years after application. Dangers of toxic buildups of selenium at first worked against the use of sodium selenate (Fleming, 1962; Gissel-Nielsen and Bisbjerg, 1970) but later use of lower levels of application have been both effective and safe (Ylaranta, 1982). These Finnish studies showed that selenate was about 10 times as effective as selenite as a fertilizer amendment, and similar results were obtained more recently in Scotland (Shand et al., 1992).

Some farm management practices can affect the levels and availability of selenium in the soil. One example is the use of gypsum (CaSO₄), which in Oregon observations tended to aggravate the incidence and severity of white muscle disease in animals grazing on lands that are already low in selenium (Schubert, 1961). Very high levels of dietary copper (>800 ppm) or of zinc (>2100 ppm) tended to precipitate the effects of selenium deficiency in poultry (Jensen, 1975). On the other hand, some practices other than direct selenium application may enhance plant and animal selenium status. Thus, the use of superphosphate fertilizers tends to increase forage selenium levels since superphosphate itself is a source of selenium (Robbins and Carter, 1970). The extent to which this improvement develops depends ultimately on the selenium content of the phosphate rock from which the superphosphate was made. Aside from the inherent selenium in superphosphate preparations, however, the application of phosphorus, per se increases the availability of both natural and supplemental selenium to alfalfa (Carter et al., 1972). The practice of liming apparently can cause slight increases in the uptake of selenium by plants (Cary et al., 1967), whereas feeding elevated levels of calcium increased selenium retention (Lowry et al., 1985).

**Selenium in Plants**

A number of factors influence the selenium status of plants, and these have been reviewed by Johnson et al. (1967). The most influential of these is the nature of the plants themselves, and in this connection Wyoming workers have classified plants into three main groups - Primary and Secondary Selenium Accumulators, and Non-Accumulators (Rosenfeld and Beath, 1964). To illustrate the really remarkable ability to take up selenium from the soil by some of these plants, Beath (1937) reported finding a selenium level of 14,990 ppm in a sample of *Astragalus racemosus*, which is a primary accumulator. By contrast, most plants, even when grown on seleniferous soils, only contain about 10 ppm of selenium, or less. Robinson (1936) has recorded selenium concentrations of from 0.1 to 1.9 ppm in wheat samples gathered from all over the world.

Knowledge of the varying abilities of plants to mobilize selenium from the soil has led to a number of applications. Farmers in areas of selenium-deficient soils have learned to choose forage plants that can take up higher levels of selenium, so as to avoid selenium deficiency problems in their livestock. In New Zealand it was noted that a grass, Brown top (*Agrostis tenuis*) would accumulate 2 to 7 times as much selenium as white clover (*Trifolium repens*) (Davies and Watkinson, 1966).
At the other end of the supply scale, a process termed “Phytoremediation” uses plants to remove excess amounts of selenium from naturally seleniferous soils or from soils contaminated with selenium from industrial sources (Banuelos and Meek, 1990). Hybrid poplar trees (Populis tremula x alba) can transfer significant quantities of selenium to the air, with the volatilization rate being very much higher (230-fold) for selenomethionine than for selenite, and 1.5-fold higher for selenite than for selenate (Pilon-Smits et al., 1998).

Soil/Plant Relationships

In the course of investigations with selenium in soils and in plants, it became obvious that the chemical form in which the selenium occurred strongly influenced its bioavailability. Early administration of selenium to animals favored the use of selenite, mainly for reasons of safety, but it is now generally acknowledged that selenate is more available biologically. In Finnish trials with selenium added to fertilizers, it was shown that a given response was obtained with only a tenth as much selenate as when selenite was used (Koivistoinen and Huttunen, 1986). Reduction of selenium from higher oxidation states occurs both in the soils and in the course of metabolic processes in animals. This lowers the bioavailability of the selenium residues and helps prevent any toxic accumulation of the element. It also means that selenium supplementation by fertilizer amendment has to be a continuing process.

Recent studies with both plants and animals tend to favor the supplementary use of organic selenium preparations – most frequently selenium-enriched yeast. It has been shown repeatedly that organic selenium is more highly available to animals and humans than are inorganic forms (Ortman and Pehrson, 1998; Mahan et al., 1999). This, in turn, argues for the use of selenate-enriched fertilizers which would then produce feed and forage crops with enhanced levels of readily-available organic selenium for animal/human use, since crop plants convert selenium from inorganic to organic form.

Selenium Status in Animals and Humans

The selenium status of animals is commonly assessed by determining selenium levels in their blood. In early studies (Hartley, 1967) recorded levels of 0.05 µg Se per liter of blood in normal sheep, as compared with 0.016 µg Se per liter in animals with symptoms of white muscle disease, and 0.01 in animals exhibiting “ill-thrift.” Liver selenium levels have sometimes been promoted as indicators of Se storage, and Allaway et al. (1966) have proposed 0.21 µg Se/kg as the critical hepatic content, below which white muscle disease in animals may be expected. Hair has sometimes served as a non-invasive sampling site for monitoring selenium status (Oster et al., 1988), as has the use of toenails (Ovaskainen et al., 1993).

There is a close association between intake of dietary selenium and the level of activity of glutathione peroxidase, in several organs, however such activity is affected by several nutritional and environmental fractions (WHO, 1987). A useful comparison of three methods for assessing the selenium status of cattle has been presented by Waldner et al. (1998). Serum Se, whole blood Se
and glutathione peroxidase levels can all be run on a commercial basis and are available to veterinary clinicians. The authors conclude that whole blood or serum Se status is most consistent when measured at a herd level.

There has been increasing interest recently in the selenium status of humans, since its health-protective effects have become better understood. Alaejos and Romero (1993) recommend urinary selenium concentrations as an index and suggest that 24-hour excretions of 10-200 µg Se/day are not associated with problems of either deficiency or toxicity. Thomson et al., in New Zealand, have also provided useful information on 24-hour urine sampling. Belgian workers have suggested that selenium levels in capillary whole blood might be used to assess selenium status of children (Van Dael et al., 1994). Capillary blood values did not differ significantly from venous blood levels and were easier to obtain. The foregoing, which is no more than a minuscule sampling of the vast literature on selenium, attests to the usefulness of this micronutrient element in the production of domestic food animals and in protecting the health of both animals and humans.

