THE TRACE ELEMENT MANGANESE [1]) THE STATE OF RESEARCH IN THE NETHERLANDS

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It was not until the beginning of the present century that interest was focussed on the grey speck disease of oats, although it had already been known 30 to 40 years before.

SJOLLEMA and HUDIG (1909) demonstrated the importance of manganese sulphate as a reducing agent, but without considering manganese deficiency as being the cause of this disease. As a result of their investigations they came to the conclusion that "....in all probability the oats disease is due to a change in the condition of the soil which weakens and in some cases halts a physiological function which we may briefly describe as root activity". They refer to the alkalinity of the soil together with the nature of the organic material as being the cause of this reduced root activity. SJOLLEMA believes that to excessive amounts of materials (peroxides) harmful to the plants are very probably formed by increased autoxidation caused by the alkaline reaction. According to this theory the good effect of manganese sulphate would be due to the catalytic decomposition of these harmful materials.

GERRETSEN (1936) later demonstrated that harmful materials, although of a different source than that mentioned by SJOLLEMA, were an important factor in the occurrence of typical symptoms. HUDIG and MEIJER (1919) concluded from their experiments that traces of organic matter and an alkaline reaction were necessary for the disease to occur. They ascribe a certain role to the micro-organisms as these are required in order to decompose the organic matter.

SAMUEL and PIPER (1928) were the first to demonstrate that manganese deficiency is responsible for the symptoms of grey speck disease of oats. After the investigations of SJOLLEMA and HUDIG et al. the cause of this disease received little more notice in the Netherlands. The investigation lost much of its practical significance once the reducing agent had been discovered. Moreover, attention was claimed by two other soil diseases, viz. magnesium deficiency ("Hooghalen disease") and copper deficiency ("reclamation disease").

ABERSON and EVERSMAN (1927) found that the disease could be prevented by sterilising the soil. SAMUEL and PIPER (1927) made the same observation, but were unsuccessful in sterilising with formaline. They therefore concluded that the good effect of sterilising with steam was primarily due to the decomposition of the manganese and in no case to destruction of any noxious organisms present. GERRETSEN (1936) again investigated the importance of micro-organisms in the occurrence of grey speck disease of oats. By taking additional precautions to prevent re-infection he succeeded in curing deseased soil by sterilising it with formaline. GERRETSEN concluded from his experiments that micro-organisms played an important part in causing the symptoms of grey speck disease.

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[1]) Received for publication May 12, 1958.

The function of manganese in the plant

Manganese has a function in all kinds of enzymes and has an effect on the formation of vitamins. Gerretsen (1949 and 1950) has demonstrated the importance of manganese for photosynthesis. Measurements of redox potentials of leaf suspensions make it seem likely that manganese plays a decisive part in the oxidation-reduction system of photosynthesis. Iron counteracts the effect which manganese has on photosynthesis. It is possible that this may afford an explanation of the iron-manganese antagonism observed by various research workers. Various symptoms occurring in grey speck disease of oats are explained by Gerretsen (1956) as being due to the fact that manganese is essential for photosynthesis. Thus he sees, for example, a connection between resistance to cold and manganese deficiency. The resistance to cold is determined, among other things, by the osmotic pressure of the cell sap. The higher the osmotic pressure, the more difficult it will be to withdraw water from the cell content during the freezing period. The osmotic pressure not only depends on the salts, but to a very great extent on the assimilates present in the cell sap, among which the sugars occupy an important position.

Symptoms of manganese deficiency

Leaf chlorosis is a general symptom of manganese deficiency. A plant deficient in manganese has a light-green colour, but the typical leaf symptoms vary. Oats are the most sensitive cereal crop. The leaf symptoms appear in the early spring on the first leaves to be formed. The basal parts are somewhat yellow and greyish-brown necrotic spots or bands appear on the leaves. These spots gradually spread.

Typical signs of manganese deficiency in *oats* are considered to be sagging of the leaf, forming a sharp kink, and the fact that the leaf-top remains green longest. Gerretsen (1936) observed that in sterile cultures chlorosis first occurred in the youngest leaves.

In *wheat* the symptoms are more or less the same as in oats, except that the spots have a more greyish colour. In wheat whitish-brown bands may be formed between the veins.

In *barley* the leaf becomes pale green. Very light and dark-bordered, and occasionally also rust-brown spots appear between the main veins, starting from the margin. These spots coalesce on the leaf margin. But in the centre of the leaf they consist of necklace-shaped rows.

