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Iron Toxicity in Wetland Rice and the Role of Other Nutrients

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ABSTRACT

Iron (Fe) toxicity is a widespread nutrient disorder of wetland rice grown on acid sulfate soils, Ultisols, and sandy soils with a low cation exchange capacity, moderate to high acidity, and active Fe (easily reducible Fe) and low to moderately high in organic matter. Iron toxicity reduces rice yields by 12–100%, depending on the Fe tolerance of the genotype, intensity of Fe toxicity stress, and soil fertility status. Iron toxicity can be reduced by using Fe-tolerant rice genotypes and through soil, water, and nutrient management practices. This article critically assesses the recent literature on Fe toxicity, with emphasis on the role of other plant nutrients, in the occurrence of and tolerance to Fe toxicity in lowland rice and puts this information in perspective for future research needs. The article

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emphasizes the need for research to provide knowledge that would be used for increasing rice production on Fe-toxic wetlands on a sustainable basis by integration of genetic tolerance to Fe toxicity with soil, water, and nutrient management.

Key Words: Iron toxicity; Soil reduction and ferrous iron in soil solution; Plant nutrients; Rice tolerance for iron; Rice yield loss; Integrated approach to reduce iron toxicity.

INTRODUCTION

Iron toxicity is caused when a large amount of Fe(II) is mobilized in situ in soil solution or when interflow brings Fe(II) ions from upper slopes.[1–3] Iron toxicity occurs when the rice plant accumulates a toxic concentration of Fe in the leaves. The occurrence of Fe toxicity is associated with a high concentration of Fe(II) in soil solution.[4] High concentrations of Fe in soil solution also decrease the absorption by the rice plant of other plant nutrients, especially P and K.[3,5]

Iron toxicity symptoms vary with cultivars and are characterized by a reddish-brown, purple bronzing, yellow, or orange discoloration of the lower leaves. Typical Fe toxicity symptoms are generally manifested as tiny brown spots starting from the tips and spreading towards the bases of the lower leaves. The spots coalesce on the interveins of the leaves with progressing Fe toxicity. With increased Fe toxicity, the entire leaf looks purplish brown followed by drying of the leaves, which gives the plant a scorched appearance. The symptoms commonly develop at maximum tillering and heading stage of the rice plant, but may be observed at any growth stage. The roots of the plants affected by the disorder become scanty, coarse, short and blunted, and dark brown in color. With alleviation of the Fe toxicity the roots may slowly recover to the usual white color.

Iron toxicity occurs in acid Ultisols and Oxisols, and in acid sulfate soils that are rich in reducible Fe. The symptoms of oranging or bronzing observed in rice growing on strongly acid soils in Sri Lanka were attributed to Fe toxicity.[4] Since its first report in 1955,[4] Fe toxicity has been reported in several countries including China, India, Indonesia, Thailand, Malaysia, the Philippines, Sri Lanka, Vietnam, Brazil, Burundi, and Colombia.[3,6–14] The nutritional disorders of rice known as *Akagare Type I* in Japan[7] and *bronzing* in Sri Lanka[4,7] are attributed to Fe toxicity.[4,15] The nutritional disorder known as *Akiochi* in Korea[16] is also suspected to be associated with Fe toxicity. Iron toxicity has been

reported throughout the West African region in Benin, Ivory Coast, Burkina Faso, the Gambia, Guinea, Guinea-Bissau, Liberia, Nigeria, Niger, Senegal, Sierra Leone, and Togo.[2,17–26] In West Africa, Fe toxicity has been reported to reduce rice yields in wetlands by 12–100% depending on the intensity of toxicity and tolerance of the rice cultivar.[19–21,26,27] In West and Central Africa, Fe toxicity affects rice growth and yield on about 30% of the lowland swamp soils in rainfed and irrigated lowland areas.[24]

Cultural practices such as planting date, ridge planting, water management, and presubmergence of soil can be manipulated to reduce Fe toxicity in rice.[28–32] The most cost-effective approach is the use of Fe toxicity-tolerant rice cultivars.[8,21,33,34] Under extreme Fe toxicity conditions, a combination of tolerant cultivar and improved cultural practices may give the best results.[23,27,35]

Iron toxicity is a complex nutrient disorder and deficiencies of other plant nutrients, especially phosphorus (P), potassium (K), calcium (Ca), magnesium (Mg), and zinc (Zn), have been considered to affect its incidence in rice.[12–14,18,36–40] Rice grown in acid sulfate soils may involve deficiencies of several nutrient elements, especially P, Ca, Mg, and Zn.[22,41–45] The deficiency of other nutrients may play an important role not only in the management of Fe toxicity, but also in the Fe toxicity tolerance of a cultivar. The objective of this article is to critically review the recent literature on Fe toxicity, with emphasis on the role of plant nutrients, in the occurrence of and tolerance to Fe toxicity in lowland rice and put this information in perspective for future research needs. The ultimate goal is to provide knowledge, by stimulating research that would be used for increasing rice production in Fe toxic wetlands on a sustainable basis by integration of genetic tolerance with soil and nutrient management.

OCCURRENCE OF IRON TOXICITY

General Conditions

Ponnamperuma[46] listed seven important criteria for the occurrence of Fe toxicity in rice grown in submerged soils:

- pH of the dry soil, less than 6.5.
- High reserve soil acidity.
- High reactivity and content of Fe(III) oxide hydrates.

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- Soil temperature, low temperature brings late but high and persistent concentrations of water soluble Fe.
- Salt content, salts increase Fe concentration in soil solution.
- Low percolation rate in the soil.
- High interflow of Fe from adjacent areas.

Iron toxicity related to interflow of Fe is especially important in West Africa, where land has undulating topography and rice is grown along the toposequence rather than in the rice paddies.[47] Under these conditions, the hydrology and the interflow of Fe from upper slopes (with Fe-rich lateritic soils) to foot-slopes and to valley bottoms in the inland valleys is a common feature, and is invariably the cause of Fe toxicity occurrence in rice grown in the valley bottoms and at times in the hydromorphic (transition between upland and the inland swamp) zone of the continuum.

Iron toxicity may also be a hazard for inland swamp (wetland) rice soils in which the main nutrient disorders for dry land crops are manganese (Mn) and aluminum (Al) toxicities, and the deficiencies of macronutrient elements.[46,48]

In the field, Fe toxicity often occurs on infertile, light-textured soils in valleys or on foot-slopes where the adjacent upper slope soils have laterite horizons. According to van Breemen and Moormann[9] such conditions are common in the low-country wet zone of Sri Lanka, in Kerala and Orissa states in India, and in many countries in West Africa including the Ivory Coast, Liberia, Nigeria, Senegal, and Sierra Leone. Iron toxicity occurs in acid Ultisols and Oxisols, and in acid sulfate soils. Iron toxicity is a major nutrient disorder of lowland rice on young strongly acid sulfate soils. Aluminum toxicity is a major problem, in addition to Fe toxicity, for wetland rice production in acid sulfate soils, which do not experience the usual increase in pH[1,49] during submergence due to lack of soil reduction.[50]

The most dramatic chemical change that occurs when a soil is submerged in water and undergoes reduction is that Fe(III) oxide hydrates are reduced to Fe(II) compounds. Patrick and Reddy[51] reported that the amount of Fe that can undergo reduction usually exceeds the total amount of other redox elements by a factor of 10 or more. This occurs in a submerged soil when the redox potential is less than 180–150 mV.[51,52] As a result of soil reduction the soil color changes from brown to gray. The concentration of water-soluble Fe, which at submergence rarely exceeds 0.1 mg L^{-1}, may increase to a few hundred milligrams per liter within a few weeks following submergence.[1,53,54] In young acid sulfate soils the peak values of Fe in soil solution may be as high as a few thousand milligrams per liter.[41,54,55]

