History of Zinc in Agriculture1,2

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ABSTRACT

Zinc was established as essential for green plants in 1926 and for mammals in 1934. However, >20 y would pass before the first descriptions of zinc deficiencies in farm animals appeared. In 1955, it was reported that zinc supplementation would cure parakeratosis in swine. In 1958, it was reported that zinc deficiency induced poor growth, leg abnormalities, poor feathering, and parakeratosis in chicks. In the 1960s, zinc supplementation was found to alleviate parakeratosis in grazing cattle and sheep. Within 35 y, it was established that nearly one half of the soils in the world may be zinc deficient, causing decreased plant zinc content and production that can be prevented by zinc fertilization. In many of these areas, zinc deficiency is prevented in grazing livestock by zinc fertilization of pastures or by providing salt licks. For livestock under more defined conditions, such as poultry, swine, and dairy and finishing cattle, feeds are easily supplemented with zinc salts to prevent deficiency. Today, the causes and consequences of zinc deficiency and methods and effects of overcoming the deficiency are well established for agriculture. The history of zinc in agriculture is an outstanding demonstration of the translation of research into practical application. 

Introduction

Zinc always has been a significant mineral element for agriculture. However, realization of this significance initially developed slowly. The first indication that zinc could have an impact on agricultural production appeared in 1869 when Raulin (1), a student of Louis Pasteur, reported that zinc was a required nutrient for the growth of Aspergillus niger, a fungus that causes black mold in some agricultural produce, including grapes, onions, and peanuts. That remarkable finding remained dormant until 1911 when Bertrand and Javillier (2) confirmed the finding of Raulin. Three years later, Mazé (3) reported that maize grown by using hydroponic methods required zinc for growth and development. This report stimulated attempts to confirm that zinc was required by plants, but the crude methods and the impurity of supposedly pure chemicals used to grow plants produced inconsistent results. Thus, the requirement of zinc for plant life was questioned until 1926 when Sommer and Lipman (4) showed that zinc was required for the growth and development of sunflowers (Fig. 1) and barley. This finding resulted in zinc being generally recognized as essential for higher green plants.

The first indication that zinc might be required by animals appeared in 1905 when Mendel and Bradley (5) reported that zinc was a constituent of the respiratory pigment (hemocyanin) of a snail. The first suggestion that zinc might be required by higher animals appeared in a 1919 report by Birckner (6), in which he stated that “from its constant occurrence in the yolk of eggs as well as in cow’s and human milk, it is inferred that the element zinc exerts an important nutritive function, the nature of which is not at present understood.” There were several attempts in the laboratories of some eminent nutrition researchers, including Gabriel Bertrand (7,8), James McHargue (9), and Lafayette Mendel (10) to show that zinc was nutritionally essential for higher animals in the 1920s, but these attempts were stymied by the use of diets that apparently were deficient in other essential nutrients, especially vitamins. These early studies found some small favorable effects on the growth of rodents, but it was not until 1934 that the essentiality of zinc for higher animals was firmly established. Wilbert Todd, Conrad Elevehjem, and Edwin Hart (11) at the University of Wisconsin found that zinc was nutritionally essential for higher animals in the 1920s, but these attempts were stymied by the use of diets that apparently were deficient in other essential nutrients, especially vitamins. These early studies found some small favorable effects on the growth of rodents, but it was not until 1934 that the essentiality of zinc for higher animals was firmly established. Wilbert Todd, Conrad Elevehjem, and Edwin Hart (11) at the University of Wisconsin found that zinc was essential for the growth and well-being of the rat. Shortly thereafter, Gabriel Bertrand and R.C. Bhattacherjee (12) reported that zinc was required by the mouse. These animals may be considered as being associated with agriculture.


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because they are agricultural pests and have been used as experimental models by agricultural scientists.