In *rye*, the least sensitive of the cereals, the leaf is light-coloured. When there is a serious deficiency of manganese whitish-yellow to whitish-brown bands are formed without a dark margin. According to Rademacher (1952) the top dries first in the case of rye.

Beet is also very sensitive to manganese deficiency. The young plants have a more than normally light-green colour between the veins. Immediately along the veins the colour is the normal green. The leaves are somewhat erected and the leaf margins curl up somewhat. Brown, dry spots gradually form in the light areas. When these spots coalesce large holes may be formed in the leaf. De Haan (1934) showed that the above-mentioned symptoms in beets are due to manganese deficiency.

Potatoes are less sensitive to manganese deficiency. The symptoms in potatoes have been studied by Van Schreven (1935, 1939). Chlorosis occurs in the

top part of the plant. A few days later numerous necrotic spots appear along the veins. The spots generally form on places which were previously chlorotic, but they occasionally appear without the tissue being chlorotic.

In *peas* manganese deficiency causes marsh spot. As early as 1894 MANSHOLT reported in the Netherlands agricultural journal the occurrence of brown spots inside peas. The external appearance of the peas is usually normal. If the pea is split in two a brown spot can be observed on the flat inner surface of one, and generally two cotyledons. These spots may vary greatly in size from a scarcely visible dot to about half the entire surface. When the seed is severely affected the spot is deep-set and the discoloured part may extend to the skin. The spots are situated in the centre of the cotyledon, usually in the extension of the plumule. The plumule may be wholly or only partially brown and withered.

MISS DE BRUYN (1933) showed that the symptom of marsh spot in peas was not caused by parasitic attack. She also ascertained that this symptom occurs in its worst form in the heaviest peas and that the greatest number of affected peas are to be found in the thickest pods. The seed quality of peas with marsh spot depends on which part is injured.

MISS LÖHNIS (1936) expressed the opinion that there was a minimum of a particular element. She found that the manganese content of diseased peas was about 0.0075 % and of healthy peas 0.0125 %. This led her to assume that marsh spot was caused by a deficiency of manganese.

MISS LÖHNIS' (1936) investigation was followed up by KOOPMAN (1937) who carried out manganese spraying experiments. It was found that spraying with a 1 % manganese sulphate solution immediately after flowering and repeating the spraying some three weeks later considerably reduced the percentage of marsh spot. OVINGE (1937) found that the percentage of marsh spot could be reduced by dressing the soil near flowering time with 200 kg of manganese sulphate per hectare. In general dressing about flowering time gave better results than early application. MISS DE BRUYN (1939) proved that marsh spot is caused by manganese deficiency.

As regards taste, peas with marsh spot are inferior for consumption. Where the percentage is 15–20 % they have no more than a middling to fairly good taste. With percentages of between 20 and 30 % the taste is usually on the borderline between adequate and inadequate, while with percentages of over 30 % the taste is predominantly inadequate (VEENBAAS and POLL, 1950). The permissible percentage of marsh spot is smaller for consumption than for seed. The standard established for 1957 was 15 % marsh spot, including 4 % badly affected.

The maximum percentages allowed by the Netherlands General Inspection Service for Agricultural Seeds and Seed Potatoes (N.A.K.) are 15 %, charging the percentage slightly affected for $^1/_3$. No relation has been found between cooking quality and marsh spot. There is no increase in the percentage of marsh spot on storing.

The external symptoms of manganese deficiency in peas are little known in the Netherlands. According to RADEMACHER (1952) the youngest leaves turn yellowish-green between the veins, and they remain small and curl downward. In severe cases there is no pod formation, and in less severe cases the seed formation is normal but with marsh spot. Probably in most cases the

external symptoms are not or hardly visible, since there is no further development of the plant if manganese deficiency occurs during growth.

Beans develop chlorosis between the veins when suffering from manganese deficiency. In severe cases the entire leaf is yellow to yellowish-green with the exception of the veins which stand out in marked contrast. The seeds may have marsh spot.

Red clover and *lucerne* with manganese deficiency have retarded development; the leaves become light green to bronze yellow, and the veins remain green.

Deficiency of manganese in *flax* causes chlorosis and a twisted top.

