IRON TOXICITY OF RICE AS A MULTIPLE NUTRITIONAL SOIL STRESS

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Introduction

To meet the food requirements for the world population in the future, agricultural production should increase by 60% at the end of this century (Dudal, 1980). The greatest contribution to this increase is expected from the intensification of agriculture in the tropical areas. In the densely populated areas of South and Southeast Asia an estimated 100 million ha of potentially arable land still remain idle, mainly because of yield-affecting soil constraints such as acidity, alkalinity, salinity, toxicities and/or excess of organic matter (Ponnamperuma *et al.*, 1981). In addition, some millions of ha of cultivated Asian soils produce restricted rice yields because of aluminum and/or iron toxicities and several nutrient deficiencies. In particular, with the introduction of high yielding rice varieties and the increased use of nitrogen, iron toxicity has become a well recognized, widely distributed disorder in Southeast Asia, South America and Africa (with various lines of *Oryza sativa* var. *indica*) as well as in Japan, Italy and Spain (*O. sativa* var. *japonica*) (Table 1). Iron toxic plants usually occur in local spots (Philippines, Sri Lanka, Java, Liberia, Sierra Leone, etc.) or may cover large areas (of young acid sulfate soils) in coastal plains and inland valley swamps (Brunei, Kalimantan, Liberia, Sumatra, Colombia). To the modern rice varieties excesses of minor elements such as Fe (together with Mn) may provide main obstacles to the delivery of full potentials on a great variety of wetland soils, although big differences in tolerance between the various traditional and modern varieties have been observed (Jayawardena *et al.*, 1977; Mahadevappa *et al.*, 1979). Soils that have been recognized as iron toxic mainly belong to the acid sulfate soils (Sulfaquepts) or are found among the poorly drained colluvial – as well as alluvial valley areas (Oxi- and Ultisols), often with a continuous supply of soluble iron by interflow and/or upwelling from adjacent slopes (Ota and Yamada, 1962; Tanaka and Yoshida, 1970; Van Breemen and Moormann, 1978). "Bronzing" as well as "yellowing" has been reported also on organic soils and oligotrophic mucky areas of coastal regions or freshly reclaimed valley swamps (Histosols). In all these soils a relatively high concentration of soluble iron (caused by intensive bacterial iron-reduction) combined with a relatively low pH (< 5) have been claimed to be responsible for this disorder. However, toxic Fe levels vary between 30 and about 2000 ppm (Tanaka *et al.*, 1966; Tanaka and Yoshida, 1970; Moormann and Van Breemen, 1978). Although such considerable differences may be explained by different soil properties as well as by the great variation in rice tolerance to iron (Tadano, 1976; Yamanouchi and Yoshida, 1981), other factors should be sought in order to account for these remarkable observations. Curing or preventing iron toxicity is not a simple procedure, since neither drainage nor the incorporation of compost, lime, MnO_2, potash or sodium nitrate completely eliminated this problem (Ota and Yamada, 1962; Nhung and Ponnamperuma, 1966; Sahu, 1968; Tanaka and Tadano, 1972). In fact, little is known on the prerequisites and conditions of soil that culminate in the intoxication of rice. If iron toxicity is induced by specific environmental and/or physico-chemical conditions, these constraints should be shared by most of the iron toxic soils. In the present study, a large number of different soils, clearly showing iron toxicity symptoms, was collected from a large variety of geographical sites

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and subjected to extensive chemical analyses, together with the damaged leaves.