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Production of other reduction end products such as sulfide in submerged soils under highly reduced conditions may also contribute to the occurrence of Fe toxicity. It is known that respiratory inhibitors such as sulfide lessen the oxidizing capacity of rice roots and may thus increase the susceptibility of the rice plant to Fe toxicity.[56] The susceptibility of rice to Fe toxicity is also greatly influenced by the physiological status of the plants.[15,41]

Level of Iron in the Growth Media

The reported levels of Fe in culture solutions that cause toxicity vary from as low as 10 mg Fe L^{-1} up to 500 mg Fe L^{-1} or higher.[36,57] The wide range in the reported Fe toxic levels may be due to differences in the form and source of Fe used, varietal tolerance, the concentrations of other nutrients, temperature, and solar radiation.[57,58] Also, as van Breemen and Moormann[9] pointed out, during most solution culture studies no precautions were taken to prevent the oxidation of soluble Fe(II) to insoluble ferric hydroxides and the actual concentrations of Fe(II) might have been lower than those reported. For example, in a recent study of Fe toxicity in rice in a hydroponic system, Bode et al.[57] reported that 5–10% of the Fe(II) in the nutrient solution was oxidized to Fe(III) in experiments lasting up to 3–4 weeks. Sources of Fe such as Fe chelates [Fe(III) EDTA] which keep Fe in solution in the face of root action to precipitate Fe by effecting pH and redox potential changes, may induce Fe toxicity symptoms at lower concentrations compared to Fe sources without chelates (ferrous sulfate and ferrous bicarbonate). Another problem in pot experiments with submerged soils is that the concentration of Fe(II) is not always distributed homogenously. The concentration of Fe(II) in solution may be lower in the surface layers where most roots are generally concentrated in small pots.[9] It has been observed that using the same soil, the intensity of Fe toxicity to a rice cultivar is generally lower in greenhouse pots than under field conditions.[59] The concentration of Fe(II) in the soil solution that develops Fe toxicity symptoms in rice seems to vary with the pH of the soil solution. The critical limit was about 100 mg L^{-1} at pH 3.7 and 300 mg L^{-1} or higher at pH 5.0.[15]

A study of the kinetics of Fe(II) release under flooded condition in an iron-toxic Ultisol (pH water, 5.2; pH KCl, 3.9; organic C, 12 g kg^{-1}; DTPA extractable Fe, 325 mg kg^{-1}) from the humid savanna zone in Korhogo, Ivory Coast (West Africa) showed that the concentrations of Fe(II) in soil solution varied from 50 to 150 mg L^{-1} during 3–10 weeks after flooding in greenhouse pots.[53] In the field, rice plants growing on

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the soil showed severe Fe toxicity symptoms. The results suggest that Fe(II) concentrations in soil solution, ranging from 50–150 mg L^{-1}, cause Fe toxicity to rice on this site in Korhogo, Ivory Coast. Furthermore, on this gently sloping site, the contribution of ferrous Fe through interflow is low and less important than the release of ferrous Fe in situ in causing Fe toxicity in wetland rice.[34,35]

The occurrence of Fe toxicity in lowland rice can be influenced by reduction products such as sulfides and organic acids. The critical concentration of Fe(II) for the development of Fe toxicity symptoms varies from as low as 10 to as high as 500 mg L^{-1}, depending largely on the nutrient status of the plant and the presence of reduction products. In the absence of harmful levels of reduction products and an adequate supply of plant nutrients, the rice plant suffers from Fe toxicity at Fe(II) concentrations higher than 300–500 mg L^{-1}.[9,57]

Because the severity of Fe toxicity depends on several environmental factors, it is somewhat difficult to correlate the Fe status of the rice plant with Fe content in the growing medium. The reported critical concentrations of Fe in the soil solution that produce Fe toxicity in rice varies greatly and there seems to be no simple relationship between Fe concentration and the occurrence of Fe toxicity.

SOIL REDUCTION AND IRON TOXICITY

Soils on which rice is grown can experience a range of redox potential. Data on the range of redox potentials encountered in soils ranging from well drained to flooded conditions are summarized in Table 1, and can serve as a useful guideline for classifying soil reduction under diverse soil conditions.[51,52] Soil reduction is a process in the submerged soils which greatly influences Fe toxicity in wetland rice:

- Soil reduction mobilizes Fe(II) in soil solution. The concentration of Fe(II) is negligible in nonreduced soils.
- Some of the reduction products, such as dissolved sulfides, may increase the susceptibility of the rice plant to Fe toxicity.
- Production of reduced organic substances may interfere with Fe toxicity through their influence on rice plant root growth.

The influence of flooding on soil reduction and Fe(II) production have been extensively reviewed.[1,49,53,60,61] Those aspects of soil reduction that affect the occurrence of Fe toxicity by influencing the kinetics in soil

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Table 1. The range of oxidation-reduction potentials usually encountered in well drained and waterlogged soils.[51]

Soil moisture condition	Redox potential (mV)
Well-drained (aerated)	+700 to +500
Moderately reduced	+400 to +200
Reduced	+100 to −100
Highly reduced	−100 to −300

Table 2. Approximate redox potentials at which the main oxidized components in flooded soils become unstable.[51,52]

Reaction	Redox potential (mV)
O_2-H_2O	+380 to +320
NO_3-N_2, Mn^{4+}-Mn^{2+}	+280 to +220
Fe^{3+}-Fe^{2+}	+180 to +150
SO_4^{2-}-S^{2-}	−120 to −180
CO_2-CH_4	−200 to −280

solution of Fe(II), dissolved sulfides, and organic reduction products are dealt with in this discussion.

The concentration of Fe(II) in soil increases following flooding due to reduction of Fe(III) oxide by bacteria oxidizing organic matter. The reduction of Fe(III) to Fe(II) takes place at a redox potential of 180–150 mV. A rapid increase in Fe(II) following flooding is favored by low initial soil pH, a sustained supply of organic matter, the presence of easily reducible Fe, a high fertility status of the soil and the absence of compounds with a higher oxidation state than Fe(III) oxide, especially oxygen, Mn(III, IV) oxide, and nitrate (Table 2) in the soil.[1,51–53]

The reduction of Fe(III) to Fe(II) can be illustrated by the following equation:

$$Fe_2O_3 + 1/2CH_2O + 4H^+ = 2Fe2^+ + 5/2H_2O + 1/2CO_2 \quad (1)$$

In the above equation, Fe oxide serves as the source of reducible Fe and organic matter (CH_2O) serves as the electron donor. The forms of Fe important in redox reactions are largely mixtures of X-ray amorphous materials and goethite of variable but low water solubility. Amorphous

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Fe oxide is more easily reducible by bacterial activity than crystalline Fe oxide.[62–64]

The concentration of water-soluble Fe(II) is greater at lower redox potential. The average concentrations of water-soluble Fe are highest in acid sulfate soils with high content of reactive Fe(III) oxides. The concentration of Fe(II) in these soils may be as high as 5000 mg L^{-1}.[65] In general, acid soils high in organic matter and reducible (active) Fe attain high concentrations of Fe(II) during the initial period of submergence. The solution concentration then decreases roughly exponentially to levels of 50–100 mg L^{-1} that persist for several months. On the other hand, soils high in organic matter but low in active Fe attain high concentrations of Fe(II) that persists for several months.[1] Application of organic matter enhances the reduction of Fe in soils but the prevailing soil pH will determine the effect of organic matter on the concentration of water-soluble Fe in the soil. The presence of nitrate or low temperature retards the release of water soluble Fe, but may not prevent a delayed increase in solution Fe concentration.

