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SELENIUM SUPPLEMENTATION

A Survey of Selenium Treatment in Livestock Production

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Reports of the striking results of selenium supplementation in correcting disease and increasing livestock production in New Zealand prompted a survey of selenium work at each experiment station in the U. S. and at the principal agricultural centers abroad. The survey showed that such selenium deficiency diseases as white muscle disease of cattle and sheep are widespread, though often unrecognized. Selenium therapy prevents these diseases and many cases of scours and general ill thrift. Correction of subacute deficiencies gives striking increases in weight gain, fertility, and survival rates. Recommended methods of treatment include injection and oral drenching. As dosages are well below toxic levels, the safety factor is high. While a number of research problems still await solution, better diagnosis of known deficiency diseases and wider recognition of available methods of treatment can bring immediate practical benefits in livestock production.

SELENIUM, long considered toxic to livestock, has recently been found to be a trace element essential for animal life. In 1957 it was identified as a key factor in nutrition (*46, 49*). Already more than one hundred publications have appeared describing fundamental investigations or practical applications of selenium in animal nutrition.

Particularly striking reports on selenium treatment have appeared from New Zealand (*19, 22–25, 40, 50*), where large areas are deficient in selenium. Applications of minute doses of selenium compounds have cured such disorders as white muscle disease in cattle, sheep (stiff-lamb disease), horses, and swine, and exudative diathesis in poultry. Selenium treatment has also corrected such poorly defined conditions as ill thrift, infertility, and chronic scouring. Perhaps most important, even in areas where no clinical symptoms of deficiency were observed, selenium treatment has often given marked increases in weight gain, reproductive rate, and wool yield.

To determine the extent of selenium deficiency in soils and feeds and the potential significance of selenium treatment in world livestock production, a survey was conducted among the state agricultural experiment stations in the United States and in the principal agricultural research centers abroad. Replies were received from several federal agencies, all 50 states, Puerto Rico, the six Australian states, Canada, New Zealand, South Africa, Turkey, and the United Kingdom. Besides completing a questionnaire, many respondents sent summaries of unpublished work, and several New Zealand workers sent detailed reports of both experimental and practical field results. Sources of such unpublished data are listed in section A of the Literature Cited. The survey was conducted in the period September 1961 to January 1962.

Selenium as a Nutritional Factor

The research that eventually led to the discovery of selenium as a nutritional factor originated in studies of brewer's yeast as a protein supplement in Europe during World War II. German research workers found that rats on a yeast diet developed liver necrosis (*26*). Wheat germ and wheat bran showed protective activity against this disorder. On fractionation, vitamin E (α-tocopherol) was identified as the main protective agent (*45*).

Attempts by American scientists to duplicate the necrotic liver syndrome by feeding rats a diet of brewer's yeast were unsuccessful. However, with a diet based on torula yeast, Schwarz reproduced the results obtained in Europe (*44*). He gave the name Factor 3 to the unidentified material present in American brewer's yeast but not in torula yeast or European brewer's yeast. L-Cystine was erroneously considered to be Factor 2 which, like vitamin E, appeared to provide protection against necrotic liver damage.

A second connection between vitamin E and Factor 3 was established when Scott and coworkers discovered that brewer's yeast would prevent exudative diathesis in chickens. This condition was recognized as a vitamin E deficiency which could be induced with a torula yeast diet (*47*).

Selenium was finally identified as the key component of Factor 3 in 1957

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(*46, 49*). This discovery provided a new avenue of research for workers investigating disorders responsive to vitamin E. It also made possible the identification of seleno-cystine as the protective factor in L-cystine. These developments have recently been reviewed in detail by Schwarz (*43*).

The precise metabolic role of selenium and vitamin E is still unknown. McLean, who was one of the first to observe growth responses to selenium, has aptly summarized the current status as follows: "There can be little doubt that traces of selenium are required by the animal for normal metabolism, that vitamin E and selenium are interrelated in their metabolic functions, and that vitamin E cannot completely replace the need for selenium" (*34*).

Whatever its function, selenium has been found to give positive responses to a variety of disorders in different species. Table I, adapted from Schwarz (*43B*) with added information on goat from Tustin (*51*), gives symptoms responsive to selenium treatment in 13 animal species. As a result of the effectiveness of selenium in these muscular and metabolic disorders, experimental work at the National Institutes of Health is now being extended to include its effect on related human diseases (*43*).