Table 1 Occurrence and designation of iron toxicity in tropical and subtropical countries

Nation	Local designation
Tropics	
Columbia	"Anaranjamiento" (oranging")
India	"Ufra", "Chatra, "Bhangiphuti", Khaira", "Dakhina"
Indonesia	"Mentek" (red disease) or "Mutut" (retarded flag leaf)
Malaysia	"Penyakit Merah" (red disease)
Philippines, Sri Lanka	"Bronzing", "Yellowing"
Pakistan	"Pansukh"
Taiwan	"Chin-seng-tien" (suffocating")
Subtropics	
Italy	"Crodatura"
Japan	"Akagare I und II" (red wiltering) [1]
Spain	"Faille"

1) "Akagare" III is ascribed to Zn deficiency

Constraints of iron toxic soils

In Table 2 the main physicochemical features of various iron toxic soils within the Philippines are presented. These soils were collected at those parts of the field that carried affected rice with distinct "bronzing" and/or "yellowing" symptoms. The data on the corresponding leaves and varieties are presented in Table 3. From Table 2 the following general results are obvious. First, the pH of the soils ranges between neutral (pH = 7.4) and acid (pH = 4.4). Second, the total amount of iron (Fe_t) as well as its "active" part (Fe_o) varies considerably. Apparently, a low pH as well as a high amount of Fe are not essential factors for iron toxicity. Third, the total cation exchange capacity (CEC) should be regarded as relatively low (except for the Lapulapu site at Palawan which is relatively rich in organic matter). Such a low CEC implies both a restricted buffering feature as well as a limited supply of available nutrients. Fourth, the amount of exchangeable bases – in particular K and Ca – should be considered insufficient to support the normal development of a rice crop. Further, a clear deficiency is recorded in "available" phosphate (Olsen) and Zn (with most of the samples listed). Apparently, iron toxic soils have several essential constraints in common, all of which may interfere, alone or in combination, with the normal and healthy development of a rice crop. These soil deficiencies are indeed confirmed by the results of the leaf analyses (Table 4). Apart from clear P and K deficiencies, most leaves revealed also low amounts of Zn. Lack in Ca is not shown by the leaves, probably because of its immobile nature and accumulation in the lower (oldest) leaves.

Table 2 Physico-chemical properties of iron toxic soils collected in the Philippines

Location	pH (H_2O KCl)	C %	N %	CEC (meq/ 100g)	exch. cations K	Na	Ca	Mg	P [2] (Olsen) ppm	Zn [2] (K and P) ppm	Mnt	Fet [3]	Feo [3]	Feo/Fet
					meq/100g						%			
Lapulapu (Palawan)	7.4/ 6.0	1.1	0.16	24.8	0.02	0.04	1.3	22.4	0.9	nil	0.28	11.2	1.0	0.09
Bangkatan (Mindoro)	5.7/ 4.6	1.5	0.18	9.5	0.09	0.18	2.8	0.5	3.0	3.7	0.007	5.6	0.6	0.11
Calapugan (Luzon)	6.3/ 4.1	1.0	0.11	10.7	0.11	0.34	7.9	7.1	3.0	0.1	0.001	6.0	0.6	0.10
Labo (Cam. Norte)	4.9/ 3.9	2.5	0.27	9.5	0.26	0.12	2.5	1.3	5.0	6.4	0.002	3.2	1.1	0.34
Abuyog (Sorsogon)	4.6/ 3.5	1.7	0.17	12.4	0.07	0.40	5.2	1.5	7.1	1.0	0.002	2.8	0.9	0.32
San Dionisio (Panay)	4.4/ 2.9	1.5	0.14	8.4	0.08	0.06	0.5	0.18	2.0	2.7	0.003	2.3	0.6	0.26
Maahas clay (IRRI) 4)	6.7/ 5.6	1.3	0.17	29.3	1.24	1.26	14.9	8.5	11	2.0	0.23	7.3	1.9	0.26
Critical [5] levels	–/–	–	0.20	–	0.20	?	~10	2–5	10	2.0	–	–	–	–

1) the texture ranges from sandy loam to clay

2) extracted with 0.5M $NaHCO_3$ and 0.05N HCl, respectively (Castro, 1977)

3) Fet = total amount of Fe; Feo = acid oxalate extractable "amorphous" and active Fe

4) fertile soil of the International Rice Research Institute, Manila/Philippines

5) data mainly from IRRI report (1969) as well as Tanaka and Yoshida (1970)

Table 3 Mineral contents of leaves clearly showing iron toxicity symptoms (bronzing or yellowing)