The increase in concentration of water-soluble Fe following flooding of soils can be described for most mineral soils by the equation[65]:

$$\mathrm{Eh} = 1.06 - 0.059 \log \mathrm{Fe}^{2+} - 0.177\ \mathrm{pH} \quad \text{or} \tag{2}$$

$$\mathrm{pE} = 17.87 + \mathrm{pFe}^{2+} - 3\ \mathrm{pH} \tag{3}$$

According to Eq. (3), at a pE = −0.73 (Eh = −42 mV), pH = 7, and ionic strength of soil solution = 0.03 moles L^{-1}, at 25°C, the concentration of water-soluble Fe(II) is 400 mg L^{-1}—a toxic concentration for the occurrence of Fe toxicity. On the other hand, when the Eh is high, for example at pE = 2.0 (Eh = 118 mV), and at pH 7, the concentration of water-soluble Fe(II) is only 0.7 mg L^{-1}—a deficient concentration for the occurrence of iron deficiency. Thus the increase in solubility of Fe following soil reduction benefits rice. But in soils rich in reducible Fe and organic matter, soil reduction mobilizes large amounts of water-soluble Fe and is the apparent cause of Fe toxicity to rice.

Narteh and Sahrawat[53] studied the effects of flooding on changes in chemical and electrochemical properties of 15 rice soils from West Africa. They found that 4 weeks after flooding, the soil solution Eh (mV) can be predicted from the concentration of Fe(II) (mg L^{-1}) in soil solution and soil solution pH:

$$\mathrm{Eh} = 409 - 4.09 \log \mathrm{Fe(II)} - 59\ \mathrm{pH}; \quad R^2 = 0.99 \tag{4}$$

Moreover, the changes in soil solution pH corresponded to changes in soil solution Eh. The stability in Eh-pH relationship was recorded 4

weeks after flooding of the soils and the relationship was described by the following equation[53]:

$$\Delta \mathrm{Eh} = -16 - 48\ \Delta\ \mathrm{pH}; \quad R^2 = 0.84 \tag{5}$$

Ponnamperuma[48] reported that the peak values of water soluble Fe for over 100 wetland rice soils ranged from 6600 mg Fe L^{-1} at pH 5.67 for an acid sulfate soil to 0.07 mg Fe L^{-1} at pH 7.25 for a calcareous soil low in organic matter. The Fe(II) hydroxide potential ($pH - 1/2\ pFe^{2+}$) appears to be a characteristic of a given soil and has been to be reported constant over long periods of submergence.[46,48]

According to Patrick and Reddy[51] the effect of redox potential and pH on the final chemical equilibria of a flooded soil is much greater when these parameters are acting together than when each is acting alone. Microbial reduction processes such as Fe(III) reduction to Fe(II) are favored by a near neutral pH. Indeed, Moraghan and Patrick[66] found that Fe(III) reduction in a submerged soil at controlled soil pH 7 was faster than at controlled pH 5. However, the equilibrium concentration of Fe(II) in soil solution was higher at pH 5 than at 7. This was attributed to the fact that reduced Fe produced at near neutral pH underwent secondary reactions that resulted in the precipitation of Fe as various oxides, hydroxides, and carbonates. On the other hand, at low soil pH the reduced cations remained in solution or on the exchange complex.[67,68] Soil pH influences the accumulation of extractable Fe in a submerged soil.[66] In addition, the interactive effects at various redox potential-pH combinations influence the release of labeled Fe from strengite.[67]

Because acidity is consumed during reduction of $Fe(OH)_3$ to Fe(II) (see Eq. (1)), this causes an increase in soil pH. Normally soil pH values in the range 6.4–7.0 are attained within 2–5 weeks after flooding.[1,49,53] However, in some acid sulfate soils low in Fe(III) oxides relative to soil acidity, there is a lack of pH increase brought about by soil reduction following flooding.[50]

Ponnamperuma[1] showed that after a peak in water-soluble Fe, the activity of Fe(II) in most rice soils is related to pH. The peak in water-soluble Fe appears to be controlled by $Fe(OH)_3$-Fe^{2+} system, causing precipitation of Fe(II) when the pH reaches a threshold level. However, according to Moore and Patrick[43] the form of Fe that usually precipitates after reaching a peak is $FeCO_3$, unless sulfides are present.

van Breemen and Moormann[9] summarized the changes in pH and dissolved Fe following flooding of young acid sulfate soils from Vietnam and the Philippines (pH 3.5–3.8), one older acid sulfate soil from Thailand, and three other acid soils (pH 4.0–4.8) high in active Fe and low in Mn. They found that in the acid soils, the pH increased rapidly as

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the amounts of water-soluble Fe(II) increased during the first four weeks after flooding. The pH continued to increase when Fe rose to peak values and remained practically constant at the 6.4–6.8 pH range as Fe concentrations decreased sharply. The acid sulfate soils showed a slower rate of increase in pH and a more rapid increase in Fe(II) in solution in the first week of flooding. Thus the pH initially remained low despite the release of large quantities of Fe(II). The maximum concentration of Fe in acid sulfate soils can often be maintained for prolonged periods until sulfide, produced from sulfate reduction, lowers the concentration of water-soluble Fe by precipitation as Fe(II) sulfide. However, sulfate reduction is slow when the pH remains below 5,[69] and consequently the decrease in the concentration of Fe(II) is also slow.

Temperature has a marked effect on the kinetics of release of water soluble Fe. Cho and Ponnamperuma[70] studied the effect of temperature (15°, 20°, 30°, 38°C) maintained constant or fluctuating between low and high range, on the kinetics of release of water soluble Fe in three Philippine soils with a range in pH, organic matter (O.M.) and free Fe (Maahas clay: pH 6.6; O.M. 2%; free Fe 2.18%, Casiguran sandy loam: pH 4.9; O.M. 5.3%; free Fe 0.35%, Luisiana clay: pH 4.8; O.M. 3.2%; free Fe 3.30%). Temperatures greater than 30°C hastened and sharpened the peak of water soluble Fe. On the other hand, low temperatures broadened the peak of water soluble Fe. The results show that low temperature (20°C) may slowly mobilize Fe(II) in solution but high levels of Fe are finally reached and persist for long periods of time.[70]

Sahrawat and Singh[71] observed that on an iron-toxic site in the savanna zone of Ivory Coast, Fe toxicity intensity in irrigated rice was higher in the dry than wet season due to prevailing higher atmospheric temperature and evapotranspiration. Consequently, Fe toxicity scores based on the extent of Fe toxicity symptoms on the foliage of rice plants were higher and yields were lower in the dry than in the wet season for the 12 rice cultivars evaluated.

Salinity level in the soil has a significant effect on the kinetics of Fe(II) release in submerged soils. High salinity favors the production of Fe(II) in soil solution.[72] High concentrations of salts such as sodium chloride or magnesium chloride in soil can aggravate Fe toxicity by decreasing the oxidizing power and Fe-retaining power of the rice roots.[15]

SULFIDE PRODUCTION

Within a few weeks after submergence, the concentration of water-soluble sulfate in normal and near neutral pH soils becomes extremely

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low. However, the reduction of sulfate to sulfide is slower in acid soils, especially those with pH lower than 5.[1,69] The disappearance of sulfate within four to six weeks after submergence is due to its reduction to sulfide (H_2S). Patrick[52] and Patrick and Reddy[51] established that sulfate is unstable in soil suspensions maintained at redox potential of −120 to −180 mV (Table 2).

