

Selenium deficiency in deer: the effect of a declining selenium cycle?

Werner T. Flueck and JoAnne M. Smith-Flueck

Flueck, W.T. and Smith-Flueck, J.M. 1990. Selenium deficiency in deer: the effect of a declining selenium cycle? - Trans. 19th IUGB Congress, Trondheim 1989.

Selenium (Se) was discovered as an essential trace element in 1957. Se is an integral part of the enzyme system glutathione peroxidase (Glutathione:H₂O₂ oxidoreductase, EC 1.11.1.9) which functions in detoxification of H₂O₂ and organic hydroperoxides. Clinical deficiency in ruminants is expressed as white muscle disease, impaired reproduction, reduced rate of growth, decreased immune response and sudden death. The common underlying biochemical lesion results from oxidative damage to cell components. Since body distribution of glutathione peroxidase varies greatly between species, the pathophysiology also varies significantly. Cervids have been shown to be susceptible to Se deficiency and exhibit similar symptoms as domestic ruminants. A biological Se cycle was first described in 1964. Basic features of such a cycle include the presence of organisms which reduce and oxidize selenicals. However, only a few micro organisms have been described capable of oxidizing selenicals, and the overall balance tends to be towards reduction. Factors which may contribute to an apparent decline of Se availability include: i) soil acidification; ii) soil contamination with heavy metals; iii) fertilizer effect; iv) plant community composition; and v) rate of biomass removal and fire. Thus, land management practices and air pollution appear to be major factors which can alter the Se cycle dynamics, and may expose the vulnerability of free-ranging herbivores to Se depletion.

W.T. Flueck and J.M. Smith-Flueck, Wildlife and Fisheries Biology, University of California, Davis, California 95616, USA.

Introduction

Selenium (Se) is well known for its toxic properties. However, its significance to mammals resides in the role as an essential trace element in controlling toxic oxygen species (Flohé and others 1979). Antioxidant defense systems have thus been a conservative feature amongst mammals and include endogenous enzyme systems and dietary compounds. It is important to note that underlying biochemical lesions of most diseases are ultimately caused by toxic oxygen species (Levine and Kidd 1985).

Proper cycling of Se through the soil-plant-animal system is essential to protect animals from oxidant stress. This paper first describes functions of Se, then reviews factors which alter Se cycling, and concludes by discussing Se in cervids.

Selenium deficiency

Clinical symptoms and pathology.- Se deficiency in domestic ruminants is largely

associated with muscular degeneration, reproductive problems and illthrift. Most prominent is white muscle disease (WMD), which has occurred most widely in sheep and in cattle. Signs are variable and depend on the muscle masses affected. Young affected with WMD at birth usually die within a few days. More commonly, young animals are affected at 3-6 weeks of age and show a stiff and stilted gait and an arched back. Abnormally high levels of serum enzymes are present indicating damaged muscles.

Reproductive problems include impaired fertilization in cattle and sheep due to incompetent uterine musculature (Segerson and Ganapathy 1979), retained placentas (Buck and others 1981), embryonic death and WMD of postpartum young (Kott and others 1983), and impaired spermatogenesis and sperm function (Brown and Burk 1973).

Young animals are more susceptible to Se deficiency due to greater demands from growth, and may exhibit deficiency signs even if the mother is clinically normal (Suttle 1983).

Subclinical effects.— Much literature is available on pathology of overt Se deficiency. However, subclinical deficiency in animals is most difficult of all to recognize. This is especially important in free-ranging animals such as deer, since it is difficult to make adequate observations on individuals. Subclinical deficiencies can only be positively identified by production response trials where productive performance of Se treated animals is compared to untreated or control animals foraging under the same conditions (Millar 1983). Lack of response to Se supplementation reported in literature is most probably due to experiments with animals not depleted of this element or when therapeutic doses were insufficient (Whanger and others 1969, Hall 1987, Stowe and others 1988).

Subclinical effects include growth deficit (Millar 1983), decreased feed conversion efficiency (Norman and others 1989), decreased reproductive rate (Millar 1983, Flueck 1989), and increased susceptibility to infections (Arthur and others 1982, Boyne and Arthur 1986).

Biochemical rational.— Se as an essential trace element was discovered in 1957 by Schwarz and Foltz. Subsequently, Rotruck and others (1973) proved Se to be an integral part of glutathione peroxidase (GSH-Px; glutathione:H₂O₂ oxidoreductase, EC 1.11.1.9) which detoxifies H₂O₂ and organic hydroperoxides to their respective alcohols (Flohé and others 1979). Thus, broad specificity of GSH-Px provides the basis for interactions with multiple metabolic pathways (Flohé and others 1979). High GSH-Px activities are generally found in liver, kidney, erythrocytes, stomach, spleen, heart, lungs, and lens. However, since body distribution of GSH-Px varies greatly between species, the pathophysiology also varies significantly.