These benefits are significant enough that government agencies in two countries, Finland and New Zealand, have supported use of selenium in fertilizers on a national scale. Such decisions were only made after careful study of the countries’ selenium status and the results have been equally carefully monitored (Ekholm et al., 1991; Watkinson, 1987). Studies in some other countries, e.g. Denmark (Gissel-Nielsen, 1986) have provided extensive background data but have not yet led to national supplementation practices. Undoubtedly other countries will follow suit (see, for example, Rayman, 1997).

Selenium in Maps

Some of the many investigators of selenium have accompanied their accumulated data with maps of their countries illustrating where selenium deficiency, adequacy or toxicity may be expected to be encountered. In other cases, sections of countries have been mapped where the complete picture is unavailable. We have attempted to assemble such diagrams as far as possible, along with some written assessment of selenium distribution. The best part may well be the reference citations, which will key readers to the original research.

Historical

Interestingly, some mapping of selenium’s effects, as we now know them, particularly those occurring at the toxic end of the supply scale, were drawn before the element was definitely identified. Looking back over time, the first such map was probably one prepared by Marco Polo to document his travels to remote parts of China for Kubla Khan in the 13th century (Figure 1).

Polo commented in a journal which he prepared many years later, with reference to problems encountered by merchants of the time who travelled the “silk road,” that in the area of the modern province of Shanxi, “… they cannot venture among the mountains with any beasts of burden, on account of a poisonous plant growing there, which if eaten by them has the effect of causing the
hoofs of the animals to drop off” (Polo, M. translation, 1967). It is generally believed today that the plant involved was one of the selenium accumulators described earlier which concentrates selenium salts to toxic levels (Trelease, 1942) although the specific plant type is unknown.

**Australia**

Australia has been a center for microelement research, to a considerable extent due to the genius and untiring labors of E.J. Underwood (1971), whose classic publication, “Trace Elements in Human and Animal Nutrition” is generally recognized as a fundamental reference source. It is significant, too, that the ingenious “heavy pellet” method of administering supplementary selenium to ruminants was developed in Australia (Kuchel and Buckley, 1969).

It has been known for some years that Australia has areas of both inadequacy and toxicity of selenium. Peterson and Butler showed that seeds of *Neptunia amplexicaulis* from seleniferous
soil areas in central Queensland, contained 123 µg Se per seed, while ‘change hoof disease’ has been attributed to horses grazing a known selenium accumulator, Morinda reticulata, also in Queensland (Knott and McCray, 1959). It has been commented that management of Se-inadequate areas seems more feasible than of the toxic ones, since the former are amenable to such agricultural practices as plant species selection, fertilization and irrigation. Sometimes supplemental feeding of crops from Se-adequate or even toxic areas may be helpful (Johnson, 1975). Conor Reilly (1992) has commented that selenosis is not an important problem in Australia, occurring chiefly in remote areas where agriculture is not a major occupation. Of far greater consequence are regions of selenium deficiency, which occur right across Australia. Reilly observes that although there is no evidence that selenium deficiency in humans is a widespread problem in Australia, “perhaps there are grounds for its use pharmaceutically, as a protective agent.”

A map has recently been produced showing areas country-wide where farm animals may be at risk to either selenium inadequacy, or toxicity (Judson and Reuter, 1998). These authors comment that “soil tests are an unreliable guide to the selenium status of animals, and the test is not offered commercially in Australia” (Figure 2).

Figure 2. Areas in Australia where livestock may be at risk from selenium deficiency or toxicity, from Judson and Reuter, 1998
Selenium deficiency symptoms are usually apparent in the winter rainfall areas of southern Australia where animals are ranged on acid soils receiving more than 500 mm of annual rain. In New South Wales, selenium deficient livestock are found on the Central and Southern Tablelands and Slopes (Hart, 1985) and on the Northern Tablelands (Langlands et al., 1981). In Queensland there are selenium-deficient areas on the southeastern coast, associated with tertiary volcanic soils (Noble and Barry, 1982).

Areas at risk to Se Deficiency or Toxicity Reported

Detailed maps have been prepared for some localized areas where selenium problems exist, both deficiencies and toxicities. Figure 3 links incidence of white muscle disease, a selenium-responsive disorder, to the local geology in West Australia.

Figure 3. Geology of southwestern Australia, showing occurrence of white muscle disease, a selenium-responsive disorder, from Godwin, 1975
At the other end of the supply spectrum, incidence of selenium toxicity in an area termed the “Poison Strip” has been plotted on a map of Queensland (Figure 4). Here, a seleniferous, limestone shale supports the growth of selenium-accumulator plants (McCray and Hurwood, 1963).

A more extensive map of Queensland (Figure 5) identifies the spectrum of selenium levels in crops from severe deficiency to toxicity. The toxicity area noted in Figure 4 is represented in the solid black circles near the top center of this map.

Figure 4. The Tambo Formation (hatched), a high-selenium area in Queensland, from McCray and Hurwood, 1963
Figure 5. Selenium levels in cereal grains and soybeans from central and southern Queensland, from Noble and Barry, 1982.
Low selenium areas have been quite precisely proscribed for the state of Victoria (Figure 6), and selenium status of the blood of livestock on Kangaroo Island, off the coast of South Australia has also been plotted (Figure 7).

Figure 6. Marginal and low selenium areas in Victoria, Australia (from Hosking et al., 1990)

Figure 7. Areas on Kangaroo Island, South Australia, where selenium in animal blood is deficient, borderline, or adequate for normal health (Judson and Obst, 1975)
New Zealand

New Zealand is properly acknowledged as a pioneering area for biological research with selenium. Much of the country has soils that are selenium-deficient because of volcanic parent material (North Island) or from excessive leaching (South Island). The stimulus for selenium supplementation in New Zealand came from its animal agriculture community which supports a significant part of the country’s economy. As Christine Thomson, a prominent research scientist at the University of Otago has put it, “New Zealand has brought together one of the largest sheep populations and one of the most-selenium deficient environments in the world.” There is no question, also, that sheep have served as a very important and useful animal model in selenium research (Thomson, 1989). The total selenium in the topsoils of New Zealand has been listed in Maps 89 and 90 of the New Zealand Soil Bureau Atlas (Wells, 1967) and these are reproduced as Figures 8 and 9. The range of selenium concentrations is from 0.1 to 4.0 ppm and Watkinson (1962), who pioneered in soil selenium analysis and development of selenium fertilization technology, notes that where soils contain less than 0.5 ppm, there is a high incidence of selenium-responsive disease in sheep.