The reduction of sulfate to sulfide has practical implications for the fertility of submerged soils and rice growth. Sulfide production can influence Fe toxicity at least in two ways. First, sulfide produced in highly reduced soils may combine with the Fe(II) in solution and precipitate by formation of FeS and may decrease the amount of Fe in solution and partially alleviate Fe toxicity. Secondly, a large body of literature suggests that sulfide inhibits respiration and the oxidizing power of rice roots and may retard the uptake of plant nutrients and rice growth.[15] In the reduced soil the bulk of sulfide is present in the solid phase as FeS, but even low concentrations of dissolved sulfide ($<0.1\,mg\,L^{-1}$) may be directly harmful to rice.[73] Small amounts of dissolved sulfide may also aggravate Fe toxicity in rice.[56,74] Reasonably high levels of both sulfide and Fe(II) can occur in the soil solution even at near neutral pH values if carbon dioxide is produced rapidly.[15] Tanaka et al.[56] proposed that under the conditions described above, Fe toxicity is likely to occur more frequently because sulfide destroys the oxidizing power of the roots and more Fe enters the rice plant. Blomfield[75] also provided evidence to show that under highly reduced soil conditions much of the sulfur in the soil was lost as hydrogen sulfide gas even in the presence of excess Fe(III), or in the presence of dissolved Fe(II) in the solution.

Tanaka et al.[36,56] reported that Fe absorption by the roots of rice plants from a culture solution containing high levels of Fe was enhanced considerably when the roots were trimmed at the tips or damaged by hydrogen sulfide.

PRODUCTION OF REDUCING ORGANIC SUBSTANCES

Low redox potential in submerged soils generates toxic organic reduction products as a result of anaerobic decomposition of the organic matter. A low redox potential alters the course of organic matter decomposition from the production of carbon dioxide, nitrate, and sulfate at high redox potential to the production of methane, ammonium, mercaptans, organic sulfides, and hydrogen sulfide through a large number of intermediary metabolites.[1,15,76] Due to anaerobic metabolism

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of organic matter, a range of organic substances are formed. Some (the lower fatty acids) are toxic to the rice plant. Also, some of the dissolved reduction products such as phenolic compounds that are related to fulvic acids can reduce Fe(III) to Fe(II) and may also aggravate Fe toxicity by hampering the oxidizing capacity of the rice roots. Indeed, some of the reduction products occur in small concentrations but can inhibit rice growth. For example, organic acids are reportedly toxic to the rice plant at concentrations of 10^{-2} to 10^{-3} M.[3,15]

IRON TOXICITY IN RELATION TO SOIL CHARACTERISTICS

Several studies made in Asia and Africa show that Fe toxicity is a major nutrient disorder of rice grown on acid sulfate soils, Ultisols and sandy soils with a low CEC, moderate to high in acidity and active Fe, and low to moderately high in organic matter.[9,13,23,28,40,43,44,46,48,77,78] van Breemen and Moormann[9] made an analysis of soil data in relation to Fe toxicity in Asian soils and suggested that Fe toxicity is common in the young acid sulfate soils (Sulfaquepts) but is rare on the older, more deeply developed acid sulfate soils (Sulfic Tropaquepts) which do not produce high levels of Fe(II) upon submergence. Iron toxicity in soils other than acid sulfate soils is often associated with other nutrient disorders.

Benckiser et al.[77] summarized the physicochemical properties of 25 mineral (nonacid sulfate) Fe-toxic soils from Brunei, China, Indonesia, Liberia, Philippines, and Sri Lanka. The data (Table 3) show an association of soil characteristics, especially those related to soil fertility, with Fe toxicity.

At a very low pH (<4.0), the physiological activity of the rice plant is decreased drastically and this weakens the root functions, which allow rice plants to tolerate high concentration of Fe in soil solution.[15,41] Additionally, under conditions described above (low pH) lower concentration of Fe in soil solution may cause Fe toxicity.

In a recent study of Fe toxicity in lowland rice in the highland swamps of Burundi, Genon et al.[13,40] reported that the exchangeable Fe fraction in the flooded soils was related to Fe toxicity in rice. Organic matter content in soils plays an important role in mobilizing Fe, cations such as K, Ca, Mg, Al, and Mn, which in turn influence the exchangeable Fe fraction [Fe/(Ca + Mg + Fe + Mn + Al)] which was found to be related to Fe toxicity. The sum of exchangeable cations was found to increase with organic C more than did exchangeable Fe, and this resulted

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Table 3. Range and mean in the physicochemical characteristics of 25 mineral iron-toxic soils from Brunei, China, Indonesia, Liberia, Philippines, and Sri Lanka.[77]

Soil characteristics	Range	Mean	Critical level
Texture	Loamy sand to clay	—	—
Total N (%)	0.07–0.80	0.20	0.20
Organic C (%)	0.7–7.4	2.5	[a]—
pH (water)	4.3–7.4	5.2	—
Total Fe (%)	0.4–11.2	3.6	—
Oxalate extr. Fe (%)	0.3–1.5	0.8	—
Total Mn (%)	0.002–0.90	0.09	—
Extr. Zn, 0.01 N HCl (mg kg^{-1})	0.3–7.1	2.4	1–2
CEC (me $100\,g^{-1}$)	1.8–31.6	11.7	20
Exch. cations (me $100\,g^{-1}$)			
K	0.01–0.16	0.08	0.20
Ca	0.02–21.8	3.6	10
Mg	0.09–7.7	2.6	2–5
Olsen P (mg kg^{-1})	0.5–8.0	4.8	10
Bray 1 P (mg kg^{-1})	4.0–36.0	14.9	26

[a]— = Not determined.

in lower exchangeable Fe fractions in most organic soils. Moreover, organic soils have large CEC and their adsorbed Fe fraction remains relatively low. It was concluded that peaty soils (>25% organic C) exhibited a lower Fe toxicity hazard than the mineral soils with intermediate organic C content (10–25%).[40]

OTHER FACTORS INVOLVED IN IRON TOXICITY

In addition to the factors discussed above, several plant and growth environment related factors affect the occurrence of Fe toxicity in rice. For example, it has been shown that the age of the rice plant affects its tolerance to high levels of Fe in soil solution and that the tolerance is lower at the early stage than at later growth stages. This may be due to the fact that the Fe-excluding power of the roots of young rice plants is extremely low.[3,15] However, the young rice plant has higher oxidizing power and Fe-retaining power than at later growth stages. Tanaka et al.[36] reported that the concentration of Fe in the culture solution that caused Fe toxicity was lower at the vegetative growth stage of rice than at

later growth stages. A concentration as low as 75 mg Fe L^{-1} was enough to cause Fe toxicity in the rice plants at the vegetative growth stage.

Similarly, several factors associated with the chemical environment of the growth media such as pH, accumulation of respiratory inhibitors such as hydrogen sulfide, organic acids, and other reduction products in soil solution may make the rice plant more susceptible to Fe toxicity.[15] Presence of salts such as chlorides of sodium and magnesium can also aggravate Fe toxicity by decreasing the oxidizing power of the rice roots.[3] These, and other factors that are not clearly defined, affect the occurrence of Fe toxicity in rice plants in a complex manner that cannot be resolved solely by the Fe concentration of the growth media.

RICE TOLERANCE FOR HIGH IRON CONCENTRATIONS

The physiological status of a rice plant growing under submerged soil conditions greatly modifies its ability to tolerate high concentrations of Fe. Tadano[79] suggested that three functions of rice roots were involved in counteracting Fe toxicity:

1. Oxidation of Fe in the rhizosphere, which helps to keep Fe concentration low in the growth media.
2. Iron-excluding power of the roots, which excludes Fe at the root surface and thus prevents Fe from entering the root.
3. Iron retaining power of the roots, which retains Fe in the root tissue and thus decreases the translocation of Fe from the root to the shoot.