In *floriculture* manganese deficiency chiefly occurs in roses and chrysanthemums, and to a lesser extent in *Primula obconica, Syringa* and *Gerbera.* There is also chlorosis and the veins remain green. Moreover in lilac brown spots appear on the leaves. (WEZENBERG, 1955).

In *fruit trees* (apple, pear, cherry, peach and grape) manganese deficiency causes chlorosis of the leaves. This chlorosis is characterised by irregularly shaped yellow bands between the veins, while the tissue along the veins remains green. All varieties of peach have practically the same degree of sensitivity, but plums and apples vary in sensitivity. The Czar, June Blood and Golden Japan varieties of plum are very sensitive, whereas Ontario and Utility scarcely ever exhibit chlorosis. The Cox's Orange Pippin, Lombartscalville and Transparant de Croncels varieties of apples are more sensitive to manganese deficiency than other varieties. Except for type IX, apples on weaker rootstocks are more sensitive to manganese deficiency than on strong rootstocks. Manganese deficiency often occurs in type IV in particular. The Doyenné du Comice, Bonne Louise d'Avranches and Conférence varieties of pear are more sensitive than other varieties. Pears on a quince rootstock (especially on quince C) are more sensitive. (BUTIJN, personal communication).

In *market gardening* manganese deficiency chiefly occurs in tomatoes and cucumber. The leaves become chlorotic between the veins and are thin. Lettuce suffering from manganese deficiency has thin leaves and is more sensitive to "whiteness" *Bremia lactucae.*

FACTORS AFFECTING THE OCCURRENCE OF MANGANESE DEFICIENCY

1 *Micro-organisms*

GERRETSEN (1936) found that oats in quartz sand having a low manganese content did not display the typical symptoms of grey speck disease under sterile conditions, although the symptoms did occur when a little diseased soil was added. Under sterile conditions only the growth was retarded, this being accompanied by slight tillering, a reduced root system and a smaller grain yield. There was a complete absence of the typical infiltration spots which pass over into leaf spots. Such spots occurred, however, as soon as the soil was mixed with diseased, unsterilised soil, with roots of diseased plants or bacteria isolated from these roots. In view of these observations it is necessary to distinguish between the purely physiological effect of manganese deficiency (retarded growth) and the symptoms described as typical of the grey speck disease.

The root system of plants suffering from typical grey speck disease always appears to be more or less seriously affected and sometimes entirely rotted

away, whereas the healthy plants have a sound root system. The root-top is the first part to be affected. This is shown by the gradually spreading glassy consistency of the root-tops which turn yellow or brown at the same time. In a more advanced stage the root-top often disappears and the roots themselves are injured. The alkaline decomposition products formed when the roots are thus affected are conveyed by the flow of sap to the leaves where they destroy the tissue and give rise to the typical leaf spots. GERRETSEN demonstrated in two ways that the typical leaf spots are caused by these alkaline decomposition products, since he found that the cell content of such a leaf spot turns red with a 0.1 % phenol red solution, indicating that the cell content has a pH of over 8.2. Moreover, the leaf spots accumulate below the plane of the cut when the flow of sap is interrupted by making an incision in the leaf. Hence the above shows that the leaf spots are secondary symptoms. According to GERRETSEN, however, it is also possible for necrotic spots to form without the intermediary of micro-organisms when there is extreme manganese deficiency. According to him the carbohydrate deficiency due to reduced photosynthesis when there is a shortage of manganese may cause autolysis of the protein compounds in the cells. Ammonia is formed as the final product, where by the cell sap gets an alkaline reaction.

In addition to these bacteria which injure roots, GERRETSEN distinguished a further group which plays a part in this disease. When the pH is high these bacteria are capable of precipitating in the form of manganese dioxide the manganese available to the plant.

2 *Acidity*

Manganese deficiency mainly occurs in soils which have a neutral to alkaline reaction. LUNDEGÅRDH found that the solubility of manganese in natural soils is at the minimum near the neutral point, and increases towards the acid and alkaline side. Consequently HUDIG et al. first of all recommend lowering the pH when grey speck disease occurs.

MASCHHAUPT (1934) found that manganese deficiency could be cured by applying lime too. In a "rodoorn" [2]) soil having a pH of 5.7 and a humus content of 12.5 %, he succeeded in curing manganese deficiency by applying 15 to 20 tons of CaO per hectare. Some years afterwards, however, wheat sown on this plot was found to be suffering from serious manganese deficiency.