The following maps (Figs 8 and 9) indicate extensive areas of selenium deficiency in New Zealand. They were drawn over three decades ago, however, and current evidence suggests that even larger areas of deficiency are involved (Turner, personal communication, 1999). Soil maps are not infallible sources of information on available selenium, and some problems with them have been pointed out by Shirer (personal communication, 1999); soil maps present values for total selenium, much of which is probably elemental or selenite, and largely unavailable to plants. Moreover, increased use of fertilizers and improved farming methods have increased the production of forage dry matter on New Zealand farms, and this tends to dilute the available selenium, so that areas that were previously Se-adequate have now become borderline Se-deficient. Also, the levels of blood selenium recommended for optimum production of meat and milk have been steadily increased and this extends areas of deficiency.

Continuing studies of localized areas have more precisely identified deficiencies of selenium and other trace elements in forage crops (Grace et al., 2000). Moreover, it has been shown that the selenium status of dairy cows can be assessed from milk-selenium concentrations (Grace et al., 2001).

Drs. Andrews, Hartley and Grant (1968) mapped areas in New Zealand where selenium-responsive diseases occur in livestock (Figure 10) and it is noteworthy that this and the soil-selenium maps are almost identical.

Watkinson (1974) has observed that cereal grains are often grown on low-selenium soils in New Zealand, and that they generally contain about one-tenth as much selenium as grains from Canada and the U.S., from which New Zealand imports grain. In years when New Zealand experiences crop failures, increased importation of higher-selenium wheat from Australia significantly increased levels of selenium in human blood (Robinson and Thomson, 1988). Because of the beneficial effects of selenium supplementation on the productivity of their animal industries, New Zealanders have enthusiastically adopted the technology of selenium fertilization and some
Single Factor Map showing
TOTAL SELENIUM IN TOPSOILS

Figure 8. Total Selenium in Topsoils: North Island, New Zealand. Reproduced with permission from Manaaki Whenua Press, Lincoln, New Zealand, August 1999
Figure 9. Total Selenium in Topsoils: South Island, New Zealand. Reproduced with permission from Manaaki Whenua Press, Lincoln, New Zealand, August 1999
1.2 million hectares of an estimated 4.5 million hectares of selenium-deficient farmland were so fertilized in 1998. Despite the obvious benefits to animal production, selenium supplementation has not been officially recommended as a human health practice in New Zealand, although doubtless considerable of it does occur. It will be interesting to see whether this concept will change in the future, as attention is directed toward health-protective effects of selenium, given at slightly higher than the earlier-established “nutritional” levels (see Combs, 1998). Some sections of the New Zealand human population, e.g. infants, do seem to be at risk of selenium deficiency (Dolamore et al., 1990). Investigators at Otago University, who have long monitored selenium nutrition in New Zealand, suggest that research showing that higher selenium intakes may be beneficial for protection against cancer and psychologic function needs to be considered in future deliberations on selenium requirements (Duffield et al., 1999).

Figure 10. Liveweight responses of lambs to selenium supplementation (from Andrews et al., 1968)
Europe

The various European countries have had a long and productive history of investigation into selenium’s effects on animal and human health. One of the earliest to investigate both selenium’s protection against white muscle disease and also its beneficial effect on the growth rate of lambs was Dr. Kenneth Blaxter at the Hannah Dairy Research Institute and later the Rowett Institute in Scotland (Blaxter, 1962). And in Denmark, selenium status of soils and plants and early observations on the efficacy of selenium fertilization were assessed by Gissel-Nielsen (1987), who mapped selenium status of European crops (Figure 11).

The Scandinavian Countries

Extensive studies of selenium problems in animal nutrition have been carried out in Norway, and much of their findings has been summarized in a supplement to the Norwegian Journal of

Figure 11. Selenium status of forage crops in Europe, from Gissel-Nielsen, 1987
Agricultural Sciences (Froslie, 1993). This centered on animal, rather than human nutrition, and has led to extensive supplementation of domestic animal diets, but not up to this point, to widespread human use. Actually, symptoms of what we now recognize as a selenium-responsive myositis were observed very early (Slagsvold and Lund-Larsen, 1934). Soils of Norway are generally recognized as low, or very low, in selenium, except for a strip of the country along the west coast (Wu and Lag, 1988). Selenium supplementation of animal feeds was begun, in Norway, in 1980 and has improved both selenium blood levels and animal production. This has resulted in an increase of human selenium intake of 5-10 \( \mu \text{g} \)/day (Froslie et al., 1985). Overnes (1993) has provided a good summary of the selenium status of Norwegian livestock, including a map that originated with Froslie (1979) (Figure 12).

In Sweden, concern has been raised about the effects of industrialization and subsequent “acid rain” on the environment. Specifically, “black lakes” have been identified, in which fish exhibit a high mercury content. An interesting technology was developed, whereby the lakes were
seeded with selenium, which effectively tied up the mercury and saved the fish (Bjornberg, 1989). A Swedish study recently examined the effects of increasing acidity on the selenium content of forest soils (Gustafsson et al., 1993). At sampling sites mapped in Figure 13, they showed definite lowering of soil pH but no increase in soil selenium, apparently because adsorbed organic anions competed successfully for adsorption sites.

Supplementation of growing/fattening diets for pigs with either inorganic (sodium selenite) or organic (selenized yeast) sources of selenium showed no advantages in weight gains or glutathione peroxidase activity (Ortman and Pehrson, 1998). Human selenium status appeared similarly satisfactory; however, Svikumar et al. (1992) suggest an advantage to selenium supplementation of a lactovegetarian diet.

