SELENIUM IN FOOD-PLANTS AND FEEDS. TOXICOLOGY AND NUTRITION*

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ABSTRACT

Lack of selenium in feeds is an economic problem in several countries because of the occurrence of white muscle disease in calfs and lambs and exudative diathesis in chicks. A relation between kwashiorkor in human infants and low selenium intake has been suggested recently.

The toxicity of high selenium intake for cattle and horses is well known; a teratogenic action has been described for various animal species and cancerogenic and anti-cancerogenic effects have been claimed. The effect of chronic selenium intake in humans has been little studied.

A survey in a high selenium area of Venezuela among lll school children revealed high selenium blood and urine levels (0.81 mcg/ml and 0.64 mcg/ml respectively) and slightly low values for hemoglobin and hematocrit. No correlation between high selenium blood values and low hemoglobin and hematocrit values was detectable. Protrombine activity is low and transaminases and alkaline phosphatase values are high in rats kept on a high selenium diet for 6 weeks. These parameters were normal in the children studied. Dermatitis, pathological nails, and loose hair were observed more often in children from the seleniferous than from the control area. The incidence of congenital malformations in humans was not higher in the seleniferous region than in the whole country. It is concluded that under the specific conditions of the survey no serious toxic effects of selenium were apparent.

Most essential elements may exhibit some toxic effects when ingested in excessive amounts. In the case of selenium, the doses required to prevent deficiency symptoms and those which might produce toxicity signs are very close. Exact levels are not easy to define because they depend on many factors, namely animal species, age, sex, nutritional and other physiological conditions, and the selenium compound used. A level of 0.1 ppm in the diet is considered satisfactory under most conditions to prevent deficiency symptoms for the chick (Thompson & Scott, 1969). Toxic effects may be observed at levels of 3 ppm, so that the safety range would be 1 : 30 or less.

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The essential role of selenium was discovered by Schwarz & Foltz (1958) when they fed a diet of torula yeast to rats. The animals developed liver necrosis if the diet was not supplemented with methionine, vitamin E and a third factor, called factor 3. This was later identified as an organic selenium compound. Inorganic selenium salts can replace factor 3, but higher doses are required.

The exact biochemical function of selenium is unknown. It is required for the production of formic dehydrogenase in *E. coli* (Pinsent, 1954). Very low blood levels of the enzyme glutathione peroxidase have been reported recently in selenium deficient rats and some chemical evidence of an association between this enzyme and selenium has also been brought forward (Ganther et al., 1972). Anemia, observed in selenium deficiency, could be explained by the lack of this enzyme which is necessary for the protection of red blood cells from H_2O_2.

Selenium deficiency diseases have been observed in cattle, sheep, horses, goats, swine, deer, mink, poultry, rats, and mice. The so-called white muscle disease, stiff lamb disease and nutritional myopathy in calves and lambs, have important economical implications, as has the fatal exudative diathesis in chicks and turkeys. They occur in reasonably well defined areas in the US, New Zealand, Finland, Great Britain, South Africa, Japan, Turkey, and Russia (Rosenfeld & Beath, 1964). Affected animals may respond to selenium administered by injection, added to the feed or to the soil of pasture land. Low selenium contents in the soil is most frequently associated with these diseases. In areas where gypsum fertilization is practiced the incidence is higher. The animals most frequently affected are sheep and chicks. Experimentally the disease symptoms can be produced by the feeding of legumes and various grasses grown in selenium-low soils.

Ewes fed alfalfa containing 2.5 ppm of selenium for 150 days and then a low selenium diet, transmitted protective amounts to lambs born 10 months later. Residual effects of selenium in the soil were evident 2 years after selenium application as determined from the selenium content of alfalfa (Allaway et al., 1966). It has been stated, however, that the use of applications of selenium to soil, to protect animals from white muscle disease, is considered inefficient in terms of the amount of selenium required. It involves some hazards of selenium toxicity. Shipment of feed grains containing selenium into areas of selenium deficiency is a common practice in the United States. The addition of inorganic selenium as sodium selenate to salt and to additives to feed, or its incorporation into pellets for sheep as well as the injection of selenium are other means to combat selenium responsive animal diseases (National Academy of Sciences, 1971).