The oxidation of the bivalent manganese by micro-organisms, demonstrated by GERRETSEN, takes place in the pH range of 6.3 to 7.8 (pH-H_2O), i.e. in the range in which manganese deficiency also frequently occurs in practice. DE GROOT's (1955) investigation shows that on diluvial sandy soils the only factor determining the chance of manganese deficiency is the pH, although other factors are of importance on marine soils.

3 *Organic matter*

It is not clear what part is played by the organic matter in the conversion of bivalent manganese. In the Netherlands manganese deficiency was first found on soils having a pH of over 6 and a high content of organic matter. These were sandy and reclaimed peat soils (grey speck disease of oats).

HUDIG and MEIJER (1919) found that serious manganese deficiency symptoms occurred when organic material was added to sand cultures. When oat leaves

2) Group of the clay over peat soils.

were added instead of cellulose the plants remained healthy, but they became diseased when the leaves were extracted with NaOH or acids before being added. Apparently the leaves were more readily attacked by micro-organisms after this extraction.

De la Lande Cremer (unpublished) obtained manganese deficiency in his pot culture experiments by adding an aqueous extract of wheat straw. Hudig et al. obtained the same result with an extract of oat roots. Hence in general manganese deficiency increases when organic matter is supplied, so that ploughed-up grassland is usually regarded as dangerous and likely to lead to manganese deficiency. However, in 1947 a green dressing applied in previous years in the Wieringermeer district had a good effect with regard to manganese deficiency. Various foreign research workers (including Mattson, 1948) observed that when the soil has a high pH the organic matter is able to reduce higher manganese oxides to bivalent manganese.

4 *Fertilisation*

Generally speaking it can be said that the better the development of the plant the greater is the chance of manganese deficiency when insufficient manganese is available. All physiological acid fertilisers reduce the manganese deficiency. Hence unlike nitrate fertilisers, ammonia fertilisers will usually cause a reduction in the manganese deficiency. This effect is indirect and is caused by a shift in the pH. E. G. Mulder (1941) demonstrated that when the pH remains constant, plants fed with ammonia nitrogen have a somewhat greater need of manganese than those dressed with nitrates.

The *Aspergillus niger* mould, which is often employed as an indicator plant in studying deficiency in trace elements, also requires more manganese when fed with nitrogen in the form of ammonia than in the form of nitrate. Hudig et al. (1926) found that manganese deficiency occurred on a neutral soil with copper deficiency after the latter had been cured. E. G. Mulder (1938) obtained the same results and also found that the microbiological oxidation of manganese was promoted by the addition of copper. In aqueous cultures with rye it was found that more manganese was required as more copper was introduced; this did not occur in the case of barley, however, although when a heavy manganese dressing was applied a MnO_2 precipitate was found to form on the barley roots of the cultures which had copper, unlike the cultures which were poor in copper. Hence this indicates that the biological oxidation of manganese is activated by copper. A generous dressing of copper sulphate on a soil deficient in manganese but not deficient in copper was found to increase manganese deficiency in canary-seed.

The occurrence of manganese deficiency in the Netherlands

Manganese deficiency is of frequent occurrence in the Netherlands. From an enquiry into the occurrence of marsh spot in peas, Ovinge (1935) concluded that the various varieties were not all equally susceptible and that the phenomenon occurred in its worst form in late marine clay polders, particularly on the heavier soils.

In 1954 an investigation into the occurrence of manganese deficiency was started by the T.N.O. Agricultural Experimental Station (the present Institute for Soil Fertility). This reconnaissance revealed that on diluvial sandy soils

manganese deficiency rarely occurred on entire fields, but was usually found in small patches. A great deal of manganese deficiency occurs in the pumped-up soils in the district south of the Rotterdam harbour-works. It also occurs on Goeree-Overflakkee on the deep-ploughed fields, dune sands, and light and medium-heavy soils. It is also of frequent occurrence in the Haarlemmermeer district and the Anna Paulowna polder, as well as in the Biesbosch district. A great deal of manganese deficiency is also found in the deep-ploughed fields in the Legmeer district and in the northern dune-sand area on ploughed-up grassland. In Zealand Flanders it is of fairly general occurrence, e.g. in the Braakman polder. Manganese deficiency occurs on the deep-ploughed fields in the Vier Ambachts polder, although it was unknown on these fields before they were deep-ploughed. Manganese deficiency does not occur on all deep-ploughed fields in the Zevenhoven polder where the soils contain less humus and are heavier. Numerous cases of manganese deficiency have been recorded too in the clay district of North Groningen and in the North-East Polder.