Extensive studies of selenium supplementation in domestic animal nutrition in Sweden, with emphasis on the higher bioavailability of organic over inorganic sources, have been assembled by Ortman (1999). Finland was the first European country to permit the addition of selenium to animal feeds, and the extent of selenium-deficiency there was well documented (Frost, 1989).
Plant uptake of selenium from easily-leached podosols in Finland was generally poor, and a study of selenium contents of Finnish hay crops showed them generally deficient in terms of ability to sustain animal health (Sippola, 1979; Figure 14).

Mean values of selenium in Finnish hay samples were 0.014 ppm and for grains, 0.007 ppm (dry weight basis). Because of these low selenium levels, and of incidence of muscular degeneration in Finnish livestock (Oksanen, 1965), veterinary authorities approved the addition of selenium to commercial animal feeds in 1969 (Makela et al., 1993). However, unlike the parallel situation in New Zealand, the Finns became concerned about effects of low dietary selenium on the human population. It was found in the 1970’s that the average intake of selenium by the Finnish population varied somewhat with the extent to which cereal grains were imported, but generally was about 25 µg Se/person/day (Varo, 1993). Compared with a generally-recommended intake of about 70 µg Se/day, this represented a deficiency state and after careful study and discussion the Ministry of Agriculture and Forestry decided, in 1984, that from that point onward, all multielement fertilizers should be supplemented with sodium selenate. At first, two levels were proposed: 6 µg Se/kg fertilizer for grass and hay crops, and 16 µg Se/kg fertilizer for grains. The results were carefully monitored, and in 1991 the supplementation practice was adjusted to the one, lower level of 6 µg Se/kg of fertilizer for all crops. Ten years after the fertilization supplementation had begun, the selenium levels in feeds, foods and human blood had all risen to satisfactory levels and no safety problems had been recorded (Varo et al., 1994). One of the monitoring studies attested to the efficacy of the fertilizer supplementation program in terms of improved selenium content of human milk, and also identified an area of low selenium status in neighboring Estonia (Kantola, et al., 1997).

The selenium status in Denmark is shown, in general terms, in Figure 12, however a considerable mass of selenium data has been recorded, particularly by Gissel-Nielsen (1975, and Gissel-Nielsen et al., 1984). As a country, Denmark is considered to be selenium-deficient.

Figure 14. Average content of selenium in hay samples from different sites in Finland. A completely-filled circle indicates 0.04 ppm Se in the plant dry matter.
Poland

Bronislaw Zachara, long an effective investigator of selenium status of humans, has provided a

*Figure 15. Selenium concentrations in breast milk from mothers in various provinces of Poland. The black circles indicate provincial capitals; other figures show selenium concentrations.*
The British Isles

The United Kingdom, though small in total land area, shows wide variations in soil selenium levels. Some of the most highly seleniferous soils in the world are in Limerick, Tipperary and Meath counties in Ireland - containing up to 1250 ppm Se, which is about 10 times the level in the most highly seleniferous soils of the United States (Fleming, 1962; Fleming and Walsh, 1957). On the other hand, soils in northern England and Scotland tend to be selenium deficient (Sharman et al., 1959). Indeed, selenium levels in many human foods in the British Isles are low enough that concerns have been raised about their effects on health of the human population. Extensive studies have examined the selenium content of UK foods (Barclay et al., 1995). The Ministry of Agriculture, Fisheries and Food (MAFF) which monitors the nutrient values of the nation’s food supply on a continuing basis has estimated that the average daily intake of selenium was 42.8 µg/person in 1991, 38.5 µg/person in 1994, and from 29-39 µg/person in 1995 (MAFF, 1997). There was concern, therefore, not only about the low Se intake level, as compared with the calculated Reference Nutrient Intake (RNI) values of 60 µg/day for adult women and 75 µg/day for adult men, but also for the apparent downward trend for dietary selenium, which was attributed to increased use of European, rather than American wheat. Rayman (1997) has proposed that there is a need for governmental action to use supplementary selenium on a national scale and has provided a useful discussion of the impacts of selenium on human health in the U.K. (Rayman, 2000). John Arthur (1999) reminds us, from Scotland, of the metabolic relationship between selenium and iodine, and of the need for an adequate selenium status in areas of both selenium and iodine deficiency.

Germany

Selenium status in Germany has been studied by a unique procedure in which mosses from various areas of the country have been analyzed. This extensive study included samples from 1026 locations and the results are shown in Figure 16 (Siewers, U. and U. Herpin 2001). Generally speaking, Germany appears to resemble other parts of Europe, tending towards low levels of selenium in soils and plants.
Figure 16. Selenium contents of mosses in various locations in Germany.
Spain

The European map (Fig. 11) does not exhibit selenium status zones in Spain or Portugal; however, some comprehensive studies have been done and their data published (Torra et al., 1996). Healthy human adults in Barcelona, between the ages of 25 and 70, had serum selenium concentrations averaging 80.7 µg/L, with a range of from 60-106 µg/L. Significantly reduced selenium concentrations (55.9 µg/L; range 29-97 µg/L) were found in intensive care units on parenteral nutrition. The authors characterize their Barcelona serum selenium concentrations as being in the “intermediate range of selenium status.” On this basis, they classify Spanish selenium status as safe, but urge its supplementary use in cases having total parenteral nutrition. We are indebted also to Spanish workers for an excellent and comprehensive review of methods of analysis of selenium in body fluids, which includes some most helpful suggestions on how samples should be taken and stored (Alaejos and Romero, 1995).

A survey of Spanish subjects in the Canary Islands indicate dietary selenium intakes lower than the American Recommended Dietary Allowances (Romero et al., 2001), while in the Portuguese Azores archipelago, serum selenium concentrations averaging 88 µg/L were reported (Viegas-Crespo et al., 2000).

Greece

In Greece, selenium status has been assessed by measuring its content in alfalfa and various cereal grains and this has produced selenium maps, such as shown in Figure 15 (Bratakos and Ioannou, 1989). Since grains and their byproducts account for nearly half the daily intake of selenium by the Greek population, this serves also as an indicator of the status of the people. Cooking causes some losses of food selenium and it has been estimated that losses from fish are 36-46%, meats 13-41%, cereals 20-30%, vegetables 12-37%, and pulses and cereal products 5-10% of the original selenium content (Bratakos et al., 1988).