High reaction rates of GSH-Px with H₂O₂ suggest a predominant role in H₂O₂ metabolism at least in tissues or cell compartments in which competing enzymes such as catalase are missing, or present only with low activity, such as in the crystalline lens where GSH-Px is abundant, while catalase is absent. It also applies to cytoplasmatic and mitochondrial compartments of liver cells, because catalase is largely, if not exclusively, restricted to peroxisomes (Burk and others

1978). GSH-Px activity in erythrocytes, however, contributes substantially to maintain cell membrane integrity in spite of the presence of catalase. At present, the only known primary biochemical lesion associated with Se deficiency is low levels of GSH-Px (Keen and Graham 1989).

Microangiopathy seems to be a common underlying lesion in mulberry heart disease of swine, WMD in ruminants, Keshan disease of humans, and exudative diathesis of poultry (McAuslan and Reilly 1986). Platelets have relatively large amounts of GSH-Px and glutathione and Se deficiency alters platelet function and thus, affects blood clotting mechanisms (Hamberg and others 1974).

The Selenium cycle

Basic features of the cycle.— Se occurs in several different oxidation states: selenate, Se(VI); selenite, Se(IV); elemental Se, Se(0); and selenides, Se(-II), selenate being the most oxidized form in this order. However, only selenite and selenate are absorbed by plants (Gupta and Watkinson 1985). Selenides may occur in soils as stable complexes with metals (Allaway and others 1967, Comb and Comb 1986, Frost 1987).

A Se cycle was first described by Shrift in 1964. Since then, much evidence in support of a Se cycle has been advanced. Not widely discussed, though, is the apparent decrease of Se availability (Frost 1965, 1972, 1983). The rate of Se moving through certain parts of the cycle may vary greatly from one locality to another. The most important factor influencing these rates appears to be related to human activities.

A cycle implies that Se is reduced and oxidized by organisms (Fig. 1). Biological processes in microorganisms, plants and animals tend to reduce selenicals to selenides mainly as amino acids (Shamberger 1983:7). While biological oxidation of selenides to Se(0) has not been described, some microbial oxidation of Se(0) in soil occurs, but appears to play a minor role in mobilizing Se for plant uptake (Joblin and Pritchard 1981). Further, atmospheric oxidation of Se in soil is very slow (Geering and others 1968). Thus, while some organisms can oxidize Se(0), the

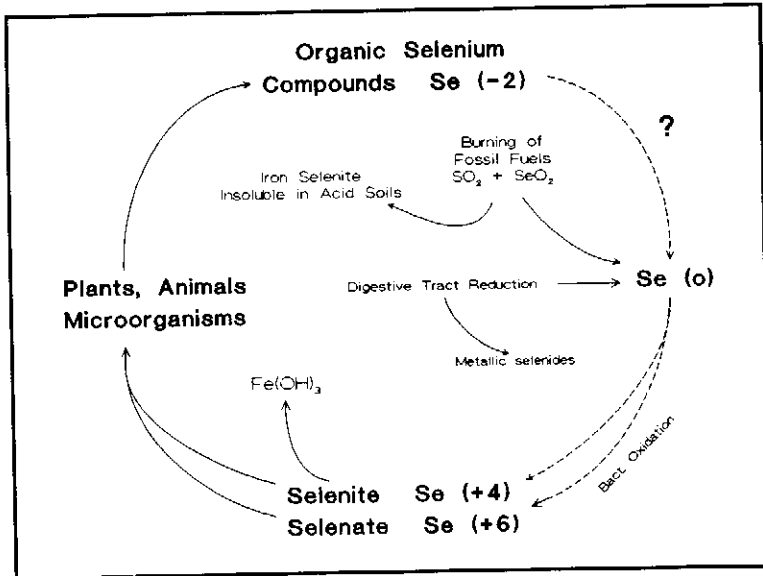


Fig. 1. The biological selenium cycle (see Shrift 1964, Frost 1972)

overall balance tends to be towards reduction (Watkinson 1981).

Anthropogenically induced shifts in the balance of the cycle.— Several authors have described an actual increased incidence of Se responsive diseases in animals (Jenkins and Hidiroglou 1972, Gissel-Nielson 1975, Stoszek and others 1980, Hastings 1979, Fischer 1982, Millar 1983, Griffiths 1986), which also appears to be the case in northern California (Flueck unpubl.). While increased exposure to numerous substances can result in increased demand for Se, there are several factors which may contribute to a decline of Se availability to plants and animals: i) soil acidification; ii) soil contamination with heavy metals; iii) plant fertilizer effects on Se uptake; iv) plant community composition; and v) the role of crop harvesting and fire. It remains to be established which aspects of Se chemistry contribute most significantly to a stable Se cycle, but local circumstances may ultimately determine which factors are most critical.

Soil acidification.— Anthropogenic production of protons is substantial. With respect to ability to neutralize, mitigate, or recover from effects of acid deposition, many areas are considered sensitive (Root and others 1980),

and intensive exposure over several decades can affect almost all soil types (Reuss 1978, Ulrich 1983, Eidg. Dept. des Innern 1984). Frost in 1972 suggested that emission of sulfur (S) dioxides and H^+ ions may lead to reduced Se availability to plants and animals.