There is no definite proof that selenium is an essential element in human nutrition, but some observations in children suffering from kwashiorkor, the

protein malnutrition disease, suggest such a role of this element. In infants that were refractory to the normal treatment, a daily supplement of 25 ug of selenium in the form of γ -diselenium divaleric acid produced an immediate response (Schwarz, 1961). Selenium blood levels of guatemalan children suffering from kwashiorkor were only half those of normal controls (Burk et al., 1967). In Jordania, a dose of 3–50 ug of sodium selenite/day produced a reticulocyte remission in kwashiorkor children (Majaj & Hopkins, 1966). Low selenium blood levels were observed in children suffering from kwashiorkor as compared to controls in a study done in Thailand. Administration of selenite enhanced weight gain considerably over children not receiving this supplement (Levine & Olson, 1970). These observations are centainly suggestive of an essential role of selenium in humans but much more work is required until final conclusions can be drawn. The levels of intake of selenium in humans vary considerably and are not easy to estimate.

Plants differ markedly in their capacity of selenium uptake and storage. For the so-called primary indicator plants it is an essential element. Species of *Astragalus* have been studied most extensively in this respect and levels of up to 11,000 ppm of selenium have been found. Plants capable of taking up considerable quantities of selenium from soil but which will also thrive on selenium – low soils are called secondary indicators (Rosenfeld & Beath, 1964).

A syndrome in cattle and horses was known for many years as alkali disease, but its relation to selenium intoxication was recognized only some 40 years ago. It is produced in livestock consuming plants in which selenium is bound to proteins and hence relatively insoluble in water. Consumption of seleniferous grains and forage grasses over a period of weeks or months causes the disease which is characterized by loss of hair and deformation of the hoofs in cattle, horses, and hogs. Feed containing 10–30 ppm selenium may cause alkali disease. The most severe lesions are found in the heart and liver (Rosenfeld & Beath, 1964).

Another type of chronic poisoning is known as 'blind staggers'. It appears in cattle and sheep when they consume seleniferous weeds which contain water soluble organic selenium compounds. In the first stage of the disease, the animals stumble over objects and lose the desire to eat and drink. Later the front legs weaken and a paralytic stage often precedes death.

Acute selenium poisoning in animals results from the consumption, often in a single feeding, of sufficient quantities of highly seleniferous weeds. Death may follow within hours (Rosenfeld & Beath, 1964). The most conspicuous lesions found in experimental selenium intoxication are focal liver necrosis.

A teratogenic action of chronic selenium poisoning has been observed in

cattle, sheep, swine, and rats (Rosenfeld & Beath, 1964).

Considerable attention has been paid to the possibility that selenium may have carcinogenic effect, since a paper on this subject was first published (Nelson et al., 1943). Some authors found more tumor-bearing rats receiving a diet supplemented with selenium than in the control group; other investigators could not confirm this observation (Harr et al., 1967). From the published data one may conclude that selenate is more likely to produce tumors than selenite (Schroeder & Mitchener, 1971). The question of the possible carcinogenic action of selenium has special importance in countries like the United States that are reluctant to allow selenium compounds to be added to feeds because of its alleged carcinogenicity. Selenium supplementation of feeds may be desirable in areas where white muscle disease or other selenium responsive diseases are common.

Table I *Production of tumors in selenium fed rats*

SOURCE	AMOUNT OF Se ppm	TIME of feeding	TUMOR RATE AS COMPARED TO CONTROLS	REFERENCE
Seleniferous Grain	5–10	18 months	higher	Nelson et al (1943)
Na_2 Se O_4	4,3	32 months	higher	Tscherkes et al. (1961)
Na_2 Se O_3	0.5–2.0	18 months	normal	Harr et al. (1967)
Na_2 Se O_4	2–8	22 months	normal	Harr et al. (1967)
Na_2 Se O_3	2 in drinking water	18–24 months	normal	Schroeder & Mitchener (1971)
Na_2 Se O_4	id.	18–24 months	higher	Schroeder & Mitchener (1971)

Some evidence obtained in the United States from human studies suggests a negative correlation between selenium and cancer incidence. According to published statistical data, cancer mortality is higher in states with low selenium levels in wheat and other crops than in those with higher levels (Schrauzer & Rhead, 1971). These interesting observations need further confirmation from other countries (Shapiro, 1972).