The same authors (Bratakos and Ioannou, 1989) have mapped selenium content of Greek soils (Figure 17).

A further, comprehensive list of selenium contents of Greek foods and of tap water and bottled water (Figure 18) has been prepared by Bratakos et al. (1987).

![Figure 17. Selenium content of Greek soft white wheat, from 93 locations. The selenium ranged from 0.019 to 0.528 ppm](image-url)
From these various studies, it was concluded that the average daily intake of selenium by Greeks was in the order of 110 µg/day - a level that indicates neither deficiency nor toxicity (Bratakos et al., 1990). No areas of toxic soil selenium in Greece were recorded.

Figure 18. Soil selenium in Greece, indicating availability of Se to plants

Figure 19. Selenium contents of tap water (tw) and bottled water (bw) in Greece
Turkey

Although it was not accompanied by a map, a very interesting assessment of the selenium status of human diets in Turkey, through analyses of milk and milk products, has been assembled by Orack and Yanardag (1996). In it, they compare milk selenium values in Turkey with those in some 15 other countries. In Turkey, the mean selenium level in cows’ milk was found to be 20.07 ± 6.93 ng/g, but there was considerable variation, from 11.28 in Adiyaman to 36.05 ng/g in Denezli. Cheeses added some selenium to the Turkish diet. The highest value was found in Lor cheese, at about 106 ng/g.

The Balkans

A good deal of most useful research on the selenium status of the Balkan countries has been conducted by Maksimovic and much of it was summarized at a conference convened by the Serbian Academy of Sciences and Arts (Maksimovic et al., 1995). Mean serum selenium levels have been correlated with endemic areas for kidney disease in a map (Figure 20).
Selenium content of soils in Yugoslavia varied over a broad range (39-44 νg/kg) and it was concluded that soils contain inadequate amounts in many agricultural regions (Jovic, 1996).

In Serbia, the investigators provided data on Se contents of soils, cereal crops and garlic and noted that “all data indicate a serious selenium deficiency.” In some regions, they reported that the Se levels in garlic, grains and human serum and hair were approaching those in the low-selenium zone of China. Mean values of serum selenium in Serbia have also been mapped (Figure 21).
Some of the lowest serum Se values in Europe have been reported from Serbia - lower than in the neighboring countries of Bulgaria (54.8 µg/L), Hungary (55.8 µg/L), Slovenia (57.0 µg/L) and Croatia (64.2 µg/L) and it was felt that selenium deficiency might be a contributing factor to high incidence of human cancers in some areas (Maksimovic and Djujic, 1998).

In Slovenia, which is bordered by the Alps, the Dinaric mountains, the Panonian lowlands and the Mediterranean, concern is less for dietary Se, which appears adequate, than for contaminant Se in the atmosphere from industrial emissions in their own and adjoining countries. They have analyzed lichens, as indicators of atmospheric status, and found average values of 0.27 ppm, which they describe as “in agreement with reported literature values” and lower than biomonitoring levels (with a different organism) in the Netherlands (Jeran et al., 1996).

Russia

Russia’s huge land area has been less well-documented as to selenium status than some other countries, but some excellent studies have been done, providing helpful means of assessment. A joint program conducted by the Institute of Nutrition/Russian Academy of Medical Sciences and the Ministry of Trade and Industry in Finland addressed the human Se status in many areas in Russia, in 1988-1990 (Golubkina and Alfthan, 1999). Serum samples were obtained from 2,462 healthy individuals aged 20-53 years, in 125 towns and settlements from 27 geographical regions of Russia (Figure 22). The lowest selenium value, 0.91 mM/l serum, came from Pscov in the west, while the highest value, 1.74 mM/l serum came from Sakhalin Island, in the east. The various selenium values are presented in Table 1. Further data were gathered on the selenium contents of important food items, like wheat flour and dried milk. Selenium in wheat flour ranged from 44-557 µg/kg, and in dried milk from 38-115 µg/kg. The dried milk was of Russian origin but the wheat flour included both indigenous and imported samples.
Although no very low or very high selenium values were found in this extensive study, still there are other, vast territories in Russia where selenium status remains to be charted. It would be unusual if some areas of Se-deficiency or toxicity did not occur in this vast country – the largest in the world.