A decrease in soil pH elicits a reduction of plant uptake of Se (Allaway and others 1967, Geering and others 1968, Gissel-Nielsen 1971a, Kubota and others 1975, Van Breemen and others 1984, Gupta and Watkinson 1985). Thus, acid precipitation has been considered to be in part responsible for a declining Se cycle (Frost 1972, 1983, Fisher 1982, Mushak 1985, Kieffer 1987). Soils with equivalent Se concentration can produce Se toxicity or Se deficiency in animals depending on soil pH. For instance, Se deficiency in North Dakota has been encountered recently due to immission from S oxides (Hastings 1979), even though North Dakota has been traditionally known to have high levels of Se and, at times, toxicity problems in livestock. Direct interaction with S may also have contributed to the situation.

Soil and plant contamination with heavy metals.— During soil acidification, increased amounts of heavy metals become available for plant uptake. Sweden, Germany, and California which have documented acid

precipitation, have cadmium (Cd) levels in wild ungulates above the German food safety limit (1.7 ppm dry wt) (Swedish Ministry of Agriculture 1982, Backhaus and Backhaus 1983, Flueck unpubl. 1984, Lindevall 1984). Heavy metals such as Cd and Hg, however, are antagonistic to Se demanding increased intake of Se for compensation. This also explains the protective action of Se as an antidote against heavy metal intoxication. Furthermore, increased solubilization of heavy metals may tie up Se in the soil by forming metal selenides (Allaway and others 1967, Comb and Comb 1986:2, Frost 1987).

Fertilizer effect.— All forms of agriculture are essentially exploitive due to removal of products which is reflected in widespread need for fertilizers. Correction of nutrient deficiencies in plants by adding fertilizer often increases the incidence of WMD (Stefferd 1956:431) and produces lower Se levels in animal tissues (Millar 1983, Gupta and Watkinson 1985). Phosphates reduce plant Se (Levesque 1974, Gissel-Nielsen 1977, Shamberger 1983) and GSH-Px activity in erythrocytes of animals foraging on these plants (Halpin and others 1982). Addition of nitrogen reduces Se levels in leafy tissue (Gissel-Nielsen 1971b, Singh 1975, Gissel-Nielsen 1979) probably through a dilution effect. S fertilization has four effects: 1) it corrects a S deficiency in plants producing a dilution effect on Se; 2) it reduces soil pH and thus, plant Se uptake (Underwood 1977); 3) sulphate inhibits the absorption by plants of selenate and possibly selenite (Gissel-Nielsen 1973, Prattley and McFarlane 1974, Shamberger 1983, Gupta and Watkinson 1985, Gissel-Nielsen 1987); and 4) S is highly antagonistic to Se in animals, reducing the availability of Se to animals (Martin and Gerlach 1972, Natl. Res. Council (NRC) 1983, Jones and others 1987), increasing Se excretion (Pope and others 1979), lowering tissue Se levels (White and Somers 1977, Pope and others 1979), and increasing the incidence of cardiac and biochemical lesions and WMD (Whanger and others 1969). S dioxide has also been shown to dilute plant Se (Frost 1972, Hoff and Davis 1983:132, Milchunas and others 1983). Further, plants appear to accumulate any excess S as sulphate in foliage where it becomes readily acces-

sible to herbivores (Turner and Lambert 1980).

High soil moisture and/or reduced soil aeration reduce plant Se, particularly during rapid "lush" plant growth due to favorable climatic conditions. Late spring and autumn when plant growth rate is rapid are seasons of greatest risk for incidence of Se responsive disease (Stefferd 1956:431, Anderson 1983, Towers and Clark 1983).

In ruminants, trimethyl selenide is the major urinary metabolite, and Se(0) and metal selenides are primarily excreted in feces (Butler and Peterson 1963, Peterson and Spedding 1963). Thus, animal urine and feces return Se to soil in relative insoluble, inert forms which are unavailable to plants. Plant uptake of Se from sheep feces over 75 days amounted to less than 0.3% and suggests that continuous foraging by ruminants could reduce the available Se in soil (Butler and Peterson 1963, Peterson and Spedding 1963).

Plant community composition.— Se concentration varies greatly in different parts of plants and also in different specimens of the same species (Burridge and others 1983). White clover (*Trifolium repens*), for instance, is a poor accumulator of Se (NRC 1983:20) and contains less Se than ryegrass (*Lolium perenne*) which, in turn contains less than browntop (*Agrostis tenuis*). Thus, changes in plant community structure such as results from improving a low-producing browntop pasture to a high-producing rye-white clover pasture can lead to a decline in animal Se status and lowered production, particularly in areas of marginal Se status (Burridge and others 1983, Clark and Towers 1983, Millar 1983). How selective feeding behavior may compound the situation remains to be established. It is known that deer avoid plants with high levels of Se (Allan and others 1984).

Other plant factors appear to interact with Se availability or requirement of animals. For instance, the incidence of WMD is greater when legumes are fed to sheep as compared to similar low Se grass or pasture, and Se requirement may then be higher (Whanger and others 1972). Also, concentration of polyunsaturated fatty acids determines in part requirements for Se.

The role of crop harvesting and fire.— Se cycles rather rapidly, especially when plant available Se is minimal with most Se tied up in the standing biomass (Swaine 1978), decaying organic matter and organic soil horizon (Gissel-Nielsen and Hamdy 1977). Biomass removal rate thus may influence the cycle significantly.