Zinc in agricultural plant production

After the discovery of the essentiality of zinc for plants, extensive areas in the world were identified as having soils deficient in zinc for the proper growth and development of agriculturally important plants. Sandy and peat soils have low zinc reserves (13). Zinc deficiency is widespread among plants grown in calcareous soils of high pH because zinc is made unavailable through adsorption to clay or calcium carbonate (14). Soils high in phosphorus decrease zinc solubility that can result in zinc deficiency in plants; this effect is likely to be found with large applications of phosphorus fertilizers on soils low in available zinc (14). In waterlogged soils, zinc deficiency occurs because of the formation of sparingly soluble zinc compounds in the oxidized rhizosphere (14). This is the basis for zinc deficiency being the most important nutritional limitation on the yields of flooded rice worldwide. In 1972, zinc deficiency was considered one of the most common plant micronutrient deficiencies in the United States (15). In 1990, a survey of 190 representative soils from around the world indicated that about 49% of them were zinc deficient (16). Fortunately, zinc deficiency can be corrected by soil applications of soluble inorganic salts such as zinc sulfate (14). By 1979, 24 US states had recommended that inorganic zinc salts be added to soils for certain crops (13). An indication of where soil zinc is deficient is provided by occurrence of zinc deficiency in humans. Published maps for zinc-deficient soils and prevalence of zinc deficiency in humans are remarkably similar (17). Low soil zinc could be contributing to the occurrence of zinc deficiency in humans because zinc application to soils significantly enhances grain zinc concentration in rice and wheat (18), which are 2 major food staples in the world. An increased zinc/phytate ratio in grains also increases zinc bioavailability for humans (18). Moreover, increased soil zinc increases the zinc content of roots, pulses, and leafy vegetables. Unfortunately, fertilizers containing zinc salts often are not affordable or are of limited availability in many parts of the world. Thus, although zinc deficiency generally is not an agricultural soil or plant production problem in the United States, it remains a significant problem worldwide.

In addition to decreasing zinc in the edible portion of plants, zinc-deficient soils result in reduced grain yields (18). Grain and seed yield are depressed more than total dry matter production by zinc deficiency, which might be partly caused by impaired pollen fertility (14). The most characteristic signs of zinc deficiency in dicotyledons are stunted growth and a decrease in leaf size or “little leaf” (14). Severe zinc deficiency can cause shoot apices to die, which has been observed in forest plantations in South Australia (19). Chlorosis often is associated with these signs of deficiency (14).

Plant species differ in their sensitivity to zinc deficiency. Corn, cotton, and apple trees are much more sensitive than wheat, oats, and peas (14). Other agricultural crops highly sensitive to zinc deficiency include citrus fruit trees, pecans, grapes, beans, and onions (13). Potatoes, tomatoes, sorghum, and sugar beets are mildly sensitive (13).

Zinc in agricultural animal production

Swine

Recognition that zinc deficiency may be a problem for agriculturally important animals did not occur until 20 y after the discovery of essentiality for rodents. In 1955, Tucker and Salmon (20) reported that zinc supplementation prevented and cured parakeratosis in swine fed a diet low in zinc. The parakeratosis could be enhanced by excess calcium and Salmon (20) reported that zinc supplementation prevented and cured parakeratosis in swine fed a diet low in zinc. The parakeratosis could be enhanced by excess calcium, and the discovery of essentiality for rodents. In 1955, Tucker and Salmon (20) reported that zinc supplementation prevented and cured parakeratosis in swine fed a diet low in zinc. The parakeratosis could be enhanced by excess calcium, and the most characteristic signs of zinc deficiency in dicotyledons are stunted growth and a decrease in leaf size or “little leaf” (14). Severe zinc deficiency can cause shoot apices to die, which has been observed in forest plantations in South Australia (19). Chlorosis often is associated with these signs of deficiency (14).

Figure 1 Sunflowers grown with (left) and without (right) zinc. Reproduced from (4) with permission.
dry matter for breeding and lactating swine (27). Swine production currently often involves large operations that have the services of nutritionists to ensure that zinc is adequate in feed. In addition, feed companies usually add zinc salts to mineral supplements or mixed rations to prevent zinc deficiency in swine. Thus, zinc deficiency is not a problem for the major part of the agricultural swine industry today.