Table 1. Regions in Russia Sampled in Assembling Selenium Status Data

<table>
<thead>
<tr>
<th>Region (Town)</th>
<th>Number of Samples</th>
<th>Year</th>
<th>Mean Serum Se µM/l</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 • Pskov</td>
<td>27</td>
<td>1995</td>
<td>0.91</td>
</tr>
<tr>
<td>2 • Irkutsk</td>
<td>52</td>
<td>1996</td>
<td>0.95</td>
</tr>
<tr>
<td>3 • Novgorod</td>
<td>320</td>
<td>1995</td>
<td>1.04</td>
</tr>
<tr>
<td>4 • Riazan</td>
<td>20</td>
<td>1994</td>
<td>1.05</td>
</tr>
<tr>
<td>5 • Briansk</td>
<td>150</td>
<td>1991</td>
<td>1.06</td>
</tr>
<tr>
<td>6 • Khabarovsk</td>
<td>20</td>
<td>1992</td>
<td>1.08</td>
</tr>
<tr>
<td>7 • Altay</td>
<td>253</td>
<td>1994</td>
<td>1.11</td>
</tr>
<tr>
<td>8 • Tula</td>
<td>40</td>
<td>1992</td>
<td>1.13</td>
</tr>
<tr>
<td>9 • Smolensk</td>
<td>15</td>
<td>1993</td>
<td>1.15</td>
</tr>
<tr>
<td>10 • Karelia</td>
<td>118</td>
<td>1991</td>
<td>1.14</td>
</tr>
<tr>
<td>11 • Bashkiria</td>
<td>188</td>
<td>1992</td>
<td>1.16</td>
</tr>
<tr>
<td>12 • Arkhangelsk</td>
<td>95</td>
<td>1991</td>
<td>1.18</td>
</tr>
<tr>
<td>13 • Mari-El</td>
<td>146</td>
<td>1992</td>
<td>1.22</td>
</tr>
<tr>
<td>14 • Leningrad</td>
<td>20</td>
<td>1992</td>
<td>1.23</td>
</tr>
<tr>
<td>15 • Moscow</td>
<td>220</td>
<td>1993</td>
<td>1.26</td>
</tr>
<tr>
<td>16 • Vladimir</td>
<td>80</td>
<td>1993</td>
<td>1.26</td>
</tr>
<tr>
<td>17 • Sverdlovsk</td>
<td>245</td>
<td>1992</td>
<td>1.27</td>
</tr>
<tr>
<td>18 • Vologda</td>
<td>200</td>
<td>1992</td>
<td>1.27</td>
</tr>
<tr>
<td>19 • Cheliabinsk</td>
<td>240</td>
<td>1992</td>
<td>1.28</td>
</tr>
<tr>
<td>20 • Krasnoyarsk</td>
<td>40</td>
<td>1991</td>
<td>1.29</td>
</tr>
<tr>
<td>21 • Tver</td>
<td>40</td>
<td>1993</td>
<td>1.29</td>
</tr>
<tr>
<td>22 • Murmansk</td>
<td>301</td>
<td>1991</td>
<td>1.30</td>
</tr>
<tr>
<td>23 • Peran</td>
<td>71</td>
<td>1992</td>
<td>1.30</td>
</tr>
<tr>
<td>24 • Kaloga</td>
<td>155</td>
<td>1992</td>
<td>1.32</td>
</tr>
<tr>
<td>25 • Gorky</td>
<td>20</td>
<td>1990</td>
<td>1.37</td>
</tr>
<tr>
<td>26 • Novosibirsk</td>
<td>20</td>
<td>1997</td>
<td>1.47</td>
</tr>
<tr>
<td>27 • Sakhalin Is.</td>
<td>58</td>
<td>1991</td>
<td>1.74</td>
</tr>
</tbody>
</table>

* Standard deviation figures are omitted. (From Golubkina and Alfthan, 1999).
India

Occurrence of toxic levels of selenium has been identified by workers at Punjab Agricultural University in India (Dhillon and Dhillon, 1991). This followed observation of selenium toxicity symptoms in animals and in wheat and involved analyses of soils, irrigation water, plants and animal tissues. Seleniferous areas showed selenium concentrations in soils that were 4-5 times higher than in non-seleniferous areas. Comparative values averaged $2.12 \pm 1.13$ and $0.42 \pm 0.24 \mu g$ Se/kg in surface soils and $1.16 \pm 0.51$ and $0.32 \pm 0.27 \mu g$ Se/kg in seleniferous and non-seleniferous areas, respectively. Sampling sites are located in Figure 23.

![Figure 23. Map identifying Se toxic areas (black circles) in Jalandhar and Hasiapur districts of Punjab, India.](image-url)
Attention has focused on sites of selenium toxicity in India and Dhillon and Dhillon (1997) have mapped sampling sites for selenium analyses of soils in the Punjab in northwest India (Figures 23 - 24). In seleniferous and non-seleniferous areas of the Punjab, total selenium in soils ranged from 0.31-4.45 µg/kg and 0.08-0.55 µg/kg, respectively. Soils containing 0.5 µg Se/kg, or more, are considered toxic.

![Figure 24. Locations of selenium-toxic soils in the Punjab.](image)

**Bangladesh**

The literature contains references to a human health problem involving high levels of arsenic intake in Bangladesh (Mudur, 2000). Since selenium has been found to have a protective effect against arsenism (Wang et al., 2001), it may be assumed that the soils and food plants in Bangladesh would tend to be low in selenium. A proposal for research to investigate this situation has been advanced (Spallholz and Boylan, 2002).
Sri Lanka

Soil selenium and iodine concentrations in soils in Sri Lanka are average to marginal compared to soils elsewhere. Interestingly, the highest values for selenium and iodine occur in the Wet Zone in southwest Sri Lanka, where goitre is prevalent, thus emphasizing the importance of the bioavailability of these two nutrient elements (Fordyce et al., 2000).

Figure 25. Soil selenium and iodine concentrations in soils in Sri Lanka

North America

Some of the earliest observations of selenium status were carried out in the United States, stimulated first by concerns about livestock poisoning involving excess Se (Moxon, 1937) and later by concerns about deficiencies, as it became evident that the myopathy “white muscle disease” was selenium-responsive (Muth et al., 1958). Much of the pioneering analytical work was done by Allaway (1972) and his colleagues at the Plant, Soil and Nutrition Laboratory of the U.S. Department of Agriculture, on the Cornell University campus. Out of this very extensive compilation of analytical data came a selenium map for the U.S. (Figure 26), which has been widely reproduced (see, for example, Oldfield, 1990).
Figure 26. Distribution of selenium in soils and crops of the United States. This map shows general areas where selenium is deficient, adequate or excessive, as regards normal animal health, from Oldfield, 1990.
Most of the maps in this Atlas emphasize areas of selenium deficiency where supplementation can bring positive results in health and animal productivity. We were reminded of the toxic dangers of excess selenium in the 1980’s, however, when abnormalities in the young of wild water fowl at the Kesterson National Wildlife Refuge in California’s Merced County suggested presence of toxic levels of selenium. Figure 27 shows the location of the San Luis Drain service area in the San Joaquin valley, which picked up selenium and other salts from irrigation runoff and conveyed them to ponds at Kesterson (Tidball et al., 1989). The excess selenium originated in the Panoche hills area and it is interesting that the toxicity problems were quite localized and beneficial responses in livestock to selenium supplementation were obtained in closely adjacent areas.

![Figure 27. Areas of selenium toxicity in California’s San Joaquin valley. The Kesterson Refuge is at the north end of the shaded area (Tidball, 1989)](image-url)
Canada

The US studies were quickly extended to include neighboring areas in Canada, thanks to work by Lessard et al. (1968), Walker (1971), Winter and Gupta (1979), Miltimore et al. (1975) and a number of others, and this has led to an expanded map (Figure 28).