Adaptation of rats to selenium intake has been observed (Jaffé & Mondragón, 1969). Adapted animals seemingly take up less ingested selenium than non-adapted rats. The liver levels of the adapted animals decrease when on a diet which causes a rise of liver selenium in the

Table II *Cancer Mortality Rates (1967) and selenium concentration in grain and forage crops in the states in the U.S.*

SELENIUM CONTENT IN GRAIN AND FORAGE CROPS	CANCER MORTALITY RATES PER 100.000 DEATHS		
	160–190	125–160	90–125
	Number of States		
Low to very low (0.005 ppm)	17	4	0
Low to adequate (0.05–0.10 ppm)	0	5	5
High, locally excessive, median (0.26 ppm)	5	10	5

Schrauzer & Rhead (1971).

non-adapted animals. In the face of the problem of selenium toxicity in cattle and its possible hazards to man, similar investigations with other animal species would be of considerable interest. It is known that some species of wild animals thrive in areas in which cattle would exhibit deadly selenium poisoning.

Several chemical elements, notably zinc, chromium, vanadium, molibdenum and cobalt enhance the toxicity of selenite (Rosenfeld & Beath, 1964). Sulfate reduces uptake of selenium by plants and animals and increases urinary excretion. Arsenite stimulates excretion via the bile (Levander & Argrett, 1969). Supplementation of selenite containing diets with vitamin E and methionine reduces the toxicity. Certain fat soluble antioxidants have a protective activity similar to that of vitamin E when fed together with a methionine supplement. Methionine in association with certain antioxidants is probably necessary for methylation of selenium metabolites, which in turn are readily excreted, thus detoxifying selenium (Levander & Morris, 1970). The most active dietary ingredient used to reduce selenium toxicity is linseed oil meal (Levander, 1972). Sodium salts of EDTA stimulated the dialysis of selenium from liver homogenates of rats fed a high selenium casein diet but not from liver homogenates of rats fed the selenium casein diet supplemented with linseed oil meal. These results suggest that a portion of selenium in the liver of rats fed linseed oil meal is bound in a manner different from that in the liver of casein fed animals. Respiratory excretion of selenium as dimethyl-selenide can be observed after the ingestion of massive doses of selenite or selenate (Hirooka & Gafambos, 1966). Trimethylselenium ion is a normal excretory product of selenium in rat urine (Palmer et al., 1969). Recent observations point to a reduction of mercury toxicity by stimultaneously ingested selenium (Ganther et al., 1972).

Little is known about toxicity of selenium in humans. Acute

intoxications have been observed under various conditions. The exposure to selenium dioxide produces severe dermatitis, burning of the eyes; dimethyl selenide may produce acute sore throat and pneumonitis (Rosenfeld & Beath, 1964). Several cases of poisoning have been reported from Venezuela in people who ate seeds of 'coco de mono' *(Lecythis ollarea),* which contained up to 2% selenium on a fat free dry base. The selenium was shown to be present mostly as seleno-crystathionine (Kerdel-Vegas, 1964). The first symptoms of the intoxication were nausea, vomiting and diarrhea followed by loss of hair one to three weeks later.

The effect of chronic selenium ingestion in humans was studied by Smith and Westfall in 1937 in a group of persons in South Dakota, who excreted considerable amounts of selenium in the urine. The most frequently observed symptoms were gastrointestinal disturbances, bad teeth, and discoloration of the skin (Smith & Westfall, 1937).