Table I. Animal Deficiency Symptoms Responsive to Selenium[a]

Source: Adapted from Schwarz (*43B*); Information on goat (*51*)

Animal	Muscular Dystrophy[b]	Growth Deficit	Necrosis: Liver	Necrosis: Kidney	Necrosis: Heart	Exudative Diathesis	Other
Cattle	3	...	...	...	3	...	Calcifications 3
Chicken	2	3	...	...	...	3	Serum protein 3
Goat	2	...	...	...	...	...	
Hamster	3	3	...	...	...	2	
Hog	3	...	3	...	...	2	
Horse	3	...	...	...	...	...	
Mink	3	...	...	...	3	...	
Mouse	3	2	3	3	3	0	Serum protein 3, pancreas atrophy 3, lung hemorrhage 1
Rabbit	...	...	3	...	...	...	Lung hemorrhage 3
Rat	2	2	3	3	1	0	Calcifications 3, paradentosis 3, lung hemorrhage 2, pancreas atrophy 2, serum protein 3
Sheep	3	2	...	...	3	...	Calcifications 3, paradentosis 3
Trout	?	?	3	...	...	...	
Turkey	2	3	...	...	...	3	Serum protein 3

[a] Code: 3 = Pronounced pathological changes; 2 = not always detectable; 1 = occasionally found; 0 = no pathological changes; ? = possible, but not clearly defined; ... = not investigated or not involved.

[b] Also known as white muscle disease in cattle and sheep.

Incidence of White Muscle Disease

The most widely recognized and clearly defined selenium-deficiency disease is white muscle disease (WMD), also called muscular dystrophy and stiff-lamb disease. WMD attacks lambs and calves primarily, but also occurs in horses (*24*), goats (*51*), and poultry (*24*), among other species. Symptoms of the disease are described more fully in the section below on diagnosis, is named from the white color of the muscles in affected animals.

In this survey, WMD was reported by all the major sheep-producing countries of the world responding:—Australia, Canada, New Zealand, South Africa, Turkey, the United Kingdom, and the United States. Together these countries represent about one third of the total world sheep population. WMD is also reported to occur in Scandinavia, Germany, France, Switzerland, Italy, South America, and Japan (*21*).

The highest incidence, possibly amounting to 20 to 30%, was reported by Turkey (*1*). New Zealand probably has the second highest rate; one estimate (*9*) places the number of New Zealand sheep which could benefit from selenium supplementation at 10 to 15 million, or 20 to 30% of the total sheep population. However, this figure includes all selenium responsive diseases, not WMD alone. Five of the six Australian states (New South Wales, South Australia, Tasmania, Victoria, and Western Australia) indicate some WMD, but no area has an extreme condition. The situation is similar in South Africa and the United Kingdom.

Of the 50 United States and Puerto Rico, 32 have experienced WMD in lambs and calves. Twelve states reported little or no WMD, but one of these experienced general nonspecific ill thrift, which may be subclinical WMD or a related selenium-responsive condition. The remaining seven states had no information on this subject. Figure 1 shows the geographical distribution of WMD in the U. S.

Note that WMD occurs in every section of the continental U. S., even in the western states where excess selenium is sometimes a problem. While direct losses are thought to be low in many cases, subclinical symptoms in the form of ill thrift of cattle and sheep, low lambing rates, and low wool yields are probably more common. Most states have not been aware of the problem for a sufficient length of time to evaluate the full extent of losses.

The wide extent of WMD in cattle has also been shown in a survey conducted in 1955 (*53*). WMD was diagnosed in 1.2% of all animals with nutritional disorders. More recent research has shown that animals often die of pneumonia due to immobility brought on by WMD, and that scours are sometimes the result of subclinical WMD. It is therefore significant that in the 1955 survey pneumonia was listed as the major cause of nonnutritional ailments (20.2%), and calf scours as a close second (17.7%). In the state of Washington, where WMD accounted for 10% of all nutritional cattle diseases, calf scours had the highest incidence (28.4%) of any nonnutritional disease.

Selenium Treatment of Livestock

WMD has long been associated with vitamin E deficiency (*35, 55*). Following reports on Factor 3-active selenium, the agricultural experiment stations at Cornell (*39*) and Oregon (*36*) began to evaluate various selenium compounds in both sheep and cattle. Similar programs were started in 11 other states, in Australia, Canada, South Africa, and the United Kingdom. Particularly significant experiments were initiated in New Zealand (*22*), where WMD had become a serious threat to sheep production, especially in the South Island.