Fire acts as a potent mineralizing agent, causing the rapid transformation of organic compounds to inorganic ones. Removal of available Se by volatilization during fires may be substantial in systems with marginal to low Se concentrations (Swaine 1978). Additionally, much higher concentrations of S than Se exist in the plant material, and a likely result of combustion is S dioxide reducing selenicals to insoluble Se(0), and the formation of insoluble metallic selenides (Swaine 1978, Comb and Comb 1986, Frost 1987).

When seleniferous coal or oil is burned, Se is introduced into the atmosphere and redistributed to the earth's surface in rain and snow. Se from these sources is probably insoluble oxides or Se(0) and may not be of immediate value to plants and animals (Shamberger 1983:169) due to the reaction with S dioxide mentioned above.

Selenium in Deer

As early as 1955, Hadlow described degenerative myopathy in white-tailed deer (*Odocoileus virginianus*) similar to WMD of domestic animals, however, Se was not yet known to be essential. Following is a review of studies on Se in cervids.

Stuht and others (1971, cited in Brady and others 1978) reported mortality and bilateral skeletal muscular dystrophy in captive white-tailed fawns from does fed defined diets containing 0.15 ppm Se and 5 IU vitamin E/kg.

Captive reindeer (*Rangifer tarandus*) and other exotic ruminants showed clinical signs including WMD, failure to conceive, stillbirths, neonatal deaths, low birth weights and retarded growth, and transient and shifting lameness in both juveniles and adults due to Se deficiency (Natl. Zool. Park 1972, 1974; Griner 1978; NRC 1983:102).

Brady and others (1978) studied 32 adult white-tailed does and their fawns over two years which received either a basal diet containing 0.04 ppm Se and 5.5 IU vitamin E/kg, or the basal diet plus 0.2 ppm Se or 45 IU vitamin E/kg, or both. Dietary Se supplements had a significant effect on plasma Se concentration and erythrocyte GSH-Px activity in both does and fawns. WMD and mortalities were only seen in fawns, invariably following capture for blood collection. Only supplemental vitamin E significantly reduced overall mortality. However, both Se and vitamin E decreased blood malondialdehyde concentration, and Se alone decreased liver malondialdehyde concentrations. Erythrocyte GSH-Px activities in the Se- and Se+ adult females was 15.5 and 30.3 U/g Hb. GSH-Px activity below 30 U/g Hb, however, is considered deficient in ruminants (Suttle 1983). Furthermore, cases of WMD were observed in the group supplemented with both Se and vitamin E.

Van Reenen (1980) reported that the incidence of post capture myopathy appeared to be much higher in deer caught in Se deficient areas of New Zealand. Further, muscular dystrophy including lesions characteristic of WMD and Se responsive diseases such as illthrift occurred in adult deer and fawns.

Pine and Mansfield (1983) reported low whole blood Se values for black-tailed deer (*Odocoileus hemionus columbianus*) in California and suggested that low concentrations may be responsible for observed recruitment problems.

Alexander (1986:151) reported occurrences of WMD in fallow deer (*Dama dama*), red deer (*Cervus elaphus*) and Pere David's deer (*Elaphurus davidianus*).

Matzke (1986) reported the prevention of necrobacillosis in fallow fawns by Se and vitamin E treatment.

An outbreak of Se responsive unthriftiness in red deer on an intensively managed farm was shown to be unrelated to vitamin E status (Knox and others 1987).

Von Kerckerinck (1987:186) reported mortality rates in fallow deer which responded to Se treatment.

Se deficiency in mule deer based on whole blood Se analysis was shown to be

wide spread in California (Ros-McGauran 1989).

Flueck (1989) reported on Se responsive fawn survival of black-tailed deer in northern California. The area has generally low Se soils and associated diseases in livestock. A deer herd with apparent reproductive problems was selected for a clinical trial. Free-ranging adult females were supplemented with Se rumen pellets and marked with radio transmitters each year from 1984 to 1987. To evaluate the trial, pre-weaning survival rates of progeny from supplemented females were compared to rates in the untreated herd. The increase in survival rate attributable to Se supplementation was equivalent to an addition of 41 fawns per 100 adult females to the herd by autumn. Mean (\pm SD) blood Se distribution in untreated deer was 0.035 ± 0.031 ppm ($n=135$, mode= 0.024 ppm) as compared to a mean (\pm SD) of 0.121 ± 0.092 ppm ($n=42$) of supplemented deer.

Conclusion

Se is important for health and reproduction of mammals including cervids. Trends of increased incidence of Se responsive disease have been noted. This may be in part the results of decreased Se availability through changes in the Se cycle. However, due to its basic biochemical functions, Se demand may also be increased under increased exposure to toxic oxygen species.

From the perspective of biologists responsible for wildlife resources in remote areas, the most critical factors affecting the Se cycle are likely from extraneous origin. These include the importation of protons and heavy metals from air pollution. However, a very likely sequel to air pollution problems in remote areas is increased use of fertilizer to aid forests to overcome the pollution stress. Thus, fertilization may become an important local factor.

Plant species composition and specific factors are responsible for variability in Se bioavailability. A change in the plant community can thus result in decreased Se availability to animals independent of soil Se conditions.