We have observed that many food products from an area of Venezuela

HEMATOLOGICAL VALUES IN RATS FED SELENIUM DIETS

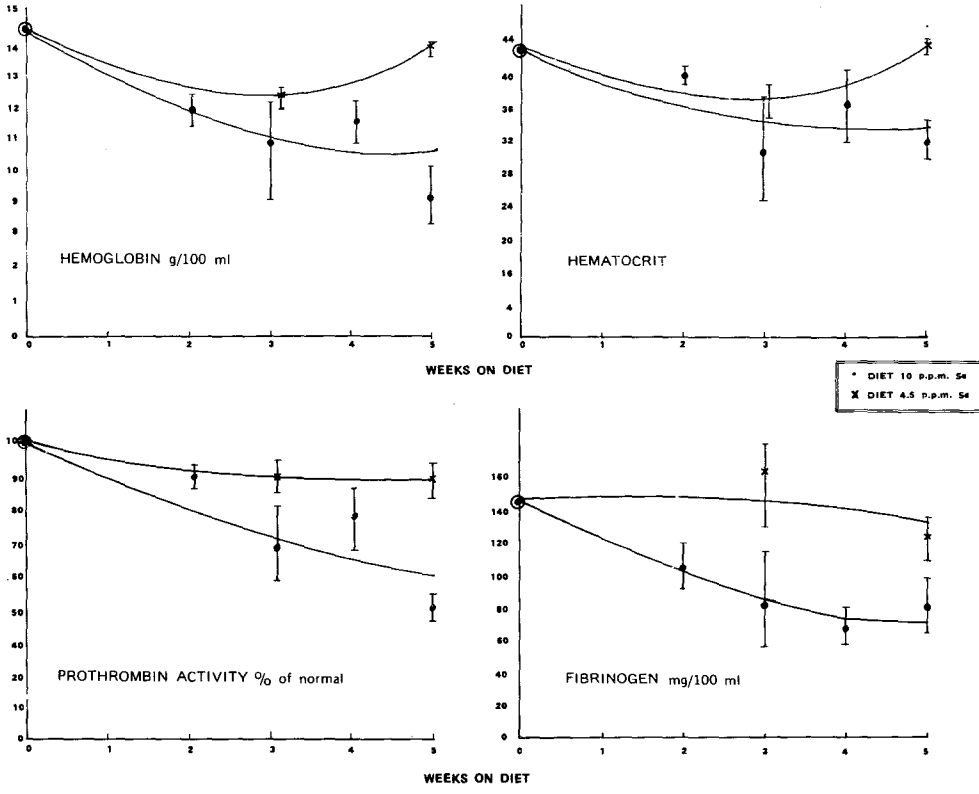

Table III *Blood levels of transaminases and alkaline phosphatase in rats fed seleniferous and non-seleniferous diets*

Selenium in diet ppm	Phosphatase U/ml	Transaminases	
		GOT U/ml	GPT U/ml
0.25	6.3	66	20
4.5	8.8	59	26
10.0	18.9	77	43

Jaffé et al. (1972).

have a high selenium content (Mondragón & Jaffé, 1971). In a study of urinary excretion of selenium in school children the highest levels were detected in the same area. The average level was over three times higher than that of children from Caracas.

To gain information which could be used in an investigation with children living in the seleniferous area, a study of the symptomatology of chronic selenium toxicity in rats fed seleniferous sesame press-cake was undertaken. The results indicated, among other findings, that hemoglobin, hematocrit, prothrombin activity and fibrinogen are lower in rats fed a selenium containing diet as compared to the controls. Blood values of glutamic-pyruvic transaminase (GPT) were much higher than in the controls, glutamic oxaloacetic transaminase (GOT) was nearly normal and alkaline phosphatase levels were high in the selenium fed animals. The animals were partially protected against selenium toxicity by high protein containing diets (Jaffé et al., 1972a).

With those results in mind, a field-study was undertaken with lll children from the seleniferous area with 50 children from Caracas serving as controls. Selenium content of whole blood was about 3 times higher in the first group than in the second (0.82 and 0.35 ppm). Urinary concentrations were 0.64 and 0.22 ppm respectively. A difference in hemoglobin and hematocrit levels between both groups was traced to different nutritional states rather than to selenium. Values for prothrombin activity, transaminases and alkaline phosphatase were normal and no correlation with blood selenium levels were apparent. Symptoms of dermatitis, loose hair, and pathological nails were more frequent among the children in the seleniferous area than in those living in Caracas. It was concluded that the intake of selenium in amounts resulting in urinary excretion of 0.6 ppm probably did not constitute a serious health hazard under the conditions of this survey, or at least, that the signs observed did not indicate the existence of such an hazard (Jaffé et al., 1972b). The level of 0.2 ppm of selenium in human