These experimental programs have progressed to different stages in the different countries. In New Zealand, where work is most advanced, and to a lesser degree in the U. S., a number of other field disorders besides WMD have been found responsive to selenium. These include generalized ill thrift and infertility of sheep, scours of cattle, hepatosis dietetica of swine, and exudative diathesis of poultry.

The present stage of research and field treatment in the countries most active in research on selenium treatment of livestock is summarized below by country.

United States. A considerable amount of experimental work on WMD has been carried out in the U. S. In Oregon, supplementing the rations of ewes through weaning with 0.1 p.p.m. selenium, as sodium selenite, consistently provided protection against WMD while vitamin E did not. In limited trials, selenium gave greater growth response in lambs than vitamin E (*41*). However, during a 5-year study on prevention of WMD, Cornell workers found

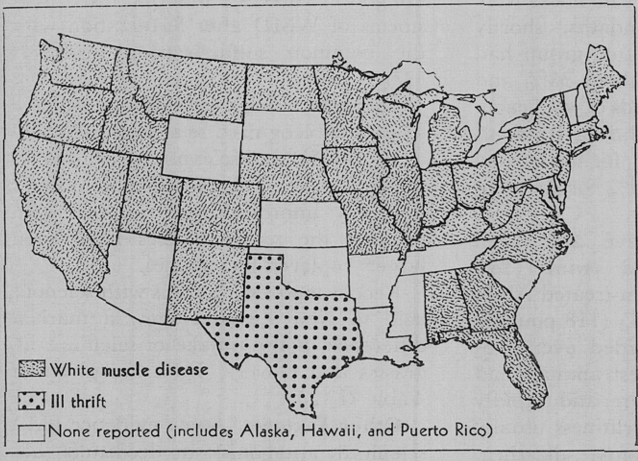

Figure 1. Incidence of white muscle disease (WMD) in the United States

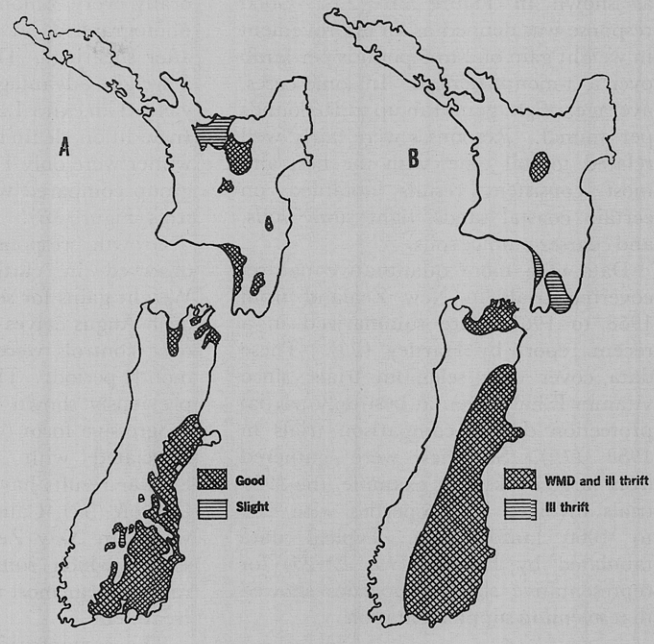

Figure 2. Coincidence of white muscle disease (WMD) and liveweight response to selenium in New Zealand ▶

(*A*) Lamb liveweight responses to selenium (*40*). (*B*) Disease incidence in lambs (23). (Adapted from maps published by the *New Zealand Journal of Agriculture* and reprinted here by permission)