Location in Philippines	var. [1]	Type	Fe (ppm)	Mn (ppm)	N (%)	P (%)	K (%)	Mg (%)	Ca[2] (%)	Na (%)	Zn (ppm)	Remarks
Lapulapu (Palawan)	Djeremas	Y [3]	517	nd	2.0	0.05	0.6	0.8	0.2	0.2	7.3	**stunted growth**
Bankgatan (Mindoro)	IR42	y	1067	1029	2.2	0.27	0.3	0.2	0.7	2.1	19.8	**irregular** growth and panicle formation
Calapugan (Luzon)	IR36	y	435	745	2.6	0.11	1.7	0.2	0.6	0.02	12.0	**classic** "bronzing"; growth retardation
Labo (Cam. Norte)	IR42	BY	649	135	2.5	0.19	0.8	0.2	0.3	0.05	14.8	**heavily** affected, abandoned field
Abuyog (Sorsogon)	IR42	Y	450	166	2.0	0.15	0.8	0.2	0.1	0.7	12.5	**yellow-orange** leaf blades, green midrib
San Dionisio (Panay)	IR36	B	922	172	2.3	0.18	1.0	0.1	0.3	0.2	20.2	**characteristic** "bronzing"
Threshold [4]	toxicity		>300	>2500								
	deficiency		< 70	< 20	2.5	0.1–0.2	1–2	0.1	0.2	?	20	

1) leaves collected in age between tillering and heading stage

2) Ca is not translocated in the plants and thus relatively enriched in the older leaves

3) Y = yellowing B = bronzing

4) **consult Tanaka and Yoshida (1970)**

Table 4 Physico-chemical propertices of iron toxic soils collected in Southeast Asia and Africa

Nation and location [1]	pH (H_2O/KCl)	C %	N %	CEC (meq/100g)	exch. cations K	Na	Ca	Mg	P (Olsen) ppm	Zn (K and P) ppm	Mnt	Fet %	Feo	Feo/Fet
					meq/100g									
Brunei (N. Borneo)														
Sinaut	4.9/3.8	1.6	0.19	9.7	0.12	0.07	0.5	1.1	3.0	5.5	0.009	3.2	1.3	0.41
Tanah Jambu	4.3/3.7	2.2	0.20	7.0	0.08	0.07	0	0.6	2.0	3.0	0.004	1.9	0.6	0.32
Indonesia (Java)														
Ciseeng	5.2/4.4	2.1	0.27	19.8	0.13	0.10	3.4	1.3	2.0	1.8	0.45	10.8	0.9	0.08
Karanwangi	5.5/4.6	2.3	0.23	28.9	0.09	0.13	21.8	6.1	5.0	0.3	0.93	9.5	1.7	0.18
Cihea	5.3/4.4	2.3	0.22	29.2	0.14	0.44	21.0	5.9	8.0	0.4	0.15	5.2	1.1	0.21
Sri Lanka Bumbuwela area														
a) Rice Res. Station	4.8/4.0	1.5	0.13	3.4	0.07	0.04	0.60	0.2	2.0	3.6	0.004	1.8	0.6	0.33
b) Polgaha Lidumulla	4.7/3.8	1.7	0.11	3.5	0.01	0.01	0.22	0.03	1.0	0.3	0.004	1.0	0.3	0.30
c) Hora Farm	4.9/3.9	0.8	0.07	2.2	0.03	0.02	0.02	0.07	0.5	0.3	0.003	0.6	0.1	0.17
Padukka	5.1/4.1	1.3	0.07	2.5	0.01	0.02	0.11	0	1.0	1.4	0.008	0.7	0.1	0.14
Pussaellwa	5.1/4.5	1.7	0.19	5.6	0.06	0.06	0.54	0.12	3.0	1.7	0.002	9.8	0.7	0.07
PR China [2] Cangcheng-Kaiping	5.2/4.1	1.0	0.09	1.8	0.08	0.04	0.50	0.09	5.0	0.7	0.01	1.1	0.3	0.21
Liberia (Africa) [3]														
Suakoko	4.8/4.0	1.7	0.14	4.4	0.09	0.05	0.60	0.12	8.0	3.1	0.006	0.8	0.6	0.75
Bong Mines	4.9/4.2	2.1	0.17	4.6	0.06	0.06	1.80	0.28	8.0	2.3	0.004	0.4	0.2	0.50
Maahas clay (IRRI)	6.7/5.6	1.3	0.17	29.3	1.24	1.26	14.9	8.5	11	2.0	0.23	7.3	1.9	0.26
Critical levels	–/–	–	0.20	–	0.20	?	~10	2–5	10	2.0	–	–	–	–