**Poultry**

The discovery of the practical nutritional importance of zinc for swine was a stimulus for determining the importance of zinc for other agriculturally important animals. In 1955, it was reported that chicks fed a semipurified diet containing soy protein required an unidentified mineral (28,29). O’Dell and Savage (30) suggested that zinc was needed by the growing chick in 1957. In 1958, O’Dell et al. (31) reported findings that clearly identified zinc as the unidentified mineral needed by the growing chick fed a semipurified diet containing washed soy protein. Chicks fed the low-zinc, soy protein–based diet exhibited decreased growth, frizzled feathers, shortening and thickening of the long bones, and enlarged hocks (31).

The findings of O’Dell et al. were quickly confirmed by other groups (32–34). Among the groups was one led by William G. Hoekstra and Milton L. Sunde at the University of Wisconsin. They assigned to me a BS thesis project that had the objective of confirming the findings of O’Dell et al. and comparing the zinc deficiency signs in growing chicks fed isolated soy protein or casein hydrolysate (35,36). **Figures 3 through 5** were presented in that BS thesis (35). Figure 3 shows the shortened thickened leg with an enlarged hock of a zinc-deficient chick fed the soy-protein–based diet. The comparison leg is from a chick fed the about the same amount of zinc but with casein hydrolysate as the dietary amino acid source. The abnormal legs apparently were uncomfortable because the chicks preferred not to stand. Figure 3 also shows that feather development was impaired in the chick fed the soy protein–based diet. Figure 4 is an example of the frizzled feathers found on zinc-deficient chicks regardless of the amino acid source. **Figure 5** shows the marked differences that the dietary amino acid source made on the signs of zinc deficiency in the growing chick. In the top row, chicks fed the soy protein–based diet containing 6, 11, and 16 mg Zn/kg had abnormal legs and preferred not to stand. Although the chicks fed the 26-mg Zn/kg diet had normal-appearing legs, their growth and development were impaired compared with chicks fed the 46-mg Zn/kg diet. In the bottom row, only chicks fed the 2- and 7-mg/kg diet showed signs of zinc deficiency that included frizzled feathers and dermatitis but did not include abnormal legs and hocks. Maximum growth was achieved with 12-mg Zn/kg diet with the casein hydrolysate–based diet. This BS thesis project was not the first to show that the dietary amino acid source made a difference in the zinc requirement for the chick. In 1958, it was reported that chicks fed casein-gelatin (32) or egg white (37) had a significantly lower zinc requirement than chicks fed soy protein as the dietary amino acid source.

Determining the basis for the difference in zinc deficiency signs and zinc requirements for the chick was an active research area in the 1960s. Some of the differences, especially growth, were attributed to protein sources containing high amounts of phytic acid, which is a potent chelator of zinc (38,39). High dietary calcium was found to heighten the inhibitory effect of phytate on zinc absorption (39,40). Vohra and Kratzer (41) reported that chelating agents such as EDTA made zinc available for turkey poults fed soy protein diets. However, increasing zinc availability by overcoming...
phytate inhibition ameliorated all deficiency signs; it did not explain the leg defect in chicks fed zinc-deficient diets with soy protein as the amino acid source. In my graduate work (42), I found that increasing dietary histidine could alleviate the leg defect without markedly affecting other signs of zinc deficiency. Work by other graduate students in the joint research group of Hoekstra and Sunde reported that high arginine was most likely responsible for the leg defect (43). Studies in the laboratories of Hoekstra and Sunde (44) and Gerald F. Combs (45) showed that the hatchability and development of the embryo were markedly impaired in chicks hatched from zinc-deficient hens. Zinc-deficient embryos exhibited abnormal skeletal development, including curvature of the spine, shortened and fused lumbar vertebrae, and, in some cases, missing toes. Severely zinc-deficient embryos had no lower skeleton or limbs. Newly hatched chicks from eggs of less severely zinc-deficient hens were weak and could not stand, eat, or drink.