Manganese deficiency is one of the most common nutritional diseases in fruit-growing.

Soil research

Two methods are evolved by the Institute for Soil Fertility for determining the amount of manganese in the soil available to plants. Gerretsen is working on the development of a microbiological manganese determination in soils and crops, and De Groot is engaged on chemical determinations of manganese.

De Groot's investigation (1956) shows that the content of exchangeable manganese does not constitute a good index to the amount of manganese available in the soil. It is better to use the content of reducible manganese. From an investigation of a great number of samples, De Groot concludes that as regards the occurrence of manganese deficiency a distinction should be made between the marine loam and clay soils and the diluvial sandy soils. According to De Groot, except for the Biesbosch district and the North-East polder, manganese deficiency may be expected on marine loam and clay soils with a high pH and humus contents of less than 2½ % when the content of reducible manganese is less than 60–70 p.p.m. When the humus contents are higher this limiting value increases to 100 p.p.m., although deficiency symptoms are not impossible at values exceeding 100 p.p.m. In the Biesbosch district, the North-East polder and Kreekrak polder, the deficiency may also occur with extremely high contents of reducible manganese.

According to De Groot (1956) the inadequate supplies of manganese in the estuary soils (Biesbosch district) might be explained by fixation to organic chelate compounds of the small amount of bivalent manganese formed by reduction of the higher oxides. This fixation would occur at a high C/N quotient. By making use of the C/N quotient De Groot was able to distinguish between diseased and healthy soils in the Biesbosch district. According to him the diseased soils in the Biesbosch district will return to health when there is a further decomposition of the organic matter resulting in a decrease in the C/N quotient. This tallies with what has been found in this area, viz. that manganese deficiency only occurs in the later sediments. In the North-East polder the C/N quotient is high in both diseased and healthy soils. This is explained by the fact that unlike the Biesbosch district, in the North-East

polder there is practically no decomposition of the organic matter. In general the contents of reducible manganese increase with the heaviness of the soil. A detailed investigation is now being undertaken into the manganese status of clay and loam soils in the Netherlands.

So far as is known, the occurrence of manganese deficiency in the diluvial humus sandy soils depends solely on the pH. All fields examined by DE GROOT which had a pH-KCl higher than 6.2 were found to be diseased, while those with a pH-KCl of less than 5.4 were healthy. Both diseased and healthy fields occurred between these two pH values. The content of reducible manganese in these soils was generally less than 60 p.p.m. The dune sand soils, in which manganese deficiency is frequently absent even at a high pH, are the subject of investigation.

CONTROL OF MANGANESE DEFICIENCY

Manganese deficiency can be prevented on *diluvial sandy soils* by ensuring that the pH-KCl does not rise beyond 5.4. Manganese deficiency can be cured on such soils by dressing with manganese sulphate and employing physiological acid fertilisers, as a result of which the pH may also permanently decrease.

The commonly used method of controlling manganese deficiency in arable crops cultivated on *clay soils* is spraying with a solution of 1½% manganese sulphate (12–15 kg manganese sulphate per hectare). In this connection it is important to know what is the most suitable period in which to carry out spraying and how frequently it should be done. Experiments conducted by DE HAAN (1934) showed that manganese deficiency recurred in beet after spraying; consequently spraying should be carried out several times. WIESBOSCH (1948) obtained a further important effect in potatoes by carrying out a second spraying. HENKENS (1955) found that winter wheat was best sprayed about the time that manganese deficiency became visible. Two sprayings resulted in a considerably better yield than one. These experiments also showed that serious leaf scorching is liable to occur in cereals. It was found that one spraying of beet scarcely affected the yield, whereas two sprayings resulted in a yield increase of nearly 10%. But in many cases no effect of spraying was observed, despite of the fact that manganese deficiency was in evidence throughout the year on the check plots. No effect of spraying was observed when the beet suffered from manganese deficiency during the beginning of the season only (not yet published).

Other still unpublished experiments show that the time of spraying is important. This causes no difficulty when one is dealing with visible manganese deficiency, but in cases of latent manganese deficiency (without visible symptoms) it is impossible to determine when the manganese supply of the plant stagnates. That latent manganese deficiency may occur is shown by an experiment carried out by the Farming Advisory Board in the Wieringermeer district (1952) where spraying potatoes with manganese sulphate resulted in a 16% increase in yield, the under-water weight also increasing. No symptoms of manganese deficiency were observed. In a similar case HENKENS (1957) obtained an average increased yield of over 3 tons, or 12%, by applying manganese sulphate (100–600 kg per hectare) to seed potatoes on a soil which had a low content of reducible manganese.