Intensity of grazing and browsing concurrent with an accelerated rate of biomass

removal by harvesting crops or animals which forage on vegetation may also be an important aspect of the Se cycle.

All these factors can be altered by human activities. Therefore, changes in the Se cycle dynamics may expose the vulnerability of free-ranging herbivores to Se depletion.

Acknowledgements.— We greatly appreciate the support by the Swiss Society for Wildlife Research which in part, made this review possible.

References

- Alexander, T.L. 1986. Management and diseases of deer. — Vet. Deer Soc. Publ., London. 254 pp.
- Allan, G.G., Gustafson, D.I., Mikels, R.A., Miller, J.M. and Neogi, S. 1984. Reduction of deer browsing of douglas-fir (*Pseudotsuga menziesii*) seedlings by quadrivalent selenium. — Forest Ecol. Manage. 7: 163–181.
- Allaway, W.H., Cary, E.E. and Ehlig, C.F. 1967. The cycling of low levels of selenium in soils, plants and animals. — In: Selenium in Biomedicine. Muth, O.H., Oldfield, J.E. and Weswig, P.H., eds. AVI Publ. Comp., Westport, CT. 273–296 pp.
- Anderson, P.H. 1983. Selenium deficiency in farm livestock in Britain. In: Trace Elements in Animal Production and Veterinary Practice. — Occasional Publ. No. 7. Suttle, N.F., Gunn, R.G., Allen, W.M., Linklater, K.A. and Wiener, G., eds. Brit. Soc. Anim. Prod., 126–127 pp.
- Arthur, J.R., Boyne, R., Okolow-Zubkowska, M.J. and Hill, H.A.O. 1982. Neutrophils from selenium and copper deficient cattle. — In: Trace Element Metabolism in Man and Animals. Gawthorne, J.M., Howell, J.M., and White, C.L., eds. Springer Verlag, New York. 368–370 pp.
- Backhaus, B. and Backhaus, R. 1983. Die Cadmium-Belastung des Reichwides im Eggegebirge. — Z. Jagdw. 29(4): 213–218.
- Boyne, R. and Arthur, J.R. 1986. The Response of Selenium-Deficient Mice to *Candida albicans* Infection. — J. Nutr. 116: 816–822.
- Brady, P.S., Brady, L.J., Whetter, P.A., Ullrey, D.E. and Fay, L.D. 1978. The

- Effect of Dietary Selenium and Vitamin E on Biochemical Parameters and Survival of Young Among White-Tailed Deer (*Odocoileus virginianus*). - J. Nutr. 108: 1439-1448.
- Brown, D.G. and Burk, R.F. 1973. Selenium retention in tissues and sperm of rats fed a Torula yeast diet. - J. Nutr. 102: 102-108.
- Buck, E.L., Schmitz, J.A. and Swanson, L.V. 1981. Incorporation of Se-75 into Endocrine Glands and Reproductive Tissues of the Prepartum Ewe and Fetus. - In: Selenium in Biology and Medicine. Spallholz, J.E., ed. AVI Publ. Comp., New York. 514-519 pp.
- Burk, R.F., Nishiki, K., Lawrence, R.A. and Chance, B. 1978. Peroxide removal by selenium-dependent and selenium-independent glutathione peroxidases in hemoglobin-free perfused rat liver. - J. Biol. Chem. 253: 43-46.
- Burridge, J.C., Reith, J.W.S. and Berrow, M.L. 1983. Soil factors and treatments affecting trace elements in crops and herbage. In: Trace Elements in Animal Production and Veterinary Practice. - Occasional Publ. No. 7. Suttle, N.F., Gunn, R.G., Allen, W.M., Linklater, K.A. and Wiener, G., eds. Brit. Soc. Anim. Prod., 77-85 pp.
- Butler, G.W. and Peterson, P.J. 1963. Availability of selenium in forage to ruminants. - N.Z. Soc. Anim. Prod. 23: 13-27.
- Combs, G.F. and Combs, S.B. 1986. The role of selenium in nutrition. - Academic Press, New York. 532 pp.
- Eidg. Dept. des Innern. 1984. Waldsterben und Luftverschmutzung. - Eidg. Drucksachen- u. Materialzentrale, Bern, Switzerland. 120 pp.
- Fischer, K. 1982. Acid Precipitation and the Concerns for Fish and Wildlife Resources. - Intern. Assoc. Fish and Wildl. Agencies Proc. 72: 19-35.
- Flohé, L., Guenzler, W.A. and Loschen, G. 1979. The glutathione peroxidase reaction: a key to understand the selenium requirement of mammals. - In: Trace Metals in Health and Disease. Kharasch, N., ed. Raven Press, New York. 263-285 pp.
- Flueck, W.T. 1989. The effect of selenium on reproduction of black-tailed deer (*Odocoileus hemionus columbianus*) in Shasta County, California. - Dissertation, University of California, Davis. 284 pp.
- Frost, D.V. 1965. Selenium and Poultry. An exercise in nutrition toxicology which involves arsenic. - World's Poultry Sci. J. 21(1): 139-156.
- 1972. The two faces of selenium - can selenophobia be cured? - CRC Crit. Rev. Toxicol. 1(4): 467-514.
 - 1983. What do losses in selenium and arsenic bioavailability signify for health? - Sci. Total Environ. 28: 455-466.
 - 1987. Why the level of selenium in the food chain appears to be decreasing. Selenium in biology and medicine. Part A. - Combs, G.F., Spallholz, J.E., Levander, O.A. and Oldfield, J.E., eds. AVI Book Publ., New York. 534-547 pp.
- Geering, H.R., Cary, E.E., Jones, L.H.P. and Allaway, W.H. 1968. Solubility and redox criteria for the possible forms of selenium in soils. - Soil Sci. Soc. Amer. Proc. 32(1): 35-40.
- Gissel-Nielsen, G. 1971a. Influence of pH and texture of the soil on plant uptake of added selenium. - Agric. Fd Chem. 19(6): 1165-1167.
- 1971b. Selenium content of some fertilizers and their influence on uptake of selenium in plants. - Agric. Fd Chem. 19(3): 564-566.
 - 1973. Uptake and distribution of added selenite and selenate by barley and red clover as influenced by sulphur. - J. Sci. Fd Agric. 24: 649-655.
 - 1975. Selenium concentration in Danish forage crops. - Acta Agric. Scand. 25: 216-220.
 - 1977. Control of selenium in plants. - Risoe Report, 370: 42.
 - 1979. Uptake and translocation of selenium-75, in *Zea mays*. - Symp. Isotopes and Radiation in Research on soil-plant relationships. International Atomic Energy Agency, Vienna, Austria. 427-436 pp.
 - 1987. Selenium in the soil-plant system. - In: Selenium in Biology and Medicine. Combs, G.F., Spallholz, J.E., Levander, O.A. and Oldfield, J.E., eds. AVI Book, New York. 775-782 pp.
 - and Hamdy, A.A. 1977. Leaching of added selenium in soils low in native selenium. - Z. Pflanzenern. Bodenk. 140: 193-198.