Rice roots diffuse molecular oxygen into the root medium through air chambers and aerenchyma in the leaves, stems, nodes, and roots, which makes the rhizosphere more oxidative than the bulk growing media. Ferrous iron in soil solution is also oxidized to Fe(III), which can be seen as deposits on the surface of the rice roots. The oxidizing power of the rice roots is greater at the growing points and at the elongating parts of the roots than at the basal parts.[3]

Under controlled conditions in pots, it was observed that the redox potential of soil solution was higher in pots with plants than without. The increase in redox potential was more prominent when plants were supplied with K than when K was not added. Thus it is concluded that rice roots maintain supplying rice plants with nutrients such as K[79] can further increase high redox potentials.

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Absorption of Fe by the rice plant is not related to the absorption of water when the concentration of Fe in the culture solution is low. On the other hand, when the concentration of Fe is high, the Fe content in the plant tops increases proportionately with water absorption and the total amount of Fe absorbed also increases.[15] Thus it is suggested that the absorption of Fe by mass flow is an important mechanism when Fe concentration is high in the growing medium.

Tadano[79] reported that the Fe-excluding power of a healthy rice plant was 87%, implying that 87% of the iron that had reached the root surface by mass flow was excluded. The Fe-excluding power of the rice roots was markedly decreased by respiratory inhibitors such as potassium cyanide (KCN) and sodium azide (NaN_3).

Similarly, the ability of rice roots to retain Fe by reduced translocation of absorbed Fe from root to shoot can affect plant tolerance to Fe. The Fe-retaining power of the rice roots is inversely related to the translocation percentage, i.e., the amount of Fe translocated to the shoot relative to the total amount of Fe absorbed by the plant. Salts such as sodium chloride and respiratory inhibitors decrease the Fe-retaining power of the root.[79]

The nutritional status of the plant, especially with regard to Ca, Mg, K, Mn, and P, greatly modifies the rice roots' Fe-excluding and Fe-retaining power. The role of K in Fe toxicity has been suggested to be very important because K is not only involved in exclusion of Fe, but also in its translocation from roots to shoots.[39,79]

Another mechanism is involved in which the rice plant is able to tolerate a high concentration of Fe in the tissue. For example, Jayawardena et al.[80] tested 17 tropical rice varieties for their tolerance to Fe toxicity, Fe content in plant tissue, and Fe-oxidizing power of roots. They found that the majority of the tolerant varieties contained a high concentration of Fe in the plant tissues and this led them to suggest that the varietal tolerance for Fe toxicity is a degree of tolerance for excess Fe rather than a mechanism of resisting the entry of Fe into roots.

Based on this brief discussion on the rice plant's tolerance to high concentrations of Fe, several mechanisms related to the physiology of the rice plant and its nutritional status may be operative and there is no simple, definitive explanation available. Rice plants counteract Fe toxicity by preventing or avoiding excess Fe(II) uptake at the roots and by tolerance of the plant tissue. Evidently, a high concentration of Fe in the plant does not automatically mean that it is under Fe toxicity stress. Perhaps what is more important is whether Fe enters the cell or not.[81,82] It has been suggested that when Fe uptake by plants is relatively slow, the cell wall and associated polysaccharides are able to

exclude Fe(II) from the symplast. Clearly, we do not understand the reaction of Fe(II) in plant tissue and further studies are needed to clarify the Fe toxicity tolerance mechanism.[82]

ROLE OF OTHER NUTRIENTS IN OCCURRENCE OF IRON TOXICITY

Rice plants are able to grow normally when the concentration of Fe in the soil solution exceeds $300\,mg\,L^{-1}$. However, Fe toxicity of rice has been frequently reported to occur in soils where the concentration of Fe in the soil solution is lower than the "critical" limit of $300\,mg\,Fe\,L^{-1}$.[9,15,54] Such differential responses of the rice plant to Fe concentration in soil solution can be attributed to factors such as age and nutritional status of the plant and chemical environment of the growth media. All these factors, except the role of plant nutrients, were covered in the previous sections.

Potassium content of rice plants exhibiting physiological diseases such as Akagare Type 1, bronzing and Akiochi (all attributed or linked to Fe toxicity) is often low (for review see Yoshida[3] and Tadano and Yoshida[15]). These studies established that the nutritional status of the rice plant affected plant tolerance to Fe. Differential tolerance to excess Fe can be due to a deficiency of the nutrient or can be through the effect of the nutrient on the rice plant's ability to exclude Fe. For example, deficiencies of K, Ca, Mg, P, and Mn are known to weaken the rice roots' power to exclude Fe.[3] Deficiencies of K, Ca, Mg, Mn, or silicon (Si) decrease the rice roots' ability to retain Fe. The ability to exclude or retain Fe in the rice plant deficient in K, P, Mg, Mn, or Si makes such a plant more susceptible to Fe toxicity than a healthy plant well supplied with these nutrients.

Deficiencies, especially of Ca, Mg, and Mn, are generally not observed on lowland rice. Phosphorus and K deficiencies deserve special attention. Results from a pot experiment showed that the rice plant deficient in K had a high concentration of Fe and severe symptoms of Fe toxicity.[15] In a recent study of Fe toxicity in central and southern Nigeria, Yamauchi[39] observed that K application reduced the severity of bronzing and increased dry matter production of rice plants in the field. The concentration and accumulation of K in the rice shoots increased when the bronzing severity decreased and the concentration of Fe was decreased, apparently by the dilution effect caused by increased dry matter production.

Howeler[38] observed that the oranging disease of rice grown in flooded Oxisols of Colombia was not a direct Fe toxicity, but was due to a nutritional deficiency, mainly of P and Mg in plants caused by relatively high levels of Fe in the soil solution. High levels of Fe in solution also inhibited the formation of new active roots. The coating of existing roots by Fe oxide further reduced their nutrient absorption capacity. The oranging symptoms on the rice plants were quite different from bronzing symptoms and occurred at rather low levels of Fe in soil solution. Bronzing, on the other hand, was a result of direct Fe toxicity resulting solely from high levels of Fe in soil solution.

A survey of various Fe-toxic soils in Asia indicated that all soils and plants were deficient in P and K, mostly in combination with insufficient amounts of Ca, Mg, and/or Zn.[18,77,78] This led to the hypothesis that Fe toxicity of rice grown on acidic mineral soils or acid sulfate soils is caused by a multiple nutritional stress. However, based on available information Fe toxicity cannot be eliminated entirely by improving the nutritional status of the plant.

Moore and Patrick[83] from a study of 132 flooded acid sulfate soils from the Central Plains region of Thailand reported that Fe uptake in the rice plant was correlated to Fe(II) activity. A better relationship was found between Fe uptake and the divalent charge fraction in soil solution attributed to Fe(II). Sylla[22] conducted field experiments on acid sulfate soils in the Gambia, the Casamance, and the Great Scarcies river basins in West Africa and found that the molar fraction Fe to (Ca + Mg) in the flag leaves of the rice plant was better correlated to Fe toxicity than the absolute concentration of Fe. These results support earlier work that implicated P, K, Ca, Mg, and Zn in the occurrence of Fe toxicity in acid sulfate soils.[18,77,84] Acid sulfate soils may involve deficiencies of several nutrient elements, especially, P, Ca, Mg, and Zn and Fe toxicity may be complicated by the deficiencies of these plant nutrients.