The Institute for Soil Fertility is investigating whether fertilising the soil

produces better results than spraying the crop; experiments with chelate compounds are also being undertaken within the scope of this investigation. In this connection a study is being made of the movement of manganese inside the plant.

GERRETSEN (1956) found that there were varieties of oats which are resistant to manganese deficiency. Admittedly these resistant varieties require manganese for normal growth, but a shortage of manganese has less serious effects in this case. According to GERRETSEN this "resistance" is due to the fact that the root system has a great resistance to saprophytic bacteria. Moreover, when there is little manganese available, the carbonic acid assimilation of the resistant varieties is twice as great per unit of surface area as that of the non-resistant varieties.

Spraying with a 5% manganese sulphate solution before the buds develop has a good effect on the leaf colour of fruit trees in the summer (D. MULDER, 1953). It is found, however, that one or two sprayings with a 0.2–0.5% solution to which 0.1–0.25% of spraying lime has been added give more satisfactory results.

In the case of fruit trees grown under glass various control methods are employed. With peaches and plums good results are obtained with the borehole method and by spraying before the buds develop. In the borehole method manganese salts (preferably not in powder form) are inserted in holes in the stem, the holes then being properly sealed off with cork and red lead. VAN KOOT and VAN DEN ENDE (1955) found that the most satisfactory material was manganese citrate. One gram of manganese citrate per sq. metre of stand is sufficient for plums; in the case of peaches it is better to employ a somewhat larger quantity. Young trees should be given rather less. The treatment should be applied early, preferably in January, and is sufficient for 3 to 4 years. The borehole method should not be employed if the stem has a diameter of less than 4 cm. A drawback of this method is that it may spread such diseases as silver leaf and bacterial canker. Furthermore there is sometimes considerable gum formation.

Good results have also been obtained by winter spraying. This is done about January on the bare wood. A quarter of a litre of 5% manganese sulphate solution is used per sq. metre of stand. The operation should be repeated every year.

According to VAN KOOT and VAN DEN ENDE the borehole method and winter spraying are unsuitable for grapes. The stem is too thin and the bark too rough, with the result that the spray liquid does not enter the inner bark. Pruning-cut treatment appears to give good results. In this treatment the pruning cuts are smeared with a paste containing 400 grams of manganese sulphate, 400 gram of sugar and 300 grams of powdered sulphur. Apparently the manganese is well absorbed by the pruning cut, since the shoots remain green. Shoots from dormant buds, however, suffer from manganese deficiency. Hence every pruning cut should be smeared over. It is advisable not to apply the pruning cut treatment too early.

Reference may be made to the review of the literature by VAN ALPHEN (1956) for a detailed conspectus of manganese spray treatments in the Netherlands and abroad.

Manganese excess

During the war Löhnis (1946, 1950) observed a peculiar injury to *kidney beans* in a soil which had a low pH. The young leaves were yellow, mostly between the veins and along the leaf margins, and the plants showed very retarded development. The older leaves exhibited a characteristic spotted pattern, viz. yellow or colourless areas with necrotic patches and a lumpy leaf surface. In severely affected plants brownish-purple spots appeared on the leaf stalks of the first leaves formed, and the veins on the back of the leaf were purplish-brown, sometimes continuously and sometimes speckled. The most severely affected plants only produced an occasional flower and no seed. A subsequent investigation undertaken by Löhnis showed that the said phenomena are to be attributed to an excess of manganese. Löhnis observed that manganese toxicity depends on other factors besides the manganese concentration. In a culture solution according to Van der Crone serious disease symptoms occurred at 10 mg manganese per litre, whereas in a solution according to Zinzadse the same amount of manganese caused no injury. The temperature also has an effect. Whereas plants in a hothouse exhibited no manganese toxicity, the symptoms did occur a day after they had been placed in the open. French beans are more sensitive than kidney beans.

The behaviour of *vetch* is about the same as that of beans. Even at an early stage purplish-brown specks occur along the margins of the young leaves, followed by continous small margins of this colour. The plants remain small and die early.