At present, it is recognized that feeding a 20- to 35-mg Zn/kg diet containing an amino acid source other than soy protein will meet the requirement of the chick (26). However, with practical corn-soy protein diets, the requirement is increased to 40 mg Zn/kg for growth, but a diet much higher in zinc (60–100 mg/kg) apparently is needed to prevent frizzled feathers (26). The zinc requirement set for laying hens is a 50-mg/kg diet, and for breeding hens, it is 65 mg/kg diet (26).

In the 1990s, zinc deficiency could be found fairly often in chickens, turkeys, and other poultry fed practical diets (46). Generally, it was not severe, but even moderate zinc deficiency reduced growth, feed efficiency, and egg production (46). At present, 98% of poultry meat in the United States is produced through integrated farming practice, with large companies controlling every aspect of production from feeding practices to placing products on the market. These companies have Ph D nutritionists ensuring that feeds contain adequate zinc by supplementing with compounds such as zinc sulfate and hydroxy zinc. Egg production also is concentrated among a few large producers that use nutritionists to ensure that poultry are fed diets adequate in zinc. In addition, commercial poultry feeds are formulated to contain adequate zinc for small flocks. Thus, zinc deficiency in the agricultural poultry industry in the United States is rare today.

Ruminants
Shortly after zinc deficiency was reported to cause parakeratosis in pigs and chicks, Legg and Sears (47) discovered that zinc supplementation could overcome a parakeratosis found in cattle under natural conditions in Guyana. In 1962, Miller and Miller (48) induced zinc deficiency in young Holstein calves by feeding a purified diet that contained only 2.7 mg Zn/kg. The zinc-deficient calves, one of which is shown in Figure 6, exhibited severe parakeratosis, reduced weight gain, and stiffness of joints with soft edematous swelling of the feet in front of the fetlocks. Calves fed a 40-mg Zn/kg diet did not exhibit this pathology.

The finding of parakeratosis alleviated by zinc supplementation in Guyana (47) was unexpected. The zinc content in forage plants needed for growth and development was generally considered high enough to meet the estimated zinc requirement of 10 to 15 mg/kg dry matter determined for weaned ruminants under laboratory conditions (46,49). The zinc concentration of pasture forage in Guyana was found to be 18 to 42 mg/kg dry matter (47). However, 14 y later, Spais and Papasteriadis (50) reported that 60% of 150 cattle herds in Greece exhibited signs of zinc deficiency, including rough coat, hair loss, and eczematous skin lesions. Shortly thereafter, zinc-responsive skin lesions and depressed growth were reported for cattle in South Africa, Turkey, India, Western Australia, and in the state of Idaho (46). In all these reports, the zinc content of forages generally ranged between 17 and 20 mg/kg dry matter (26). These lower than usual zinc concentrations for forage plants

![Figure 4](https://academic.oup.com/advances/article-abstract/3/6/783/4557955/16785457955) Frizzled feathers of a zinc-deficient chick. Reproduced from (35) with permission.

![Figure 5](https://academic.oup.com/advances/article-abstract/3/6/783/4557955/16785457955) Chicks fed 6-, 11-, 16-, 26-, and 46-mg Zn/kg diet containing soy protein as the amino acid source (top row) compared with chicks fed 2-, 7-, 12-, 22-, and 42-mg Zn/kg diet containing casein hydrolysate as the amino acid source (bottom row). Reproduced from (35) with permission.
apparently were caused by low amounts of bioavailable zinc in soils in these areas. According to McDowell (46), the critical forage zinc concentration needed by ruminants under pasture conditions is 30 mg/kg dry matter.

At the same time that grazing cattle were found to be responsive to zinc supplementation, zinc deficiency was found in grazing sheep and goats in areas where forage contained less than the critical concentration of zinc determined by McDowell. In 1972, reproductive responses (e.g., increased conception rate) to zinc supplementation were found in grazing sheep in South Australia (51). In 1983, it was reported that ewe and lamb mortality and skin lesions were alleviated by zinc supplementation in a flock of sheep in Sudan (52). Hair loss and skin lesions associated with low zinc intake also have been found in India, Turkey, and the United States (46). Other signs of zinc deficiency in sheep and goats include depressed growth, soft and deformed hooves, and swelling and lesions near the hoof joint similar to those seen in cattle (46). The wool of zinc-deficient sheep becomes loose and brittle and loses its crimp (46).