1) with texture from loamy sand to loam

2) People's Republic of China

3) **leaves were not available for analysis**

Table 5 Mineral contents of the leaves with clear symptoms of iron toxicity

Nation and location	Variety	Type	Fe (ppm)	Mn (ppm)	N (%)	P (%)	K (%)	Mg (%)	Ca (%)	Na (%)	Zn (ppm)	Remarks
Brunei (N. Borneo)												
Sinaut	SM-1	B	695	183	2.4	0.22	0.89	0.17	0.49	0.07	14.4	leaves of recovered plants
Tanah Jambu	Galoh-Paya	B	1010	155	2.4	0.15	0.69	0.08	0.34	0.13	16.5	symptoms weakly developed
Indonesia (Java)												
Ciseeng	Cisadane	Y	210	2800	2.2	0.10	0.63	0.10	0.50	0.32	14.3	stunted growth
Karanwangi	Cisadane	B	170	1360	1.3	0.12	0.66	0.27	0.58	0.64	13.5	typical "bronzing"
Cihea	Semeru	B	150	1255	1.1	0.11	0.59	0.17	0.57	0.08	16.1	retarded flag leaf development ("mutut")
Sri Lanka												
Bumbuwela-Region												
a) Rice Res. Station	BG400/1	B	1998	35	2.6	0.19	0.94	0.20	0.50	0.74	16.8	stunted growth
b) Polgaha Lidumulla Hondarawala		B	580	64	3.2	0.14	0.21	0.15	0.61	1.55	25.8	symptoms only on flag leaf
c) Hora Farm	BG 346	B	2217	191	3.4	0.18	1.34	0.14	0.45	0.13	20.2	sensitive variety!
Padukka	Kahawanu	B	1375	85	3.4	0.44	0.47	0.16	0.50	0.22	23.9	Fe-interflow and seepage
Pussaellwa	H4 ?	B	809	701	1.7	0.09	0.49	0.16	0.89	0.55	24.9	typical iron toxic site
PR China												
Cangcheng-Kaiping	?	B	660	176	0.6	0.07	2.3 [1)]	0.06	0.55	0.14	28.0	leaves at ripening
Threshold	toxicity	–	>300	>2500								
	deficiency	–	< 70	< 20	2.5	0.1–0.2	1–2	0.1	0.2	?	20	based on experience

1) relatively high in relation to N and P

In Table 5 various iron toxic soils from Brunei (Borneo), P.R. of China, Indonesia (Java), Liberia and Sri Lanka (Ceylon) are characterized. Although collected from quite different geographical locations, the same general constraints as shown in Table 3 are observed. The most aberrant result is the relatively high CEC as recorded in the soils from Java. The classical "bronzing" soils of Sri Lanka – in total more than 40,000 ha (Ota, 1968) – are all uniformly deficient in P, K, Ca, Mg as well as Zn, suggesting that essential constraints for the induction of "bronzing" should be seen in a multiple nutritional stress. The insufficient supply of P, K and Zn (partly) is reflected by the analyses of the leaves (Table 5).