- Griffiths, L.M. 1986. Mineral nutrition of farmed red deer. - *Deer Farming* (13): 12-17.
- Griner, L.A. 1978. Muscular Dystrophy in Ungulates at the San Diego Zoo and San Diego Wild Animal Park. - *Verhandlungsbericht des XX. Internationalen Symposiums über die Erkrankungen der Zootiere*. D. Kralovec, E., ed. Akademie-Verlag, Berlin. 109-115 pp.
- Gupta, U.C. and Watkinson, J.H. 1985. Agricultural Significance of Selenium. - *Outlook on Agriculture* 14(4): 183-189.
- Hadlow, W.J. 1955. Degenerative myopathy in a white-tailed deer, *Odocoileus virginianus*. - *Cornell Vet.* 45: 538-547.
- Hall, T.J. 1987. Effect of selenium on calf pneumonia. - *Vet Rec.* 121(25/26): 599.
- Halpin, C., Caple, I., Schroder, P. and McKenzie, R. 1982. Intensive grazing practices and selenium and vitamin B12 nutrition of sheep. - In: *Trace Element Metabolism in Man and Animals*. Gawthorne, J.M., Howell, J.M., and White, C.L., eds. Springer Verlag, New York. 222-225 pp.
- Hamberg, H., Svensson, J., Wakabayashi, T. and Samuelsson, B. 1974. Isolation and structure of two prostaglandin endoperoxides that cause platelet aggregation. - *Proc. Nat. Acad. Sci. U.S.A.* 71: 345-349.
- Hastings, D.H. 1979. Newborn Calf Losses Associated with Energy Conversion Facilities in North Dakota. - In: *Animals as Monitors of Environmental Pollutants*. Natl. Acad. Sci., Washington, DC. 387-388 pp.
- Hoff, G.L. and Davis, J.W. 1983. Noninfectious diseases of wildlife. - Iowa State University Press, Ames, Iowa. 174 pp.
- Jenkins, K.J. and Hidiroglou, M. 1972. A review of selenium/vitamin E responsive problems in livestock: a case for selenium as a feed additive in Canada. - *Can. J. Anim. Sci.* 52: 591-620.
- Joblin, K.N. and Pritchard, M.W. 1981. Selenium in a ryegrass pasture. - In: *Proc. New Zealand Workshop on Trace Elements in New Zealand*. Dunckley, J.V., ed. University of Otago, Dunedin, New Zealand. 93-97 pp.
- Jones, M.B., Rendig, V.V., Norman, B.B., Center, D.M., Dally, M.R. and Williams, W.A. 1987. Selenium enhances lamb gains on sulfur-fertilized pastures. - *Calif. Agri.* May-June: 14-16.
- Keen, C.L. and Graham, T.W. 1989. Trace elements. - In: *Clinical Biochemistry of Domestic Animals*. Kaneko, J.J., ed. Academic Press, New York. 753-795 pp.
- Kieffer, F. 1987. Selen, ein medizinisch bedeutungsvolles Spurenelement. - *Ars Medici.* 60-74 pp.
- Knox, D.P., Reid, H.W. and Peters, J.G. 1987. An outbreak of selenium responsive unthriftiness in farmed red deer (*Cervus elaphus*). - *Vet. Rec.* 120(4): 91-92.
- Kott, R.W., Ruttle, J.L. and Southward, G.M. 1983. Effects of vitamin E and selenium injections on reproduction and preweaning lamb survival in ewes consuming diets marginally deficient in selenium. - *J. Anim. Sci.* 57(3): 553-558.
- Kubota, J., Cary, E.E. and Gissel-Nielsen, G. 1975. Selenium in rain water of the United States and Denmark. - In: *Trace Substances in Environmental Health - IX*. Hemphill, D.D., ed. Univ. of Missouri Press, Columbia, Miss. 123-130 pp.
- Levesque, M. 1974. Some aspects of selenium relationships in eastern Canadian soils and plants. - *Can. J. Soil Sci.* 54: 205-214.
- Levine, S.A. and Kidd, P.M. 1985. Antioxidant adaptation: its role in free radical pathology. - *Biocurrents Division, Allergy Research Group, San Leandro, California.* 367 pp.
- Lindevall, B. 1984. The acid rain menace. - *Deer* 6(3): 65-66.
- Martin, J.L. and Gerlach, M.L. 1972. Selenium metabolism in animals. - *Ann. N.Y. Acad. Sci.* 192: 193-199.
- Matzke, P. 1986. Ueber einige gesundheitliche Probleme in Damwildgehegen zur Fleisch-erzeugung. - *Tieraerztl. Prax.* 14(4): 471-475.
- McAuslan, B.R. and Reilly, W. 1986. Selenium-Induced Cell Migration and Proliferation: Relevance to Angiogenesis and Microangiopathy. - *Microvas. Res.* 32: 112-120.
- Milchunas, D.G., Lauenroth, W.K. and Dodd, J.L. 1983. The interaction of atmospheric and soil sulfur on the sulfur and selenium concentration of range plants. - *Plant and Soil* 72: 117-125.
- Millar, K.R. 1983. Selenium. - In: *The Mineral Requirements of Grazing Rumi-*