Table IV *Chemical and clinical data from children of a seleniferous and a control area of Venezuela*

Seleniferous area	No of cases	Hb g/100 ml	Ht	Se in blood ppm	Se in urin ppm	Protrombin activity %	Transaminasas GOT	GPT	Cutaneous symptoms	Cases with dental caries %
All cases	111	12.8	39	0.81	0.64	95	14	8	31	36
Cases 1.0 mcg/ml	28	13.3	40	1.32	0.66	96	15	8	39	
Control area										
All cases	50	14.8	42	0.35	0.25	95	17	9	14	73

Jaffé et al. (1972).

urine has been mentioned as significative in relation to possible intoxication (Rosenfeld & Beath, 1964). The amounts of selenium excreted by the group of children of our study was over three times higher and some children excreted up to 10 times more selenium i.e. 2 ppm.

Levels of selenium in corn and wheat of certain areas in Colombia have been reported to be much higher than in Venezuela. There several areas are known as 'peladero' which means 'hair loss'. Cattle, horses, and even humans show signs of intoxication characterized by hair loss and other ill defined symptoms, unless they are regularly allowed to live in other regions for several months (Benavides & Silva Mojica, 1965). Priests and travellers of the 15th to 18th century already recorded that the corn and other foods of some regions in Colombia were so poisonous, that they caused man and animals to loose their hair and Indian women to give birth to monstrous looking babies (Rosenfeld & Beath, 1964). No modern observations on a teratogenic action of dietary selenium in humans have been reported. The health statistics from Venezuela fail to indicate a higher incidence of congenital malformations in the seleniferous areas than in the rest of the country but, as already mentioned, the selenium problem in Venezuela is

Table V *Prevalence of caries among children 14–16 years old and levels of selenium in urine, eggs, milk, and water in eastern and western oregon*

VARIABLE	EASTERN OREGON KLAMATH Co.	WESTERN OREGON Jackson Co.	Josephine Co.
Mean D. M. F. Teeth/child	9.0	13.4	14.4
Mean urinary Se (ppm)	0.037	0.074	0.076
Mean Se in eggs (ppm)	0.056	0.502	0.437
Mean Se in milk (ppm)	0.005	0.049	0.067
Mean Se in water (ppm)	trace	0.001	0.002

Hadjimarkos (1969).

Table VI *Selenium levels in some foods*

	MIN. ppm		MAX. ppm	
Milk	0.005	–	1.27	USA Smith & Westfall (1937)
	0.005	–	0.134	GERMANY Kiermeyer (1969)
	0.050	–	0.324	VENEZUELA Jaffé et al. (1967)
Eggs	0.056	–	0.502	USA Hadjimarkos & Bonhorst (1961)
	0.25	–	9.14	USA Smith & Westfall (1937)
	0.49	–	8.51	VENEZUELA Jaffé et al. (1967)
Wheat	Trace	–	18.80	USA Smith & Westfall (1937)
			180	COLOMBIA Benavides & Silva Mojica (1965)

less serious than in Colombia (Jaffé & Vélez, 1972).

The effect of seleniferous diets on teeths has been studied in Oregon, USA. There appeared to be a direct correlation between urinary selenium excretion and the prevalence of dental caries (Hadjimarkos, 1969). No such correlation was detectable in Venezuelan children, but the fluoride content of the drinking water in the seleniferous area is higher than in Caracas, a fact that may have obscured any possible effect of the selenium intake on teeths (Jaffé et al., 1972b).

The level of selenium in foods can differ very much. This is not surprising as the levels in soil differ widely and, moreover some plants are more active accumulators of selenium than others. It is therefore interesting to compare selenium levels of any one food or plant produced in different areas or

Table VII *Selenium levels in defated sesame from different countries*

> 3 ppm	< 3 ppm
USA	Camarun
Venezuela	Brasil
Colombia	Ecuador
México	Guatemala
Japan	Iran
Kenya	Israel
Madascar	
Nigeria	
Puerto Rico	
Republique Centroafricaine	
Sudan	
Tschad	
Haute Volta	
India	

Jaffé et al. (1969).

countries. The study of 136 samples of defatted sesame seeds from 20 different countries, showed that samples from 14 countries contained more than 3 ppm of selenium (Jaffé et al., 1969). This is 10 times the maximum permissible level mentioned for human food (Código Latinoamericano, 1964). Sesame press-cake is used in animal feed and the selenium may pass into milk, eggs, and meat. The use of sesame in high protein supplementary foods is also of some importance (Parpia, 1966). The possible activity of selenium in kwashiorkor and the relation between protein intake and selenium toxicity have been mentioned. This fact should be carefully taken into consideration in the formulation of high protein supplements for use in undernourished children.