The zinc requirement of sheep and goats, like cattle, is lower when fed semipurified diets than under field conditions. Between 1965 and 1993, the minimum requirement for the growth of lambs under laboratory conditions ranged from <7 to 18 mg/kg (26). The current recommendations for sheep and goats vary with size and age (53). Based on information by the National Research Council (53), the calculated zinc requirements per kilogram of dry matter for lambs range from 21 to 38 mg and from 30 to 45 mg for ewes. The calculated requirements per kilogram of dry matter for goat kids range from 15 to 23 mg, from 26–48 mg for gestating does, and from 40 to 70 mg for lactating goats.

In 1985, McDowell (54) reported that 25 tropical countries had reported definite or probable zinc deficiencies in ruminants based on clinical signs, low serum concentrations, and/or low forage zinc concentrations. Methods were developed to combat these zinc deficiencies. Treating soils with zinc-containing fertilizers to increase crop and pasture yields was found to increase forage zinc concentrations to amounts that are adequate for grazing animals (26). Under conditions in which fertilizer application is not economical, such as extensive range conditions, salt-licks containing 1% to 2% zinc usually provide sufficient intake of zinc for grazing ruminants (26). A heavy intraruminal zinc pellet also has been developed that releases zinc over at least a 6-wk period in sheep (26). Zinc adequacy can be ensured for dairy and beef cattle fed rations that include grains by using zinc supplements such as zinc sulfate.

**Supranutritional and toxic intake of zinc in agriculture**

A review of the history of zinc in agriculture reveals that in the 50 y after the report describing zinc deficiency in swine, the importance of zinc nutrition for agriculturally important plants and animals under practical or farm conditions was well established. In addition, methods to overcome or prevent zinc deficiencies were described. However, persons chronicling zinc in agriculture need to be aware of a current development that may show that supranutritional zinc has a role in agriculturally important animals throughout the world.

In Europe, organic antibiotics as growth promoters are not allowed in feeds, and just recently, the FDA announced that it will ask livestock producers, drug companies, and veterinarians to curb the use of organic antibiotics to promote growth in food-producing animals in the United States. The basis for these restrictions is an effort to prevent the generation of drug-resistant bacteria or “superbugs” that are pathogenic and impossible to treat in humans. In Europe, high amounts of zinc are being used as a growth promoter (54). High intakes of zinc apparently inhibit growth and counteract the detrimental effects of bacteria (55,56) that can prevent optimal growth and performance of farm animals, especially poultry and swine.

The amount of zinc used to inhibit the detrimental effects of microorganisms has to consider the amount that would have a toxic effect on the animal dosed. The tolerance of supranutritional amounts of zinc depends both on species and feed composition. Swine and poultry have a greater tolerance to supranutritional zinc than cattle and sheep, which explains active testing of zinc as a growth promoter in the swine and poultry industry. The maximum tolerable intake for poultry and swine has been set at a 1000-mg/kg diet (55). However, since 2000, numerous experiments have demonstrated that a supplemental 1500- to 3000-mg/kg diet as zinc oxide stimulates the growth of weaned pigs (55). The maximum tolerance level for cattle has been set at a 500-mg/kg diet and at 300 mg/kg for sheep (55).

**Concluding statements**

The essentiality of zinc for plants and mammals was discovered >75 y ago. The first report of zinc deficiency in an agriculturally important animal (swine) that could occur...
under farm conditions was 47 y ago. Within 35 y of that report, nutrition researchers, including a large number who would become Fellows of the American Society for Nutrition, had presented a relatively complete picture of the importance of zinc nutrition in agriculture. The history of zinc in agriculture is an outstanding demonstration of the translation of research to practical application. Nonetheless, zinc deficiency in agriculturally important plants and animals still remains a problem worldwide, especially in areas with less developed agricultural practices.

Acknowledgments
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