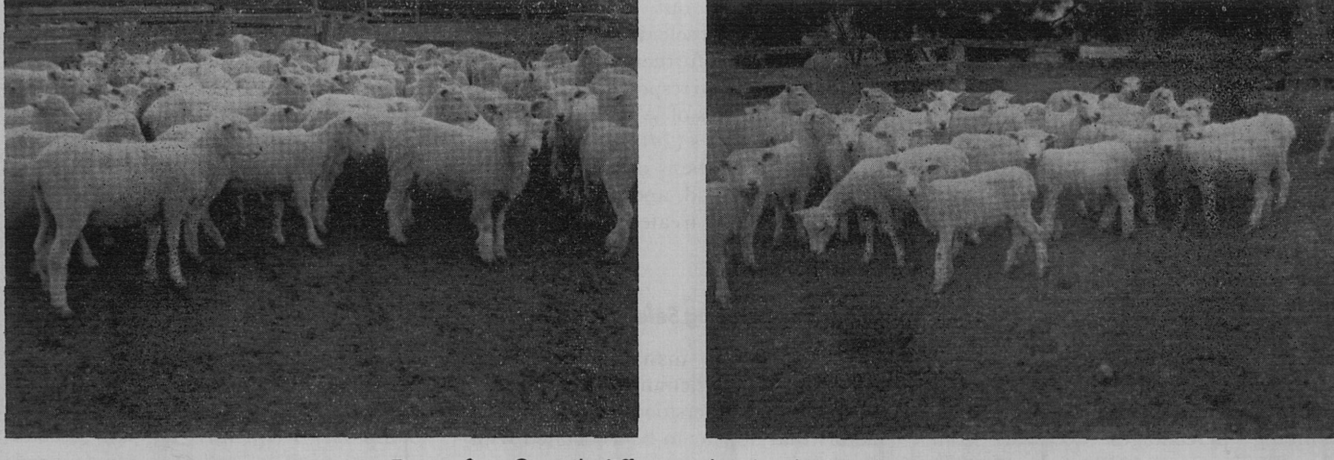

Figure 3. Growth differential with selenium therapy

(*A*) Treated Romney ewes (*B*) Control group

no differences between dietary supplementation with vitamin E or selenium (*6, 28*).

In California, outbreaks of WMD in calves and lambs were successfully halted with vitamin E plus selenium after vitamin E alone had not prevented successive occurrences (*42*). Outbreaks of WMD have also been successfully treated with selenium in Ohio (32) and Montana (*56*). Ohio workers found vitamin E equally effective, but concluded that selenium treatment was more economical (*33*).

Selenium is fairly widely employed as an injectable in WMD areas of Nevada and is believed to have greatly reduced losses from this disease (*8*).

The practical use of selenium in WMD is as yet largely confined to the western states. An injectable preparation of selenium and vitamin E, clinically tested by over 312 veterinarians on over 500,000 animals, is the most widely used treatment (*16, 42*). This preparation has also proved effective in treatment of various cases of scours and generalized ill thrift in both cattle and sheep (*16, 30*). Other responsive diseases are Knuckler's disease in cattle on feed lots and joint-ill of swine (*16*).

United Kingdom. WMD occurs in England and Scotland in both sheep and beef cattle (*3*). In Scotland, studies extending over several years (*14*) indicate that selenium, either by injection or oral administration, has given good control of WMD—in some cases, better than that obtained by injection of α-tocopherol acetate (*3*). Further work is in progress on the effects of selenium on lamb growth under Scottish conditions.

Australia. WMD occurs sporadically in many parts of Australia, and several preliminary reports on selenium treatment have appeared (*20, 52*). Exudative diathesis also occurs as a field disease in Tasmania. Considerable research on selenium treatment is in progress, but as yet there is little or no practical use (*4*).

New Zealand. New Zealand is by far the most advanced country in field investigation and practical treatment of selenium deficiency. In this country, selenium treatment is now widely practiced (under veterinary control) both for control of WMD and for treatment of much ill thrift and infertility in sheep (*2, 9, 10, 19, 22–25, 40, 50*).

While most trials elsewhere involved a maximum of several hundred lambs, the Department of Agriculture conducted 700 lamb-dosing trials involving 40,000 animals over a 3-year period (*40*). These trials showed liveweight responses to selenium in extensive areas of the South Island and considerable areas of the North Island, as outlined in Figure 2A. The areas of good response correlate well with the areas where WMD occurs, as shown in Figure 2B (*23*). Good response was defined as an improvement in weight gain of 2 to 5 pounds per lamb over a 3-month period. In some cases, average weight gains ran up to 10 pounds per animal. Responses were fairly well related to soil type, with the best and most consistent results obtained on certain coastal sands, light stony soils, and coarser pumice soils.