Iron toxicity as an indirect physiological disorder

The results presented here are consistent with a view of iron intoxication, in which multiple nutritional soil stress should be regarded as the essential triggering mechanism. The responsibility of low nutrient levels in Fe toxicity has been suggested before. Thus, Ota and Yamada (1962) ascribed "bronzing" to a deficiency in Ca (together with a toxicity of Al), whereas Inada (1965) and Ota (1968) considered a low supply of Mn as the promoter of an excessive Fe uptake. Recent studies (Van der Vorm and Van Diest, 1979) showed, however, that Fe adsorption by rice plants under acid conditions is little affected by the presence or absence of Mn. According to several other papers (Kandiah, 1952; Sahu, 1968; Tanaka and Tadano, 1972; Trolldenier, 1973; Ismunadji, 1976) potassium deficiency is claimed as an essential factor governing the tolerance for high Fe (II) concentrations in soil. Nevertheless, several experiments considering the supply of K alone failed to reduce "bronzing" symptoms (Takijima and Knaganayagam, 1971; Ismunadji, 1976). The role of Ca and K remains obscure, since plants suffering from "bronzing" may contain high amounts of Fe, Ca and N, and low levels of K, Mg and Si (Takijima and Gunewardena, 1971). Analyses of the corresponding soils, however, could have shown the lack or deficiency in K, P, Ca, Mg and Si. The role of P as *one* of the essential prerequisites for iron toxicity is quite evident, since soils low in both K and P were iron toxic even at ferrous iron levels as low as 30 ppm (Moormann and Van Breemen, 1978). These observations together with the results presented here suggest that iron toxicity can be regarded as an indirect physiological disorder induced by low levels of P, K and Ca rather than by a direct excessive influx of high amounts of mobile Fe in an acid environment (see Ottow *et al.*, 1981).

Breakdown of the iron-excluding mechanism

Rice roots, in particular those parts involved in the uptake of nutrients, are effectively equipped with an iron-oxidizing and excluding power. This mechanism (O_2 excretion, peroxidases?) prevents excessive reduced and mobile Fe (and probably Mn) from entering free space and cytoplasm. Healthy, actively metabolizing roots are coated by Fe(III) – oxides and hydroxides (lepidocrocite) that form red-brown micropedotubules. Iron toxic plants usually display a gray, gray-brown or brown-black root system that is stunted in growth, scanty and coarse. Detailed root observations (microscope!) reveal damaged and partly dissolved coatings. Older roots of the central part are mostly decayed and black (FeS by sulfate reduction). These morphological changes may be ascribed to the collapse of the iron-oxidizing and excluding mechanism in the rhizosphere. In order to understand the devices with which the rice root regulates the excessive mass flow of soluble Fe (and Mn), it is important to realize that the oxidizing power of the root surface is achieved by a sensitive balance between root exudation and O_2-supply on the one hand and the activity of the rhizoflora on the other. Determining factor of the latter is the permeability of the root membrane that regulates both influx and efflux (amount of organic exudates) (Rovira, 1969). Plants insufficiently supplied with K, P and Ca show dramatic changes in their metabolism. In K-deficient rice plants low molecular weight compounds (such as soluble sugars, amides, and amino acids) accumulate at the expense of higher molecular weight moieties, because several synthetic processes are delayed by the lack of this essential element (Ismunadji, 1976). Together with Ca, potassium is also involved with root membrane permeability, and a lack of both elements is

responsible for an increase in permeability and an enhanced metabolic leakage (Wyn Jones and Lunt, 1967). The metabolic disturbance is aggravated considerably by an insufficient supply of P (essential for root growth, energy transfer and several synthetic processes). Thus, rice plants showing severe "bronzing" and low levels of P, K and Ca exude probably considerably more low molecular weight metabolites than those well supplied with these elements. As a consequence, the density and activity of the rhizoflora increases, resulting in a higher O_2 respiration at the root surface. Under such conditions the bacteria (facultative and obligate anaerobic) will switch to Fe(III) and Mn(IV) – oxides in their immediate surroundings (micropedotubules) in order to continue their energy conserving (ATP-synthesis) reactions (Ottow, 1970; Ottow and Glathe, 1973; Munch and Ottow, 1977; 1980):