The toxic conditions associated with oranging symptoms of rice in a flooded Oxisol in Sumatra, Indonesia were attributed to Fe-induced, Mn-induced, and Al-induced deficiency of P, K, Ca, and Mg.[12] In a detailed study of 45 Oxisols under controlled conditions, it was found that Eh of the soils decreased sharply from an average value of +460 to −217 mV following 60 days of flooding. The soil pH increased from 5.2 to 6.6 and the concentrations of sodium acetate extractable Fe, Mn, Zn, Cu, molybdenum (Mo), Mg, Ca, K, and P, but not Al, increased markedly. Their water-soluble form, except Fe, decreased slightly following 60 days of flooding. Leaf tissue analyses of the rice plant showed that 13, 51, and 58% of the rice plant samples contained potentially toxic level of Mn, Fe, and Al, respectively (the assumed

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toxicity levels were 2500 mg kg^{-1} for Mn, 300 for Fe, and 300 for Al). Thirteen, 16, 2, and 3% of the leaf tissue also contained potentially deficient levels of P, K, Ca, and Mg, respectively. It was indicated that the oranging symptom in the rice leaf tissue appeared to be due to indirect toxicity of Fe, Mn, and Al, or in other words, due to deficiency of P, K, Ca, and Mg induced by Fe, Mn, and Al. These results are in accord with those reported earlier by Howeler[38] who reported that the oranging disease of rice grown in Oxisols from Colombia was due to a Fe-induced deficiency of P, K, Ca, and Mg.

The effects of plant nutrients on Fe toxicity may be through alleviation of the deficiency of other nutrient(s) and/or through their effects that influence the rice roots' ability to exclude or retain Fe.[3,15] It has been shown that deficiencies of Ca, Mg, P, and Mn weaken the Fe-excluding power of rice roots and those of K, Ca, Mg, Mn, or Si decrease the rice roots' Fe-retaining power. Thus the role of other nutrients may manifest in a combination of influences relating to the function of roots.[15]

Sahrawat[59] conducted a field experiment to determine the effect of Fe toxicity on elemental composition of Fe tolerant (CK4) and susceptible (Bouake 189) lowland rice varieties with and without application of N, P, K, and Zn. Plant samples were analyzed for macro- and micronutrient elements 30 and 60 days after transplanting rice seedlings. The results showed that there were no differences in elemental composition of the plant samples except for Fe. All other nutrient element concentrations were adequate in the plant tissue. Both Fe-tolerant and susceptible varieties had a high Fe concentration, well above the critical limit (300 mg Fe kg^{-1} plant dry wt). These results along with other results on the elemental composition of rice plant samples collected from several wetland swamp soils with Fe toxicity in West Africa suggest that "real" Fe toxicity is a single nutrient (Fe) toxicity and not a multiple nutrient deficiency stress.[59]

It would appear from the discussion that there could be at least two types of Fe toxicity of the rice plant. Firstly, in some situations Fe toxicity type symptoms may be caused by the deficiency of other nutrients such as P, K, Ca, Mg, and Zn. The plant tissue may or may not accumulate toxic concentrations of Fe. The deficiency of other nutrients could be inherent or induced by high concentrations of Fe and or Al. This is induced or "pseudo" Fe toxicity. In the second situation, the toxicity symptom in the plant is caused by toxic concentrations of Fe without any apparent deficiency of other plant nutrients. This is true Fe toxicity. However, it is possible that in the case of true Fe toxicity, a nutrient imbalance may be caused by a high concentration of Fe in the growing

medium. Undoubtedly, the management of these two types of Fe toxicities would require different management strategies, which are dealt in the next section.

ROLE OF OTHER NUTRIENTS IN THE MANAGEMENT OF IRON TOXICITY

Iron toxicity is a nutrient disorder associated with a high concentration of Fe in soil solution. A high concentration of Fe in soil solution can cause a nutrient imbalance in the growing medium, especially through antagonistic effects on the uptake of nutrients such as Zn and Mn. It has been reported that reduction in Zn uptake in the rice plant due to increased availability of Fe is more evident in soils that are marginal in Zn than in soils that have normal Zn levels. The antagonistic effect on Zn is generally more pronounced during the initial phase of flooding of the soil.[85] It has also been reported that an excess of Fe in soil solution reduced K uptake and the application of K reversed the trend and decreased the level of Fe in the rice plant. In a recent study of 45 Oxisol soils from Indonesia, Jugsujinda and Patrick[12] reported that high concentrations of Fe, Mn, and Al induced deficiencies of K, P, Ca, and Mg that caused oranging symptoms in the leaves of the rice plant. However, there was no indication that Fe interfered with the uptake of Zn. The supply of Zn probably was adequate.

It is evident that a high concentration of Fe in soil solution may cause nutrient imbalances in the growing rice plant. However, it is not clear whether the nutrient imbalance observed in the plant tissue is the cause or the result of excess Fe. The nutrients that are most affected include P, K, and Zn. The antagonistic effect of excess Fe on Zn uptake is especially crucial in soils low or marginal in the supply of Zn. In strongly acid soils, an excess Fe and Al may lead to induced deficiency of P, K, Ca, and Mg in the plant. The deficiency or lack of availability of other nutrients can also affect the rice plant's ability to decrease uptake of Fe in the tops through physiological functions carried out by roots such as Fe oxidation, Fe-exclusion, and Fe-retention.[3,15]

From the discussion in the preceding section on the role of plant nutrients in the occurrence of Fe toxicity in the rice plant, it can be hypothesized that some aspects of true or pseudo (induced) Fe toxicity can be managed by applying the plant nutrients whose deficiencies are induced by excess Fe or other toxic factors. The application of other plant nutrients can also mitigate Fe toxicity through their role via root

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functions related to reducing the amount of Fe taken up by the plant by oxidation, exclusion, or retention of Fe.

A number of studies have investigated the effects of application of other plant nutrients on Fe toxicity. Research under controlled conditions has provided interesting insights and hypotheses on the role of other plant nutrients in the management of Fe toxicity in wetland rice. Some recent studies in the field have tested the effects of plant nutrients in reducing Fe toxicity. This aspect is discussed with examples from recent research.

Benckiser et al.[78] studied the effect of N, P, K, Ca, and Mg fertilization on the performance of rice grown in pots using an Fe-toxic soil and a non Fe-toxic soil. They found that dehydrogenase activity, the number of nitrogen fixing, Fe-reducing bacteria, and Fe(II) production and the uptake of Fe by rice decreased with increased supply of K, Ca, and Mg. This effect was clearer with the rice variety IR 22, which is susceptible to Fe toxicity compared to the relatively tolerant variety IR 42. Data on the effects of plant nutrients on Fe uptake at maximum tillering stage by IR 22 and IR 42 rice plant tops (Table 4) showed that the application of K, Ca, and Mg together greatly reduced Fe uptake and accumulation compared to the control. A low supply of other plant nutrients and high Fe supply in a growth chamber experiment increased exudation (a measure of metabolic root leakage) and Fe uptake by rice variety IR 36. It was concluded that nutritional conditions, exudation by rice roots, and the Fe reducing activity of the rhizosphere were clearly related to Fe uptake by wetland rice. It was further concluded that Fe toxicity in wetland rice is a physiological disorder caused by multiple nutritional soil stress rather than by a low pH and high Fe supply per se.