Data of a more quantitative nature, covering trials in New Zealand from 1958 to 1961, were summarized in a recent report by Hartley (*23*). These data cover only selenium trials, since vitamin E had given at best only partial protection during comparison trials in 1958 (*19*). Statistics were gathered from large flocks—for example, the 1959 trials involved 11 properties with 200 to 1000 lambs each. Typical data tabulated by Hartley (*19, 22–25*) for representative sheep properties showed that selenium supplementation

- -eliminated WMD where the incidence in control flocks was 12 to 37%;
- -increased lambing percentages from 74% in untreated flocks to 116%;
- -decreased the incidence of barren ewes from 30 to 2%;
- -caused increases in liveweight of 250% over those in untreated flocks;
- -increased wool yield per lamb from 4.4 pounds (control) to 6.8 pounds over a 9-month period;
- -decreased lamb mortality rates from 30% in untreated flocks to nil.

Unpublished case histories from New Zealand properties show equally striking results (*2*, *9*). At one ranch grazing 1000 ewes in a zone of 22-inch annual rainfall, WMD had been observed for 10 years. However, infertility had recently become a more serious problem. Lambing percentages declined from 63 to 130% prior to 1948 to a low of 29% in the period 1948 to 1957. In the last four seasons, the use of selenium eliminated both WMD and the infertility problem. In the 1958–9 season, several groups of lambs were treated at docking with either selenium or vitamin E. Of 262 lambs in each group, 107 of those treated with selenium were heavy enough for slaughter by late spring while only 53 of the vitamin E group could be taken.

At the Winchmore Research Station in Ashburton, New Zealand, which has no history of any selenium-responsive diseases, selenium treatment resulted in increased lamb weight, ewe weight, and wool yield. Furthermore, the levels of response were comparable to those in other areas of New Zealand where selenium responsive disorders had been observed.

Illustrative of the weight gains resulting from selenium treatment are the flocks shown in Figure 3. The lambs were separated soon after docking, and one group (Figure 3*A*) received selenium orally every 3 months. The flocks were photographed at 13 months, shortly after shearing. The treated group had a weight advantage of about 35% and yielded an extra 1.2 pounds of wool each. In addition, death losses during a difficult winter were only 1 to 2% for the treated group compared with 10% for the controls (Figure 3*B*).

Growth responses have also been observed in cattle and swine (*23*). Weight gains for selenium-treated Aberdeen-Angus calves of 50% (118 pounds) over controls were recorded over a 5-month period. These test animals had previously shown a severe and rapidly progressive form of unthriftiness usually associated with a profuse diarrhea. Similar results have been reported from Oregon (*38*), California (*30*), and elsewhere in New Zealand (*9*, *50*). The scours which sometimes follow heavy rains appear most responsive to selenium treatment.

The response in swine was obtained with piglets 6 to 14 weeks old having symptoms diagnosed as hepatosis dietetica. No controlled field trials have yet been carried out. In most cases, however, 5 mg. of selenium administered orally eliminated further outbreaks.

Horses have also responded to selenium treatment in cases of WMD in foals and tied-up race horses (*24*).

Exudative diathesis occurs in poultry in the South Island, and outbreaks have been successfully treated with selenium (*24*).

Conditions Inducing Selenium Deficiency

The soil and management factors associated with selenium deficiency are not yet well understood. Some of the factors that appear to be associated with the deficiency are:

Leaching. Many light, highly leached soils are deficient in selenium; such soils are also often deficient in other trace elements. WMD and other selenium-responsive disorders often appear in stock grazing on irrigated land or after periods of high rainfall (35).

Flush Growth. Selenium-responsive disorders are common in the spring and (as just noted) after heavy rainfall or on irrigated, fertile soils. Often WMD and similar deficiency diseases occur after fertilization of any kind. On soils of marginal selenium content, the flush growth induced by these conditions may result in forage of unusually low selenium content.

High Sulfate. High concentrations of sulfate appear to depress the uptake of selenium by the crop (*48*). For example, the application of gypsum to pasture depressed the growth rate of selenium-responsive lambs below that of similar lambs on untreated pasture (*24*). Furthermore, use of elemental sulfur alone as a fertilizer causes WMD. Oregon workers describe frequent storms of WMD after fertilization with the common sulfur-bearing fertilizers (*37*).

Legumes. High-legume rations have long been recognized as a cause of WMD (*35*). WMD and selenium-responsive ill thrift have been diagnosed in a number of newly improved pastures. In such pasture, the native grasses have been largely replaced by legumes.