Metabolism (dehydrogenation):

$$\text{organic matter (hydrogen donor)} \xrightarrow{\text{dehydrogenases}} \text{metabolic products} + \text{ATP} + \text{e} + \text{H}^+$$

FE(III) is acting as hydrogen acceptor (hydrogenation):

$$\text{Fe-OH} + \text{e} + \text{H}^+ \xrightarrow{\text{ferri-reductases}} \text{Fe(II)} + \text{H}_2\text{O}$$

The increased oxygen consumption and the reductive dissolution of the protective Fe_2O_3 coatings culminate in a breakdown of the Fe-excluding mechanism (Fig. 1) and allow an excessive uptake of Fe by mass flow. The bleached and damaged roots may decay (and become blackened by FeS) and numerous adventitious roots are produced in the soil surface to maintain nutrient uptake and avoid high Fe(II) concentrations.

The hypothesis of iron toxicity as a multiple nutritional stress has been confirmed experimentally. In the greenhouse at IRRI on a translocated iron toxic soil (from San Dionisio, Table 2) "bronzing" could indeed be completely eliminated (with IR 26 and IR 42), if P, K and Ca were applied in combination (detailed results will be published elsewhere).

Role of Zn in iron toxicity

Whereas deficiencies in P, K and Ca should be regarded as the essential ecological prerequisites for the metabolic disorder and excessive Fe uptake, the lack of Zn (Tables 3 and 5) is more likely to act as an additional growth limiting factor. Zn deficiency in wetland rice is characterized by stunted growth, blanching at the base of the emerging leaves and by rusty brown discoloration of the other leaves (Castro, 1977). In all those cases where "bronzing" or "yellowing" is accompanied by retarded growth (Tables 3 and 5) this may be attributed primarily to an insufficient supply of Zn (Zn is essential for the synthesis of heteroauxins and internodal elongation). In fact, the coexistence of iron toxicity, P, K and Ca deficiency and low amounts of available Zn is quite common, since these stresses are shared by many of the known iron toxic soils (Table 6).

Practical implications

There is a great varietal difference in the tolerance to iron toxicity (Table 7). In general, the local, traditional rice varieties are less sensitive to iron toxicity than the modern high yielding lines (Virmani, 1976; Gunewardena, 1979). In our own field experience we observed that the traditional farmers' lines generally developed larger root systems under adverse soil stresses than the semidwarf modern varieties. This feature may provide the former with a more efficient nutrient "extracting" ability. The capacity to extract P, K, Ca, Zn and other nutrients in a relatively efficient way is an important heritable polygenic feature that should be crossed into modern lines with high yielding potentials. Breeding for multiple stress tolerance in combination with favorable plant characteristics is an urgent task for the near future. Breeding may be a simpler and less expensive method of overcoming the multiple stresses than the required, repeated application of fertilizers