Table 4. Effects of nutrients on iron uptake in IR 22 and IR 42 rice plants grown in pots with an iron-toxic soil. Relative iron uptake (%) over control at 6 weeks after transplanting.[78]

Treatment[a]	IR 22	IR 42
N	124	115
P	179	125
K	97	115
Ca + Mg	93	77
K + Ca + Mg	91	88

[a]Soil was fertilized with 100 mg N kg^{-1}as urea, 500 mg P kg^{-1} as ammonium phosphate, 100 mg K kg^{-1} as KCl, 50 mg Ca kg^{-1} as $CaCO_3$, and 20 mg kg^{-1} of soil as $MgCl_2$.

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Ottow et al.[45] reported that Fe toxicity of nutritionally poor acid sulfate and other mineral soils may be alleviated by application of nutrients, especially P, K, and Zn. They attributed Fe toxicity of rice to an ineffective functioning of the root oxidizing power as a result of an insufficient and imbalanced supply of nutrients such as P, K, Ca, Mg, and Zn.

In acid sulfate soils of Thailand, salinity, acidity, and Fe toxicity were found to be the most important constraints to rice production.[44] Iron toxicity was associated with high Fe(II) and low base status. Many individual soil parameters were correlated with rice growth and multiple correlation analysis indicated that the Fe activity ratio [the ratio of Fe(II) activity to the sum of the activities of all other divalent cations], pH, and ionic strength provided the best three-variable model describing dry matter production in the growth chamber study. In a field study, the best two variable model describing rice yields included pH and Fe activity ratio. The results of this study indicated the importance of divalent cation activity ratios when modeling rice growth and metal uptake in the acid sulfate soils.

Iron toxicity was not alleviated by application of plant nutrients (N, P, K, Ca) in an acid sulfate soil in Senegal and plants that accumulated more than 1500 mg Fe kg^{-1} in their leaves failed to give any grain yield at all.[45] Iron content in leaves of rice (cv. IR 8) plants sampled from all fertilizer treatments ($n = 23$) at the heading stage of growth (72 days after transplanting of rice) was related to grain rice grain yield:

$$\text{Grain yield (kg ha}^{-1}) = 19840754\ X\ (\text{iron content in leaves, mg kg}^{-1})^{-1.633};\quad r = 0.786$$

Yamauchi[39] reported that the severity of Fe toxicity (bronzing) in rice plants grown in pots of soils from Nigeria was affected not only by the Fe concentration in the shoots but also by the K concentration. The application of chloride salts to soil increased the severity of the bronzing whereas the application of sulfate salts was beneficial in alleviating the severity of toxicity. The application of potassium sulfate also reduced the severity of bronzing and increased the dry matter production of rice grown in the field on Fe toxic soils. The concentration and accumulation of K in the shoots increased when the bronzing severity decreased and the Fe concentration was decreased by increased dry matter production. The dry matter production was closely related with K accumulation, and K application was responsible for the dilution effect.

The effect of K in decreasing the severity of Fe may also be related to the role K plays in maintaining the oxidizing power of the rice roots.[86]

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Linear regression between the bronzing score and K concentration in the rice shoots in a field study in Nigeria suggested that the K concentration required for a bronzing score of 3 (mild Fe toxicity/oranging) was about 2.7% K (39). This is appreciably higher than the published critical concentration of 1.0% (for rice straw at maturity or leaf blade at tillering) for normal growth of rice plants.[3]

Gunatilaka and Bandara[87] made field experiments to determine the effects of P and K applications on the performance of rice varieties in rice paddies in Sri Lanka. The soils used in the study were acidic in reaction (pH 4.0 to 4.9), well supplied with organic matter and total N (organic matter 2.5 to 22.8%; total N 1250 to 3800 mg kg^{-1} soil), and low in extractable P (Olsen P 1.6 to 3.5 mg kg^{-1} soil) and exchangeable K (0.04 to 0.08 m.e. 100 g^{-1}soil). It was found that the higher rates of K (>58 kg ha^{-1}) and P (>32 kg ha^{-1}) applications effectively reduced Fe toxicity and increased the rice yields on Fe-toxic mineral and organic soils. Without application of P and K, the rice plant leaves were deficient in P and K and high in Fe. Application of P and K fertilizers increased the content of P and K, and decreased that of Fe in the leaf tissue. The tolerant variety performed better than the Fe toxicity-susceptible variety. Balanced P and K nutrition of Fe toxicity tolerant rice varieties was found to be effective in reducing Fe toxicity and increasing rice productivity.

Sarwani et al.[14] observed that Fe toxicity in rainfed wetland rice in South Kalimantan, Indonesia was as a result of excessive uptake of Fe and was associated with high soil Fe and low soil K. The results of long-term field experiments conducted at different Fe-toxic sites showed that the application of K fertilizer reduced Fe toxicity symptoms and improved rice yields.

Sahrawat et al.[23] conducted pot and field experiments to study the role of other nutrients in the management of Fe toxicity of lowland rice in the Ivory Coast (West Africa). In a pot experiment examining the role of nutrients on grain yield of rice it was observed that the typical Fe toxicity symptoms appeared on the rice plants in all treatments 4–6 weeks after emergence. Application of P alone or together with K and Zn delayed the appearance of the toxicity symptoms by 1–2 weeks but did not alleviate them. The soil was low in extractable K and Zn but adequate in extractable P. None of the treatments significantly affected grain and dry matter production, although the grain and dry matter yields were higher in treatments in which P was added alone or in combination with K and Zn.

In the field experiment conducted at a Fe toxic site at Korhogo, Ivory Coast in 1993, two varieties were Suakoko 8, an Fe toxicity tolerant cultivar, and Bouake 189, a susceptible cultivar. The effects of nine

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Table 5. Effects of field applications of nutrients on the grain yield of two rice varieties in an iron toxic soil, Korhogo, Ivory Coast, 1993.[23]a

	Grain yield ($t\,ha^{-1}$)	
Treatment	Suakoko 8	Bouake 189
Control (no fertilizer)	5.79	4.47
N	5.43	4.59
N + P	6.02	6.70
N + K	6.77	6.24
N + Zn	6.12	5.75
N + P + Zn	6.09	5.58
N + K + Zn	5.37	5.36
N + P + K	6.85	5.70
N + P + K + Zn	6.05	6.19
S. E.	0.239	0.413
C. V.	8	15

[a]The soil at the site was an Ultisol (pH, KCl 4.1; organic C 1.02%; Bray 1 P 8 $mg\,kg^{-1}$; extr. K 55 $mg\,kg^{-1}$; extr. Fe 137 $mg\,kg^{-1}$; extr. Zn 7 $mg\,kg^{-1}$).

nutrient treatments (no fertilizer, N, N + P, N + K, N + Zn, N + P + Zn, N + K + Zn, N + P + K, N + P + K + Zn) were tested. Nitrogen was applied at a rate of 100 $kg\,N\,ha^{-1}$ as urea in three splits; P was applied as at 50 $kg\,P\,ha^{-1}$ as TSP; K as KCl was added at 80 $kg\,ha^{-1}$; Zn was applied at 10 $kg\,Zn\,ha^{-1}$ as ZnO. All nutrients except N were added as basal applications. In the control (no fertilizer) treatment, the tolerant Suakoko 8 out yielded Bouake 189. The effects of different nutrient combination treatments for Suakoko 8 were not clear because of severe lodging, especially when N was applied. Application of P + K with N, and N + P + K + Zn combination significantly improved the yield of Fe toxicity-susceptible Bouake 189 (Table 5). While application of N did not affect the yield, application of P or K in combination with N and Zn significantly increased the grain yield of Bouake 189.[23]

The results on the role of other nutrients in reducing Fe toxicity were confirmed in subsequent field experiments conducted for several seasons on Fe-toxic sites in Ivory Coast, using Fe-tolerant and susceptible lowland rice cultivars with and without application of plant nutrients.[27,34,35,88] As an example, the results of an experiment conducted for four years (1995–1998) for evaluating the effects of plant nutrients on the performance Fe-tolerant and susceptible rice cultivars are given in Table 6. The application of other nutrients decreased Fe toxicity stress,