Recent topdressing trials with selenous acid in New Zealand show a marked difference in the uptake of selenium by grasses and clover. Data are given in Table II (*25*).

Other Factors. Some evidence for a selenium antagonist in feedstuffs has been presented by Hogue (*27*) and Canton and Swingle (*17*), and the latter identified an ethanol-soluble succinoxidase inhibitor. This find suggests a rather complex system involving selenium, selenium depressants such as sulfur, and selenium inhibitors.

Diagnosis of Deficiency Diseases

WMD may be improperly diagnosed even where clinical cases occur. Experienced workers find that WMD is frequently misdiagnosed in the field as pneumonia (*7*, *12*). To help differentiate WMD in lambs from pneumonia, Welch (*12*) listed the significant symptoms shown in Table III. The 8-inch board test appears particularly practical for screening lambs. The flock is merely driven through a doorway or gate obstructed by an 8-inch board held vertically on the ground. The hind legs of lambs with WMD will not clear the board.

Lambs affected with WMD show a stiff, stilted gait with an arched back. Symptoms frequently do not become obvious until animals are driven hard. Animals with severe heart involvement may die suddenly without prior signs of stiffness. In the congenital form, lambs may be born dead or may die within a few hours to 3 days of birth. Paradentosis is frequently associated with WMD.

Post-mortem findings reveal localized or widespread chalky-white discolorations of the involved muscles. Subclinical cases may be detected by analyzing for a high level of glutamic-oxalacetic transaminase in the blood serum (*15*, *31*, *33*).

The symptoms of WMD in calves are similar (*29*). Sudden heart failure appears to be the most common cause of death. The first warning sign is difficult breathing—predominantly abdominal with lack of chest movement and expiratory grunts. All forms of paralysis occur. Standing with forelegs bent at the knees is typical. The tongue may become paralyzed, and swallowing difficulties may be evident. Necropsy findings are similar to those described for lambs.

Table II. Differential Uptake of Selenium by Grasses and Clover (25)

Topdressing Rate, Ounces Se/Acre	Selenium Content, P.P.M.			
	Browntop	Ryegrass	Cocksfoot	White Clover
Control	0.022	0.012	0.012	0.008
2	0.28	0.23	0.16	0.08
4	0.45	0.25	0.18	0.08
8	0.84	0.46	0.36	0.21
16	1.54	0.86	0.68	0.34

Table III. Symptoms of White Muscle Disease and Pneumonia in Lambs (12)

	White Muscle Disease	Pneumonia
Eyes	Bright	Dull
Ears	Pointed	Droopy
Creatinuria	Present	Absent
8-Inch board test	Fail	Pass

Recognition of subclinical selenium deficiency symptoms may be even more important. Where general ill thrift or scours are encountered in a WMD area, selenium deficiency may be considered as a possible cause for investigation. As an example, the cattle Kendall successfully treated with selenium had had scours for months in spite of treatment with antibiotics and anthelmintics (*30*).

Recommended Methods of Treatment

At present, selenium treatment should be administered only under the direction of a registered veterinarian or other qualified livestock specialist. Determination of the exact dosage should be made by the administering veterinarian for each herd treated.

Where permitted, the most convenient treatment at present is probably the oral drench application recommended by Hartley (*23*). In the United States, an injectable preparation is available containing selenium in combination with vitamin E (*16*).

In both the oral drench and injection methods, sodium selenite is the selenium compound administered. Recent research suggests that barium selenate may have advantages over sodium selenite. Barium selenate is nontoxic even when injected at the rate of 1080 mg. per 100 pounds of body weight; by contrast, sodium selenite is toxic at 40 mg. per 100 pounds of body weight and lethal at 80 mg. The barium salt, injected in the shoulder at levels of 20 and 50 mg. per 100 pounds of body weight, is absorbed and eliminated more slowly and thus gives longer protection to the animal (*31*).

Research continues on other potential methods of treatment. A screening program at the National Institutes of Health has already uncovered one selenium derivative which is half again as active as sodium selenite and has 50% of the potency of Factor 3 (*43*). This derivative, diseleno-γ,γ'-di-*n*-valeric acid, was one of 200 compounds screened for protective activity against liver necrosis in rats. In another approach, California workers (*11*) are attempting to develop an orally administered, slowly soluble "Perma-pill" containing selenium, to make repeated dosing of animals unnecessary.