- nants. Grace, N.D., ed. New Zealand Soc. Animal Production, New Zealand. 38-47 pp.
- Mushak, P. 1985. Potential Impact of Acid Precipitation on Arsenic and Selenium. - Environm. Health Perspect. 63: 105-113.
- National Research Council. 1983. Selenium in nutrition. - National Academy Press, Washington, D.C. 174 pp.
- National Zoological Park. 1972. 1971 annual report. - National Zoological Park, Smithsonian Institution. Washington, D.C. Smithsonian Institution Press.
- 1974. 18-months report, July 1, 1971 - December 31, 1972. National Zoological Park, Smithsonian Institution. Washington, D.C. Smithsonian Institution Press.
- Norman, B.B., Oliver, M. and Dunbar, J. 1989. Selenium supplementation in range animals. - Proc. 4th Int. Symp. Industr. Uses of Selenium and Tellurium, in press.
- Peterson, P.J. and Spedding, D.J. 1963. The excretion by sheep of 75-selenium incorporated into red clover (*Trifolium pratense*): the chemical nature of the excreted selenium and its uptake by three plant species. - N.Z. J. Agric. Res. 6: 13-23.
- Pine, D.S. and Mansfield, T.M. 1983. Physical condition and reproductive status of the Santa Lucia deer herd, Monterey County, California. - Calif. Dep. Fish and Game, Admin. Rep. 83-3. 15 pp.
- Pope, A.L., Moir, R.J., Somers, M., Underwood, E.J. and White, C.L. 1979. The effect of sulphur on 75-selenium absorption and retention in shecp. - J. Nutr. 109: 1448-1455.
- Prattley, J.E. and Mearlane, J.D. 1974. The effect of sulphate on the selenium content of pasture plants. - Aust. J. Exp. Anim. Husbandry 14: 533-538.
- Reuss, J.O. 1978. Simulation of nutrient loss from soils due to rainfall acidity. - Corvallis Environm. Research Lab. Office of Research and Development, US Environm. Protection Agency, Corvallis, Oregon. 44 pp.
- Root, J., McColl, J., and Niemann, B. 1980. Map of areas potentially sensitive to wet and dry acid deposition in the United States. - Proc. Int. Conf. Ecol. Impact Acid Precip., Norway, SNSF Project: 128-129.
- Ros-McGauran, G.V. 1989. Blood selenium in free-ranging deer in California. - Master Thesis, Dept. Epidemiology and Preventive Medicine, University of California, Davis. 57 pp.
- Rotruck, J.T., Pope, A.L., Ganther, H.E., Swanson, A.B., Hafeman, D. and Hoekstra, W.G. 1973. Selenium: biochemical role as a component of glutathione peroxidase. - Science 179: 588-590.
- Schwarz, K. and Foltz, C.M. 1957. Selenium as an integral part of factor 3 against dietary necrotic liver degeneration. - J. Am. Chem. Soc. 79: 3292-3293.
- Segerson, E.C. and Ganapathy, S.N. 1979. Fertility in ova in ewes receiving selenium and vitamin E supplementation. - J. Anim. Sci. 49(Suppl. 1): 335-336.
- Shamberger, R.J. 1983. Biochemistry of Selenium. - Plenum Press, New York. 334 pp.
- Shrift, A. 1964. A selenium cycle in nature? - Nature 201: 1304-1305.
- Singh, M. 1975. Effect of N-carriers, soil type and genetic variation on growth and accumulation of selenium, nitrogen, phosphorous and sulfur in sorghum and cowpea. - Forage Res. 1: 68-74.
- Stefferd, A. 1956. The yearbook of agriculture: animal diseases. - US Dept. Agric., US Government Printing Office, Washington, DC. 591 pp.
- Stoszek, M.J., Willmes, H., Jordan, N.L. and Kessler, W.B. 1980. Natural trace mineral deficiency in native pronghorn antelope populations. - In: Proc. 9th Biennial Pronghorn Antelope Workshop, Rio Rico, Arizona. J.S. Phelps, ed. 71-76 pp.
- Stowe, H.D., Thomas, J.W., Johnson, T., Marteniuk, J.V., Morrow, A. and Ullrey, D.E. 1988. Responses of dairy cattle to long-term and short-term supplementation with oral selenium and vitamin E. - J. Dairy Sci. 71: 1830-1839.
- Suttle, N.F. 1983. The nutritional basis for trace element deficiencies in ruminant livestock. - In: Trace Elements in Animal Production and Veterinary Practice. Occasional Publ. No. 7. Suttle, N.F., Gunn, R.G., Allen, W.M., Linklater, K.A., and Wiener, G., eds. British Soc. Animal Prod., England. 19-25 pp.