Lucerne exhibits small yellow margins and necrotic specks on the leaves and very retarded development. The margins of the older leaves become brown and crumpled. *Red clover* develops small yellow margins on the young leaves. The plant often outgrows this at a later period. *White clover, white mustard, strawberry, tobacco, mangold, flax* and *oats* are little affected by manganese toxicity.

Subsequent investigations carried out by Löhnis (1952 and 1954) showed that potatoes are also sensitive to manganese excess. The symptoms first occur as very fine necrotic spots between the veins of the lowest leaves. The specks are most distinct on the upper side. Necrotic spots also occur on the veins and leaf stalks, and also on the stem in cases of severe affection (Van Schreven, 1939).

Miss Löhnis (1954) found that magnesium sulphate reduces the injury caused by manganese excess. Growth is restored when magnesium sulphate is applied, and manganese toxicity symptoms only occur in the older leaves. The magnesium sulphate has a highly suppressive effect on the manganese content. E. G. Mulder (1954) found that manganese excess promotes molybdenum deficiency.

The importance of manganese to animals

Numerous investigations have shown that manganese is necessary to humans and animals. Manganese is a component of arginase and activates peptidase, phosphatase and the carbohydrate- and fat-splitting enzymes. (Seekles, 1957). It is also an important factor in the detoxification process of the body (Grashuis, 1957). Thus the toxic effect of vitamin B_1 is prevented by additional manganese. Apparently manganese also plays a part in the oxidation of mono-

saccharides to ascorbic acid and would therefore be very important to animals which depend on the fodder for their uptake of vitamin C. (SEEKLES, 1957).

Manganese deficiency causes anomalies in bone formation and disturbances in the sex cycle. In male animals it promotes sterility, and in females the ovulation is disturbed. Other investigations show, however, that rats retain their normal ovulation with manganese deficiency and normal young rats are born. But the mothers do not suckle their young, which remain small and substandard. (LÜHRS, 1952).

In *birds* manganese deficiency affects egg production and incubation results, and causes perosis or "slipped tendon", a disease of which the symptoms are bent and twisted legs. It is caused by the fact that the achilles tendon grows more rapidly than the leg.

In *pigs* manganese deficiency causes stiffness accompanied by swollen knees and hocks, and crooked legs. GRASHUIS et al. (1952) found manganese deficiency in cattle. *Heifers* suffering from manganese deficiency make poor growth with under-development of the rump and slight development of the abdomen. Moreover it is noticeable that the hocks are steeply inclined and the pastern somewhat out of joint. The fecundation percentage is low and there is a good deal of abortion. The coat is withered, dry and brown-coloured.

The calcium and phosphate concentration in the fodder affects the animal's manganese function. The amount of manganese required to prevent perosis increases with increasing lime and phosphate in the fodder (BROUWER, 1957).

Manganese chiefly occurs in the liver, skin, hair, pancreas, legs, blood, kidneys and suprarenal glands, viz. decreasing from 12 to 3.7 mg per kg of dry matter. In addition the tissues contain 0.2 to 0.4 mg manganese per kg of dry matter. According to GRASHUIS determination of the manganese content in the hair gives clear indications. The manganese content of the hair is normally about 10 mg per kg. Analysis figures of VAN KOETSVELD (ref. GRASHUIS) show that the manganese content of the hair of manganese-deficient cattle falls to 2 to 5 mg per kg.

GRASHUIS believes that there is a risk of manganese deficiency in cattle when the manganese content of the grass is less than 100 p.p.m. Lower limiting values are given in foreign literature. The manganese content of the grass is substantially determined by the pH of the soil (HENKENS, unpublished). The manganese content of grass is at its lowest when the pH is high. According to GRASHUIS 4 grams of manganese sulphate per day and per animal is sufficient for the entire recovery of cows, while the amount required for heifers and calves is 2 and 1 grams respectively.

SEEKLES (1950) was unable to confirm the hypothesis of BLOKMORE et al. (1937) that there was a connection between the occurrence of grass tetany and the manganese uptake. In the Netherlands there is no difference between the manganese content of the grass in farms affected by tetany and healthy farms.

Excess manganese causes manganism. This results in a drop in the blood pressure, drowsiness, muscle-twitching, a high pulse and respiration frequency, and haematuria (LÜHRS, 1952). The extent to which the occurrence of nymphomania in cows is due to manganese toxicity is a subject of investigation (GRASHUIS).

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