In regards to human nutrition and also to analytical procedures it should be remembered that many organic selenium compounds are quite unstable and are easily lost by heating. Ordinary cooking techniques however with few exceptions, did not result in major losses of selenium from foods. (Higgs et al., 1972).

In summary, it can be stated that notwithstanding the extensive literature on the effect of selenium on the health of animals and man its role in human nutrition, both as an essential element and as a health hazard, is still rather obscure. Hopefully more investigations will be carried out in this important field.

ZUSAMMENFASSUNG

Selenmangel in Futterpflanzen ist in vielen Ländern ein ökonomisches Problem, weil es das Auftreten der Weißen Muskelkrankheit bei Lämmern und Kälbern sowie der 'exudative diathesis' bei Kücken hervorruft. In letzter Zeit wurde bei Kindern auf einen Zusammenhang zwischen Kwaskiorkor und der Aufnahme von Selen hingewiesen.

Die Toxizität von hohen Selen-gaben auf das Milchvieh und auf Pferde ist bekannt. Eine teratogene Wirkung auf verschiedene Tierarten wurde beschrieben und karzinogene und anti-karzinogene Effekte festgestellt. Wenig untersucht ist der Einfluß einer chronischen Selen-Aufnahme auf den Menschen.

Eine Untersuchung von 111 Schulkindern in einer Gegend von Venezuela mit hohem Selen-Gehalt des Bodens ergab einen hohen Selenspiegel im Blut und im Urin (0,81 mcg/ml bzw. 0,64 mcg/ml) sowie verhältnismäßig niedrige Werte für Hämoglobin und Hämatocrit. Es konnte aber keine Korrelation zwischen hohem Selen-Gehalt im Blut und niedrigen Hämoglobin- und Hämatocrit-Werten gefunden werden. Bei Ratten, die 6 Wochen lang eine Diät mit hohen Selen-Dosen erhalten hatten, waren die Protrombinaktivität niedrig, die Transaminase und Alkalie-Phosphatase-Werte jedoch hoch. Diese Parameter waren bei den untersuchten Kindern normal. Dermatitis, pathologisch veränderte Fingernägel und Haarausfall fanden sich bei Kindern der Selenzone häufiger als in der Kontrollzone. In den Selengebieten traten angeborene Mißbildungen beim Menschen nicht stärker auf als im Landesdurchschnitt.

Abschließend kann gesagt werden, daß unter den spezifischen Versuchsbedingungen keine ernsten toxischen Einflüsse des Selens auftraten.

RÉSUMÉ

La carence en sélénium des fourrages est dans beaucoup de pays un problème économiquement grave, parce qu'elle détermine chez les agneaux la maladie du muscle blanc, et chez les poussins la 'diathèse exudative'. Dans ces derniers temps, un rapport a été signalé, chez l'enfant, entre kwashiorkor et ingestion de Se.

La toxicité de taux élevés de Se chez les vaches laitières est connue; une action tératogène a été décrite chez diverses espèces; des effets cancérogènes et anti-cancérogènes ont été signalés. Par contre, nous savons peu de données sur la consommation chronique de Se chez l'homme.

L'étude de 111 enfants d'âge scolaire d'une région du Venezuela à haute teneur en Se du sol a montre des taux élévés de Se dans le sang et l'urine (0,81 mcg/ml, 0,64 mcg/ml) ainsi que des valeurs relativement basses du taux d'hémoglobine et du chiffre d'hématocrite. Toutesfois, aucune corrélation n'a pu être trouvée entre un taux sanguin élevé de Se, et des taux bas d'hémoglobine et d'hématocrite.