The combination of phenothiazine and selenium administered orally has proved remarkably effective in New Zealand in improving growth of unthrifty animals and in controlling death losses (*9*, 10). This suggests that animals may be suffering from a combination of selenium deficiency and parasite infestation, and that some synergism may occur between these two treatments. A selenized phenothiazine is commercially available in New Zealand.

Direct soil amendment may eventually prove the most economic and most beneficial method of supplementation. While acute selenium deficiency manifests itself in WMD and subnormal growth of young cattle and sheep primarily in the spring and summer, subacute deficiencies probably take their toll throughout the year. Weekly injections of selenium to ewes from lambing to weaning have proved beneficial not only to lambs but to the ewes themselves. Treated ewes not only gained more weight than untreated, but produced 5 to 15% more wool (*9*).

While some trial topdressing applications at 1 ounce of selenium per acre annually appear to have given good results in New Zealand, topdressing trials elsewhere in that country at 2 ounces of selenium per acre (as sodium selenite) had no apparent adverse effect on growth of lambs grazing at the time of application, but did have an adverse effect on fertility when applied at breeding time. Selenium content of dry forage from pasture topdressed at the 2-ounce rate was 2.80 p.p.m. after 6 weeks and 0.31 p.p.m. after 10 months.

The low level of selenium in clover in a mixed grass–clover pasture topdressed with various levels of selenium has already been noted (Table II). Further data on topdressing are expected from a cooperative trial on alfalfa currently planned by Oregon State University and the U. S. Department of Agriculture (*5*).

Therapeutic and Toxic Dosages

Excessive amounts of selenium in forage cause the toxic effect known as alkali disease, alkali staggers, or blind staggers. In the United States, this condition occurs in scattered local areas in the Rocky Mountain states. It is characterized by general dullness, lack of vitality, emaciation, stiffness, and lameness. Horses lose the long hair from the mane and tail, and cattle from the switch. Hoofs separate and may slough off.

Affected stock have generally grazed forage containing up to 25 p.p.m. selenium for several days or weeks on end. Diseased animals thus ingest some hundreds of milligrams of selenium in a relatively short period. The danger level in forage is estimated at 5 p.p.m. selenium (*13*). By contrast, the recommended dosage to correct selenium deficiency is only 2.5 mg. per 100 pounds of body weight every 1 to 3 months. Based on the limited toxicity data available, Schwarz has estimated the therapeutic index (ratio of lethal dose to therapeutically effective dose) of sodium selenite at about 100 (*43*). This factor is considerably greater than that for many common drugs administered to both humans and animals.

Accordingly, when applied under professional supervision, selenium treatment of deficient stock appears to present no hazards to animal health.

The toxicology of selenium has recently been critically reviewed (*18*), and the USDA has issued a handbook covering toxicity in agriculture (*13*).

Analytical Methods

The minute amount of selenium required by animals necessitates extremely accurate methods of analysis. Much of the basic research was made possible by the availability of radioactive sources and sensitive procedures of neutron activation analysis. This method is particularly useful in determining selenium content of plant and animal tissues, but is limited to those laboratories having access to special equipment. The practical limit of sensitivity is reported to be 0.01 μg. of selenium (*13*).

A fluorometric technique using 3,3′-diaminobenzidine and sensitive to 0.02 μg. of selenium will probably find wider use (*54*), particularly as recently improved (*24*).

Future Developments

The present survey found that basic or applied research on selenium in animal nutrition is now in progress at some 30 research centers, including at least 15 in the United States. The high level of research activity promises continuing advances both in our basic understanding of selenium and in its practical application in agriculture.

In basic research, some major problems for the future are determination of the metabolic role of selenium; development of new methods of field application to prolong the effectiveness of treatment and lower the cost of treating individual animals; determination of soil types and tillage practices liable to produce selenium deficiency; and identification of crops which, through poor selenium uptake, will induce deficiency symptoms.

Meanwhile, better practical application of knowledge already available can bring substantial immediate gains in livestock production. This involves more careful diagnosis in the field of WMD, apparent pneumonia, and selenium-responsive scours; investigation of possible response to selenium in cases of such symptoms as ill thrift and infertility, particularly in the areas of known WMD shown in Figure 1; and wider use by professionals of the permitted methods of selenium treatment already available.

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