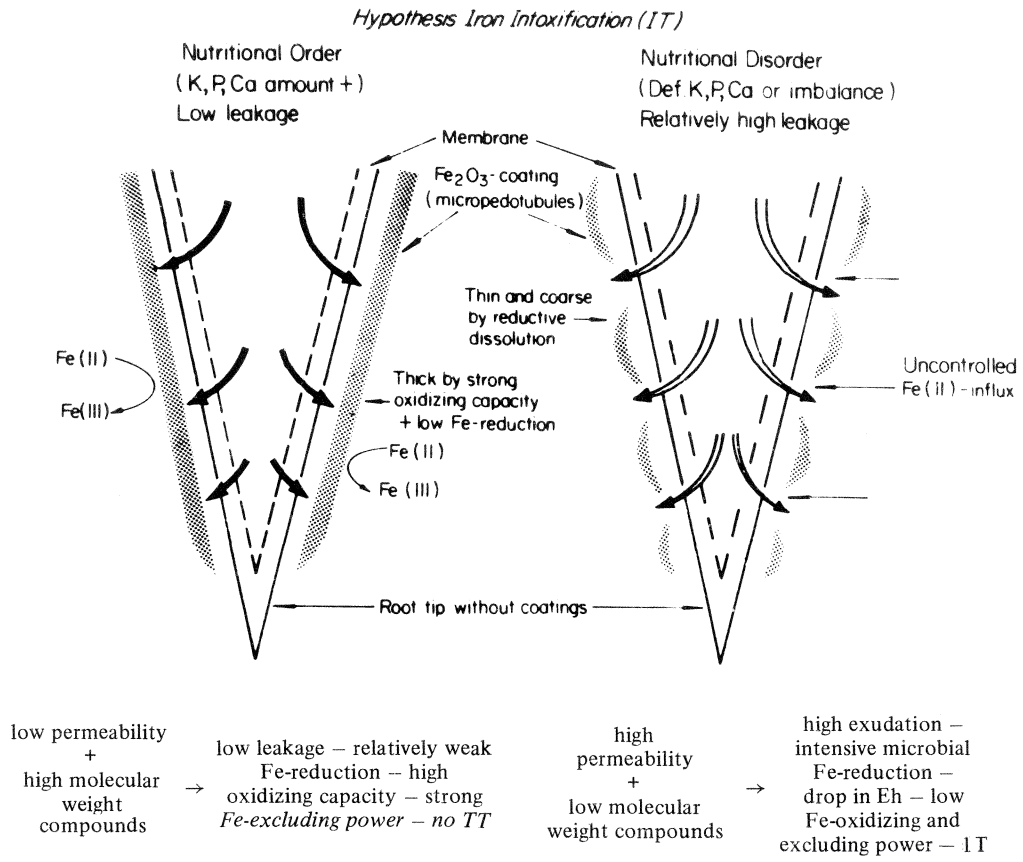

Fig. 1 **Model for iron toxicity of wetland rice as a physiological disorder caused by multiple soil stress (deficiency in P, K and Ca).**

Left: Well nourished rice plants (adequately supplied with P, K and Ca) are characterized by a low leakage of organic metabolites (exudates) and a high iron-excluding capacity (O_2 – exudation, peroxidases?) that prevents soluble Fe(II) from entering the roots

Right: Deficiencies in K (relative accumulation of low molecular weight compounds), P (disturbed energy transfer, synthetic processes and root growth) and Ca (increased root permeability) are the essential prerequisites for an increased exudation and rhizoflora activity. The high leakage in metabolites causes an increased O_2 demand and the use of Fe(III) as an alternative electron acceptor by "iron-reducing" bacteria. This culminates in the breakdown of the Fe-excluding mechanism (dissolution of Fe_2O_3 – micropedotubules)

(Ponnamperuma, 1976a, b; 1977; Mahadevappa *et al.*, 1979). Some of the latest IR varieties are relatively tolerant to multiple stresses (Table 7) outyielding most other lines on adverse soil conditions. In particular IR 42 should be mentioned in this respect.

Table 6 **Iron toxic soils and related soil stresses as observed in Southeast Asia (data taken from Ponnamperuma, 1976)**

Soil group	Other growth limiting factors
Acid sulfate soils (Gleysols) [1)]	Acidity; salinity; N, P and base deficiencies; deep water
Oxi-and Ultisols (Ferralsols)	P and several base deficiencies; low Si and Zn content
Histosols (Histosols)	N, P, K, Nz, Ca and Cu deficiencies; H_2S toxicity; deep water

1) Approximate equivalents in FAO Soil Classification are listed in parentheses

Table 7 **Relative evaluation of different IR-varieties with respect to relevant adverse soil condition on a scale 0–9 (0 = no information, 1 = almost normal plant development and 9 = dead or nearly dead plant) (data taken from Ponnamperuma, 1976)**