Figure 28. Selenium contents of forages and grains, relative to their adequacy for animal health, in Canada and the United States (after NRC, 1983)

This map is broadly generalized and suggests that in Canada areas of selenium deficiency tend toward the east and west coasts, with the central, prairie provinces being largely adequate. There are many localized variations, however, and persons interested are referred to the literature citations for more exact data.
A useful selenium map for feedstuffs in the Maritime provinces has been provided by Agriculture Canada, for example (Winter and Gupta, 1978) and is shown here as Figure 29.

![Figure 29. Selenium in Canadian maritime province feedstuffs](image)

In Canada’s smallest province, Prince Edward Island, the selenium content of forage and cereal grains varied from 0.004 to 0.043 ppm - concentrations that are considered deficient in livestock feed (Gupta and Winter, 1975). Some interesting observations have been made in Canada’s far north where selenium levels in musk oxen liver (average 0.10 ppm) were determined on Victoria Island in the Arctic ocean (Salisbury et al., 1992). These values would be considered deficient for cattle and sheep.

Recent evidence from the four provinces, British Columbia, Alberta, Ontario and Quebec, confirmed the inverse relationship between cancer incidence and selenium status (Morris et al., 2001). Protective effects appeared to be maximized at between 0.9 and 1.0 ppm Se, using the toenail monitor. Selenium status declined from west to east in Canada.
China

Much of our knowledge of the biological effects of selenium has been gained in China where research on selenium has continued, on a broad scale, for many years. Like Canada and the United States, China is a very large country and it is not surprising that wide variations in selenium status exist in the soil, indigenous food and forage plants, and in the tissues of animals and humans. In fact, China, rather than known selenium-low countries like Finland and New Zealand, was the first world site to record selenium deficiency disease in humans. **Keshan Disease** is an endemic cardiomyopathy that was first reported in 1935 in Keshan county, of Heilongjiang province, in the northeast corner of China (Yang et al., 1984). Medical interest in the disease was heightened by its discovery in a number of different locations (Reilly, 1996) and several medical teams were assembled to study it. Eventually, Keshan disease was found to be prevalent in a broad zone running from northeast to southwest, from the border of Heilongjiang province and Russia in the north, to that of Yunnan province and Myanmar (Burma) in the south. The areas of incidence have been roughly mapped (Figure 30). Interestingly, selenium deficiency (Keshan disease) and selenium toxicity occur within 20 km of each other in Enshi District (Fordyce et al., 2000). Availability of selenium to plants is apparently inhibited by adsorption onto organic matter, or Fe oxyhydroxides in the soil which suggests that application of Se-fertilizers may not improve crop selenium levels as much as expected. Relating selenium to local soil geology as well as to incidence of Keshan disease revealed the surprising fact that in certain villages in Hebei province, the highest selenium levels were associated with the highest incidence of Keshan disease, which emphasizes that the amount of soil selenium is less important than its biological availability (Johnson et al., 2000).

![Figure 30. Regions of endemic Keshan Disease in China (from Ge and Yang, 1993)](image-url)
Although Keshan disease responds positively to selenium supplementation, which has eased the lives of large numbers of sufferers, it appears that other causative factors may apply, too. One of these is the presence of Coxsackie virus, and this has led to the very interesting observation that this virus becomes pathogenic when it operates in a selenium-deficient environment (Beck et al., 1994). The levels of selenium in individuals suffering from Keshan disease are certainly low: scalp hair from persons in Keshan disease areas contained an average of 0.074 µg Se/g, as compared with 0.343 µg Se/g in non-Keshan disease areas (Wang et al., 1979). A separate condition, Kaschin-Beck (“big-joint”) disease is also believed to be selenium-related and generally occurs in the Keshan disease belt across China.

At the other end of the supply scale, selenium poisoning has been recognized in China for an even longer time than selenium deficiency. People in Enshi county of Hubei province in south China showed symptoms - thickening of nails and loss of hair - that had been associated with excess selenium (Yang, G.Q., 1982). Small pockets of selenium toxicity have been identified in Enshi county, where mean concentrations of selenium in soil and in corn grain are 4.06±1.24 µg/g and 6.47 µg/g and 6.45 µg/g, respectively (Zhu and Zheng, 2001). It has been suggested that Se may be essential for corn growth Concurrently, another selenotoxic area was found in Shanxi province (Chen and Mei, 1980).

The extensive data available on selenium content of Chinese plants have been plotted in map form by Liu et al. (1987), and are presented here as Figure 31.
Figure 31. Selenium content ranges in Chinese feeds and forages (Liu, et al., 1987)

More recently, an extensive map, charting the distribution of selenium in feedstuffs and forages in China has been prepared by Yuquin Duan of the Chinese Academy of Animal Science in Beijing (personal communication, 1998) and this is presented as Figure 32.

Data used in the assembly of this map (Fig. 30) were gathered over three years, and included analysis of 11,473 feed and forage samples including corn, barley, sorghum, millet, potatoes, oilseed meals, wheat bran, rice polishings, alfalfa, orchard grass, wheat grass, fescues and straws. They defined severely deficient areas as those where sample Se content was less than 0.02 ppm; deficient areas, 0.03 - 0.05 ppm; moderate areas, 0.06 - 0.09 ppm, and adequate areas where feeds contained over 0.10 ppm Se.
Figure 32. Distribution of Se in Chinese Feeds and Forages
Japan

A recently published map (Figure 33) reports on selenium status of soils at 150 sampling sites throughout the islands of Japan (Mizutani et al., 2001) and shows generally adequate levels of soil selenium. One gathers that there is not too much concern about possible selenium deficiency in Japanese diets because of the high consumption of fish, a good source of the element (Yokida, 1991). High selenium levels have been reported in dolphins, seals and whales, which feed on fish and represent the highest position in the marine food chain (Shibata et al., 1992).

Figure 33. Map of selenium levels in surface soils in Japan. The inset shows the southern Ryukyu Islands
Thailand

Selenium status in Thailand has been investigated by scientists from the Japanese National Institute of Animal Industry, as part of a program to promote self-sufficiency in animal production (Kamada et al., 2000). Since cattle are grazed in Thailand and fed little concentrates, selenium deficiency in soils and herbage directly impacts animal production. Selenium concentrations in herbage were higher in laterite areas than where soils were sandy or peat, however they were borderline to deficient in all three soils type areas. The plasma selenium in cattle averaged about 35 ppb, which is well below the level considered to be sufficient (70 ppb; Smith et al., 1988). The area of study is shown in Figure 34.