- Swaine, D.J. 1978. Selenium: from magma to man. - Trace Substances in Environmental Health - XII. Hemphill, D.D., ed. University of Missouri-Columbia, Columbia, Missouri. 129-134 pp.
- Swedish Ministry of Agriculture. 1982. Acidification today and tomorrow. - The Stockholm Conf. on the Acidification of the Environment. 232 pp.
- Towers, N.R. and Clark, R.G. 1983. Factors in diagnosing mineral deficiencies. - In: The Mineral Requirements of Grazing Ruminants. Grace, N.D., ed. New Zealand Soc. Animal Production, New Zealand. 13-21 pp.
- Turner, J. and Lambert, M.J. 1980. Sulfur nutrition of forests. -In: Atmospheric Sulfur Deposition. Shriner, D.S., Richmond, C.R., and Lindberg, S.E., eds. Ann Arbor Science, 321-333 pp.
- Ulrich, B. 1983. Effects of acid deposition. - In: Acid Deposition. Beilke, S. and Elshout, A.J., eds. D. Reidel Publ. Comp., 31-43 pp.
- Underwood, E.J. 1977. Trace Elements in Human and Animal Nutrition. Fourth edition. - Acad. Press, New York. 545 pp.
- Van Breemen, N., Driscoll, C.T. and Mulder, J. 1984. Acidic deposition and internal proton sources in acidification of soils and waters. - Nature 307: 599-604.
- Van Reenen, G. 1980. Trace Elements and Deer Management. - The Deer Farmer: 25-31.
- Von Kerckerinck zur Borg, J. 1987. Deer farming in North America. - Panther Press, Rhinebeck, New York. 225 pp.
- Whanger, P.D., Muth, O.H., Oldfield, J.E. and Weswig, P.H. 1969. Influence of sulfur on incidence of white muscle disease in lambs. - J. Nutr. 97: 553-562.
- Weswig, P.H., Oldfield, J.E., Cheeke, P.R. and Muth, O.H. 1972. Factors influencing selenium and white muscle disease: Forage types, salts, amino acids, and dimethyl sulfoxide. - Nutr. Rep. Int. 6: 21-37.
- White, C.L. and Somers, M. 1977. Sulphur-selenium studies in sheep I. The effect of varying dietary sulphate and selenomethionine on sulphur, nitrogen and selenium metabolism in sheep. - Aust. J. Biol. Sci. 30: 47-56.

3021

Eutrophication changes basic living conditions for European wildlife

Hermann Ellenberg

Ellenberg, H. 1990. Eutrophication changes basic living conditions for European wildlife. - Trans. 19th IUGB Congress, Trondheim 1989. (Only abstract).

Nutrient input - especially of nitrogen (N) - into ecosystems has raised since years. This is due to "active" agricultural - and to "passive" sources like immission and deposition from the air of gases and particles as NO_x and NH_x. In West Germany during the 80ies the "passive" input of N averages to more than 40 kg N/ha/years. This is equal to "active" fertilization during the 50ies. Today almost no single hectare with N-deficient conditions remains. This omnipresent eutrophication - apart from contributing to forest decline and bio-corrosion of buildings - changes most effectively the structure of the vegetation and the microclimate. Some large, social and/or mobile wildlife species are favoured by these conditions. Many populations of small, and/or sedentary species decline due to the same causes. N-deposition from the air renders most nature conservation strategies ineffective and urges up-to-date-research into the changed living conditions for European wildlife.

H. Ellenberg, Federal Research Institution for Forestry and Wood Products, Dept. Wildlife and Hunting, Leuschnerstr. 91, D-2050 Hamburg 80, FRG.