var.	Toxicities					Deficiencies		Total relative [1)] score
	Salt	Alkali	Peat	Iron	Boron	Phosphorus	Zinc	
IR5	4	7	0	6	4	5	5	31
IR8	3	6	5	7	4	4	4	33
IR20	5	7	4	2	4	1	3	29
IR22	5	7	0	3	3	3	3	24
IR24	3	5	0	3	4	1	4	20
IR26	5	6	6	6	4	1	5	33
IR28	7	5	6	4	4	3	5	34
IR29	6	7	5	4	4	5	3	34
IR30	5	6	3	3	3	3	3	26
IR32	5	7	5	3	3	3	5	31
IR34	5	5	3	3	3	3	3	25
IR36	3	3	3	3	3	7	2	24
IR38	5	5	3	5	3	1	3	24
IR40	5	6	5	3	2	1	2	23
IR42	3	4	5	3	2	3	4	24
IR43	3	7	7	4	2	3	3	29
IR44	3	5	3	3	2	3	4	23
IR45	4	7	6	5	2	3	4	33
IR46	4	3	5	6	2	5	3	28
IR48	4	7	5	6	0	5	5	32
IR50	4	5	3	4	0	3	3	22

1) Relatively tolerant to adverse soil stresses are IR 22, 24, 36, 38, 40, 42, 44 and 50

Since most of the small farmers of South and Southeast Asia as well as in Africa can not afford fertilizers (N, P, K, Ca) in the amount and management required to prevent iron toxicity, a combination of tolerant variety and repeated manuring (ashes, compost, pig and green manure as basal dressing) may be recommended to face these multiple stresses. Particularly in iron toxic swamps, high doses of nitrogen, phosphorus, potassium and lime are probably necessary to eliminate iron toxicity, which is an impossible task for the average farmer. Even if some fertilizers are at hand, iron toxic soils need repeated treatments (approximately 3 to 5 times) before any significant improvement may be recorded. The higher the amount of nitrogen applied, the more P, K and Ca will be required to obtain a well balanced physiological state of the rice plant. Finally, a NPK treatment may still provide only limited yield increases unless the lack of zinc is corrected simultaneously. This, however, can be easily achieved by dipping seedlings overnight in a 2% suspension of zinc oxide or by incorporating into the soil surface about 5 – 100 kg zinc sulfate/ha/season (Castro, 1977; Rosales, 1979).

Summary

Appropriate management and treatment of iron toxicity can be recommended only if the cause of the responsible stress has been well recognized. In order to determine the soil stresses that are shared by iron toxic soils, samples were collected from various sites in Southeast Asia and Africa and compared by a wide range of physico-chemical analyses (texture, pH, CEC, exchangeable Na, K, Mg, Ca, available Zn, Mn_t, Fe_t, Fe_o). Rice leaves clearly showing "bronzing" were investigated for their content in N, P, K, Mg, Ca, Na, Zn, Mn and Fe. Both from field observations as well as from analytical results the following conclusions are drawn:

(1) Most iron toxic soils occur in small inland valleys, usually with upwelling and/or lateral seepage. Further, nutritionally poor swampy or acid sulfate soils usually develop iron toxicity.
(2) The general characteristics shared by the soils examined are a relatively low CEC, an extremely small amount of exchangeable K and Ca (in most cases) as well as a deficiency in available P (Olsen). The pH (H_2O) of soils varied greatly (pH 3 – 7.2) as did the Fe_o-content, suggesting that neither the acidity nor the amount of "active" Fe should be regarded as an essential prerequisite for iron toxicity.
(3) The low supply of nutrients was clearly reflected by the mineral composition of the leaves. Except for an overbalanced content of Fe (290 to 1000 ppm) and Mn (partly more than 1000 ppm), all symptoms bearing leaves revealed low or deficient amounts of K and P and often of Ca and Zn.

Iron toxicity is ascribed to multiple nutritional soil stress (insufficient supply of K, P, Ca and perhaps Zn) rather than to a low pH or high amount of easily reducible Fe. This hypothesis was confirmed by a greenhouse experiment using an iron toxic soil translocated from Panay. The results obtained (with IR 26 and IR 42) indeed showed that the disorder may be prevented by a combined supply of K, P and Ca. In the presence of these nutrients root exudation decreased as a result of an improved selective permeability of the membranes and an effective Fe-excluding mechanism. Practical implications of these results are discussed.

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