Chez les rat nourris 6 semaines à un régime riche en Se, le taux d'activité de la prothrombine était faible, les valeurs de la transaminase, de la phosphatase alcaline étaient élevées. Ces paramètres étaient normaux chez les enfants étudiés. Les dermatites, les ongles déformés, la chute des cheveux, étaient plus fréquents chez les enfants en zône sélénifère qu'ailleurs. Les déformations congénitales n'étaient pas plus fréquentes chez l'homme que dans les autres régions du pays.

En résumé, dans les conditions étudiées, il n'apparait aucun symptôme sérieux de toxicité du Sélénium.

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DISCUSSION

Professor Becker (Kiel)

1. Es war lange Zeit schwierig, Selen in biologischem Material wirklich exakt analytisch zu bestimmen, besonders bei kleinen Konzentrationen. Ist das jetzt besser möglich?
2. Auf Böden mit hohem natürlichen Selengehalt (was auch für Molybdän gilt) ist die Produktion von Nahrungs- und Futterpflanzen, auch von Getreidekörnern und dergleichen mit einem eventuell schädlichen zu hohen Selengehalt unvermeidbar und nicht zu ändern. Welche Maßnahmen könnte man treffen, um die Gefahr von Mensch und Tier abzuwenden?
3. Die willkürliche Zufuhr von Selenverbindungen mit der Nahrung ist bisher noch nicht zulässig (außer in Neu-Seeland mit 0,15 mg/kg im Kraftfutter von Nutztieren), obwohl es zahlreiche Gebiete mit natürlichem Selen-Mangel im Boden gibt. Sind Bestrebungen zur Behebung von Selen-Mängeln in derartigen Ländern bekannt?

Professor Jaffé (Caracas, Venezuela)

Zu Ihrer ersten Frage:

Wir benützen die fluorometrische Methode von Watkinson (1966) (*Anal. Chem.* 38: *92*) mit 3,3-Diaminobenzidin. Dieselbe ist einfach, schnell und zuverlässig. Wir haben einige unserer Ergebnisse mit der Neutronenaktivierungsmethode von Duftschmid und Leibetseder (1967) (Atomic Energy Agency, Wien, 353) verglichen. Die höchsten Abweichungen waren ± 10%. Die Empfindlichkeit der Methode ist etwa 0.1 mcg Se.

In Bezug auf die zweite Frage kann ich erwähnen, daß in den Vereinigten Staaten einige Gebiete mit sehr hohem Selengehalt für die landwirtschaftliche Produktion ausgeschlossen wurden und z.T. sogar eingezäunt wurden, um das Weiden von Vieh zu verhindern. Meistens wird man versuchen, Chargen von Getreide und Futtermitteln mit hohem aber nicht extrem hohem Selengehalt mit selenarmen Chargen zu mischen, um den Gehalt auf eine zulässige Höhe zu bringen.

Ebenso wird im Allgemeinen in den Fällen verfahren, wo eine Erhöhung des Selengehaltes in Futtermitteln angestrebt wird, worauf sich Ihre letzte Frage bezieht. In der Tat ist die Zumischung von Selenit oder Selenat in manchen Ländern verboten, wegen der ungeklärten Frage ihrer cancerogenen Wirkung. Das Einmischen von Produkten mit natürlichem Selengehalt in Futtermittel mit Selenmangel ist die am häufigsten angewandte Lösung des von Ihnen aufgeworfenen Problems.

Professor Genevois (Paris)

Nordöstlich der Stadt Limoges, auf einem Granitboden, fiel vor 10 Jahren eine schwere Krankheit bei den Kälbern auf: viele Tiere starben an Myopathie.

Es hat sich herausgestellt, daß es ein Selenmangel war, ein paar Milligramm Selen pro Kopf genügen, um die Tiere gesund zu erhalten. Es gibt mehrere Veröffentlichungen über diesen Fall von einem dadurch berühmt gewordenen Tierarzt. Chemische Selen – Bestimmungen im Boden und in den Weidepflanzen wurden von Dr. Didier Bertrand durchgeführt.

Professor Jaffé (Caracas)

Diese interessanten Beobachtungen bestätigen die von mir erwähnten Berichte aus New Zealand und der USA. Beachtenswert ist die enge Umgrenzung einiger Selen-Mangel-Zonen.

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