Figure 34. Narathiat province; the area of selenium study in Thailand.
South America

Seleniferous areas have been known to exist in Venezuela for some time and have led to accumulation of data on selenium levels in mothers and their children in the Venezuelan Andes areas (Bratter et al., 1991). From these data, estimates have been calculated for mean daily selenium intake in different areas: Caracas, 220 µg; Turen, La Laguna, 300 µg and El Aji, La Colonia, 450 µg. Single values occurred in the range of 100-1,200 µg. Selenium deficiency areas have been surveyed in Argentina for over 16 years. White muscle disease, a symptom of selenium deficiency, occurs in cattle and some relationship is thought to exist between dietary selenium and an enzootic syndrome called Enteque seco, which apparently also involves feeding on Solanum malacoxylon and causes soft tissue mineralization in grazing cattle (Ruksan et al., 1993). Ruksan and Zanelli (1992) have prepared a map showing areas of Se deficiency for livestock in Argentina, based on glutathione peroxidase (GPx) levels (Figure 35). They note that many animals tested had GPx values below 30 units/gram of hemoglobin, but also wrote that selenium toxic areas could also be found in the provinces of Cordoba and San Luis (Ruksan, personal communication, June, 1993). High-selenium shales in parts of the Andes mountains account for some selenium-high foods in Venezuela and for high levels of selenium in waters of the Orinoco river (Yee et al., 1987). Some signs of selenosis were found in a study of Venezuelan children (Bratter et al., 1991).

An interesting study identified significant inter-location variations in selenium status in Chile - a long, narrow country with marked climate differences from north to south. An assessment of the selenium range can be made from analysis of Se content of egg yolks from five different geographical areas in the country:

<table>
<thead>
<tr>
<th>Location Area</th>
<th>Number of Samples</th>
<th>Se (ppm)</th>
<th>Egg White</th>
<th>Egg Yolk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Far North</td>
<td>14</td>
<td>1.09</td>
<td>1.10</td>
<td></td>
</tr>
<tr>
<td>North</td>
<td>3</td>
<td>0.69</td>
<td>0.64</td>
<td></td>
</tr>
<tr>
<td>Central</td>
<td>5</td>
<td>0.59</td>
<td>0.55</td>
<td></td>
</tr>
<tr>
<td>South</td>
<td>12</td>
<td>0.55</td>
<td>0.58</td>
<td></td>
</tr>
<tr>
<td>Far South</td>
<td>7</td>
<td>0.81</td>
<td>0.90</td>
<td></td>
</tr>
<tr>
<td>All</td>
<td>41</td>
<td>0.79</td>
<td>0.81</td>
<td></td>
</tr>
</tbody>
</table>
Figure 35. Selenium deficiency in cattle, in Argentina, from Ruksan and Zanelli, 1992
Africa

The author has been unable to locate selenium status maps for the entire continent of Africa, although one would expect to find a wide range of selenium supply levels. Much excellent mineral experimentation has been done in South Africa, and an early paper (Tustin, 1959) attests to occurrence of selenium-responsive white muscle disease there. Interestingly, a recent paper (Van Nie Kirk et al., 1996) cautions against dosing sheep with selenium in reproduction during the time implantation is underway. Studies at the University of Pretoria have identified areas of Selenium deficiency in the Natal Midlands, based both on whole blood selenium levels and on glutathione peroxidase activity (Van Ryssen and Bradford, 1992). Van Ryssen (2001) of the University of Pretoria has summarized the available information on South African selenium status in map form (Figure 36). Marginal to acute Se deficiencies have been reported in the Midlands region and the mountainous area of KwaZulu-Natal province and the southern part of Western Cape province. Fairly large areas in the west-central part of the country appear to be selenium-sufficient. The situation is complicated by local choices of forage plants and many cases of definite Se-deficiency are associated with diets that are principally lucerne (alfalfa).

Figure 36. Geographical distribution of Se-status of herbivores in South Africa.
Considerable analytical data, including selenium values, have been assembled for various African countries. For example, a study of trace element levels has been carried out with several population groups in Burundi (Bensmariya et al., 1993). Investigators noted that intake of selenium by a rural population was very low - about on the scale of the Keshan disease area in China, and they attributed this in part, at least, to a very low consumption of fish by the study group. They have charted the contributions of various local food groups to total selenium intake (Figure 38). From studies of goiter and thyroid deficiency, it was learned that that condition is aggravated by a deficiency of selenium, and a belt of severe Se deficiency was identified in Central Africa (Vanderpas et al., 1990). Mpofu et al. (1999) have identified selenium deficiency symptoms in cattle in the smallholder grazing areas of Sanyati and Chinamhora, in Zimbabwe, and have provided a map, locating these areas (Figure 37). Plasma selenium in the dry season was 0.017, 0.025 and 0.017 µg/ml for calves, steers and cows.

Figure 37. Location of selenium-deficient grazing districts in Zimbabwe.
Taylor, at the University of Georgia (personal communication, Oct. 1996) has proposed a causal relationship between incidence of Human Immuno Deficiency virus (HIV) disease and selenium deficiency and has provided the following map of HIV occurrence in Africa which may therefore have value in predicting area selenium-status (Figure 39). In the area of toxicity, South African workers have examined plants and human urine from persons treated with traditional remedies and have concluded that selenium contamination of these plants does not constitute a problem in South Africa (Steenkamp et al., 2000).

Figure 39. Incidence of HIV-positive populations in Africa

Figure 38. Contributions (%) of different food groups to daily selenium intake by a rural population in Burundi (Benemariya et al., 1993)
Conclusion

With its recognition as an essential micronutrient, its facilitation of enzyme activity and its new-found role of protection against some of our most dreaded diseases, selenium has a lot to offer humanity. It is hoped that this little book, by assembling in one place much of the information on natural selenium levels in soils and plants in various parts of the world, will help achieve its considerable potential.

References

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