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Table 6. Effects of field applications of nutrients on iron toxicity score (ITS) and grain yield of iron-tolerant CK 4 and susceptible Bouake 189 and TOX 3069-66-2-1-6 lowland rice cultivars grown on an iron-toxic soil, Korhogo, Ivory Coast. ITSs are given in parentheses.[35]a

	Grain yield ($t\,ha^{-1}$)		
Treatment	CK 4	Bouake 189	TOX 3069-66-2-1-6
No fertilizer	4.3 (3)	3.4 (5)	2.9 (7)
N	4.4 (3)	4.1 (5)	3.3 (7)
N + P	5.3 (2)	4.3 (4)	4.2 (5)
N + K	4.8 (2)	4.4 (4)	3.8 (5)
N + Zn	4.8 (2)	4.6 (4)	4.6 (5)
N + P + Zn	5.0 (2)	4.4 (4)	4.2 (4)
N + K + Zn	5.2 (2)	4.6 (3)	4.6 (4)
N + P + K	5.4 (2)	4.5 (3)	4.5 (3)
N + P + K + Zn	5.7 (2)	4.7 (3)	4.7 (3)
LSD (0.05)	1.01	1.02	1.15

[a]The results presented are average of four years (1995–1998).

as judged by Fe toxicity scores, and increased grain yield.[35] Sahu et al.[89] conducted field experiments for five seasons on Fe-toxic soils in Orissa (India) and found that the application of K decreased the intensity of Fe toxicity and increased the grain and straw yield of four rice cultivars (two Fe-tolerant and two susceptible).

The results of plant analysis of the two cultivars that differed markedly in their tolerance to Fe toxicity at the Korhogo site showed no apparent differences in elemental composition with regard to macro- and micronutrients. It seemed all nutrients except Fe were adequate, based on the data on critical limits for deficiency and toxicity of nutrient elements taken from Yoshida[3] and Fageria et al.[90] for both cultivars. Both Fe toxicity-tolerant and susceptible cultivars accumulated high Fe in the rice tops, well above the critical limit for Fe toxicity. Suakoko 8 accumulated high amounts of Al, bordering toxic concentrations. The concentrations of Mg, especially in Suakoko 8 plant tops, were bordering deficiency. Application of N + P + K + Zn considerably reduced Fe uptake and accumulation in the rice plant tops (Table 7). The mechanism of reduction in Fe uptake in the rice tops by application of other nutrients cannot be established. These results support the earlier work by Benckiser et al.,[78] who reported that application of plant nutrients such as P, K, Ca, and Mg reduced the uptake of Fe in the rice tops and increased the

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Table 7. Nutrient content (mg kg^{-1}) in plant tops of iron toxicity tolerant and susceptible rice varieties under control (0) and added nutrient (NPKZn) treatments at the tillering stage, Korhogo, Ivory Coast, 1993.[23]

Nutrient element	Critical content	Bouake 189[b]		Suakoko 8[b]	
		0	NPKZn	0	NPKZn
P	<1,000	3,777	3,760	2,709	3,011
K	<10,000	26,153	33,722	24,699	34,874
Ca	<1,500	4,751	2,828	5,180	4,700
Mg	<900	962	1,015	860	852
Fe	>300[a]	900	500	704	516
Mn	<20	298	145	472	394
Zn	<10	31	33	29	39
Cu	<5	18	6	6	6
Al	>300[a]	186	272	395	328

[a]Critical limit for toxicity rest all, refer to critical limits for deficiency of nutrient elements.
[b]Without and with fertilizer treatment.

yield of rice grown in pots. As expected, the effect of other nutrients on Fe uptake and yield of rice was clearer with the Fe toxicity-susceptible variety.

PERSPECTIVES

Iron toxicity is a widespread nutrient disorder that affects the growing of wetland rice. Recent research on Fe toxicity has established the conditions that lead to the occurrence of Fe toxicity in rice. Iron toxicity occurs on soils high in reducible Fe and potential acidity, irrespective of texture, and organic matter content. Soil characteristics, including organic matter and texture, however, influence the level of Fe in soil solution at which the toxicity would occur. Iron toxicity is caused by excess water soluble Fe(II) and aggravated by low base status and the deficiencies of plant nutrients such as P, K, and Zn. Iron toxicity can be reduced by growing Fe-tolerant genotypes of rice and by application of plant nutrients such as P, K, and Zn.

There is an interaction between Fe toxicity and availability of plant nutrients and this interaction is poorly understood at the present time. This perhaps explains why the application of other plant nutrients gives

a response in the improvement of growth and yield of rice, especially in situations where deficiencies of these nutrients are induced by high concentration of Fe or in some instances induced by excess Al in soil solution.[5,12,44] However, the role of plant nutrients on Fe toxicity and its management is quite complex because nutrients such as P, K, Ca, and Mg also influence the rice plant's ability to tolerate Fe toxicity through root functions. However, it is not clear whether the nutrient requirements for such functions is higher than those required for correcting the deficiency of these nutrients. For example, Yamauchi[39] found that K concentration in rice plant at the mild oranging or mild toxicity due to Fe was considerably greater than the critical concentration for K deficiency per se (with no interference from Fe toxicity).

It must be mentioned that there is confusion in the literature relating to Fe toxicity syndrome, and that this confusion stems mainly from the inability to distinguish between the type of Fe toxicity: true (caused by high concentration of Fe in soil solution) or pseudo (probably caused by deficiency of other nutrients).

The role of other plant nutrients needs to be carefully evaluated and interpreted in the context of the two types of Fe toxicity (true or pseudo). For example, Sahrawat et al.[23] showed that in the case of a true Fe toxicity (when the content of Fe in the plant tissue was high, the concentrations of other nutrients were in the normal range), application of the other nutrients decreased Fe uptake and accumulation in the plant tissue. These results suggest that other nutrients were involved in reducing the Fe toxicity rather than in correcting their deficiency (see data in Table 7). Moore and Patrick[43] and Moore et al.[44] showed that the relative amount of Fe in solution is the most important parameter with respect to Fe toxicity in rice.

However, with the available information, the relationship between Fe toxicity (true or pseudo) and nutrient supply cannot be clearly established and will require further research. Such interactions appear important in the management of Fe toxicity in wetland rice and it is hoped that this review will stimulate further research in this important area.

Rice plants counteract Fe toxicity stress by preventing excess Fe(II) uptake at the roots and by tolerance of the tissue. The mechanisms involved in the prevention of excess Fe uptake and tolerance to high Fe concentration are not established. Clearly, there is an urgent need for further research on the reaction of Fe(II) in the plant tissue to clarify the tolerance mechanism.[81] Research should be directed towards developing more sophisticated techniques, like those based on the relationship between Fe toxicity and enzymatic activity such as superoxide dismutase,

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peroxidase and catalase in the plant tissue, for determining the genotypic differences, and the mechanisms involved in Fe toxicity tolerance.[91]

A synergy between genetic tolerance and nutrient management strategy is needed for sustainable rice production on Fe-toxic soils.[23,27] The intensified use of these stressed soils is unavoidable in the face of an effort for meeting the food needs of ever growing population. A clear understanding of the role of plant nutrients in Fe toxicity and its management is a prerequisite for developing a sustainable nutrient management strategy for the Fe-toxic soils. Such research is also needed for exploiting the yield potential of Fe toxicity tolerant genotypes of rice on a sustainable basis.

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