Iron toxicity in rice—conditions and management concepts

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Summary—Zusammenfassung

Iron toxicity is a syndrome of disorder associated with large concentrations of reduced iron (Fe^{2+}) in the soil solution. It only occurs in flooded soils and hence affects primarily the production of lowland rice. The appearance of iron toxicity symptoms in rice involves an excessive uptake of Fe^{2+} by the rice roots and its acropetal translocation into the leaves where an elevated production of toxic oxygen radicals can damage cell structural components and impair physiological processes. The typical visual symptom associated with these processes is the "bronzing" of the rice leaves and substantial associated yield losses.

The circumstances of iron toxicity are quite well established. Thus, the geochemistry, soil microbial processes, and the physiological effects of Fe^{2+} within the plant or cell are documented in a number of reviews and book chapters. However, despite our current knowledge of the processes and mechanisms involved, iron toxicity remains an important constraint to rice production, and together with Zn deficiency, it is the most commonly observed micronutrient disorder in wetland rice. Reported yield losses in farmers' fields usually range between 15% and 30%, but can also reach the level of complete crop failure.

A range of agronomic management interventions have been advocated to reduce the Fe^{2+} concentration in the soil or to foster the rice plants' ability to cope with excess iron in either soil or the plant. In addition, the available rice germplasm contains numerous accessions and cultivars which are reportedly tolerant to excess Fe^{2+}. However, none of those options is universally applicable or efficient under the diverse environmental conditions where Fe toxicity is expressed. Based on the available literature, this paper categorizes iron-toxic environments, the steps involved in toxicity expression in rice, and the current knowledge of crop adaptation mechanisms in view of establishing a conceptual framework for future constraint analysis, research approaches, and the targeting of technical options.

Key words: avoidance / ferrous iron / leaf bronzing / *Oryza sativa* / plant adaptation / tolerance

Eisentoxizität in Reis – Bedingungen und Managementkonzepte

Eisentoxizität ist ein multiples Stresssyndrom, das mit hohen Konzentrationen reduzierten Eisens (Fe^{2+}) in der Bodenlösung einhergeht. Da es nur in überstauten Böden auftritt, beeinträchtigt es in erster Linie die Erzeugung von Nassreis. Das Auftreten von Eisentoxizität setzt eine übermäßige Aufnahme von Fe^{2+} durch die Reiswurzel und dessen akropetalen Transport in die Blätter voraus. Dort bedingen die zweiwertigen Eisenionen die Bildung von toxischen Radikalen, die strukturelle Zellbestandteile zerstören und physiologische Prozesse beeinträchtigen können. Das typischerweise damit verbundene makroskopische Symptombild ist eine Kupferfärbung der Blätter („bronzing"). Damit verbunden sind erhebliche Ertragseinbußen.

Die Bedingungen, die zum Auftreten von Eisentoxizität führen, sind relativ gut untersucht. So sind zum Beispiel die Geochemie, bodenmikrobiologische Prozesse und die physiologischen Effekte von Fe^{2+} in Geweben und Zellen in zahlreichen Artikeln dokumentiert. Allerdings bleibt die Eisentoxizität trotz unseres derzeitigen Wissenstandes auch weiterhin ein entscheidender Begrenzungsfaktor für den Reisanbau in zahlreichen tropischen Gebieten und stellt neben Zinkmangel das wichtigste Mikronährstoffungleichgewicht im überstauten Nassreis dar. Ertragsverluste belaufen sich in der Regel auf 15–30 %, allerdings kann es bei massiver Toxizität auch zu einem totalen Ernteausfall kommen.

Diverse pflanzenbauliche Maßnahmen verringern den Fe^{2+}-Gehalt der Bodenlösung oder stärken die Abwehrmechanismen von Reis gegenüber Fe^{2+}. Ferner liegen zahlreiche Sorten und Zuchtlinien vor, die eine gewisse Toleranz gegenüber erhöhten Fe-Gehalten aufweisen. Allerdings reichen offensichtlich weder die Anbaumaßnahmen noch die Sortenwahl aus, um die schädigende Wirkung von Fe^{2+} an den unterschiedlichen eisentoxischen Standorten und Klima- und Anbausituationen zu kompensieren. Auf der Grundlage der publizierten Fachliteratur kategorisiert der vorliegende Beitrag die Bedingungen, unter denen Eisentoxizität auftritt, die Wirkungsweise von Fe^{2+} und die Symptomausprägung an Nassreis sowie die derzeit bekannten agronomischen Managementoptionen und pflanzlichen Anpassungsmechanismen. Ziel dieser Übersichtsarbeit ist die Erarbeitung eines Konzepts für die zukünftige Problemanalyse und Schwerpunktsetzung in der Forschung sowie den zielgerichteten Einsatz von Technologien für ein verbessertes Management von Eisentoxizität im tropischen Nassreisanbau.

1 Introduction

Lowland rice is being cultivated on approximately 128 million hectares of irrigated and rainfed land (*Maclean* et al., 2002). As many as 100 million hectares show some sort of nutritional constraint to rice growth caused by either nutrient deficiencies or toxicities (*Brady*, 1982). Among the toxicities, iron toxicity is well-recognized as the most widely distributed nutritional disorder in lowland-rice production (*Dobermann* and *Fairhurst*, 2000). On acid soils, it is one of the most important constraints to rice production, and together with Zn deficiency, it is the most commonly observed micronutrient disorder in wetland rice (*Neue* et al., 1998).

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Iron toxicity is a syndrome of disorder associated with large concentrations of Fe^{2+} in the soil solution. Although most mineral soils are rich in iron, the expression of toxicity symptoms in leaf tissues and a reduction in rice yield occur only under specific flooded conditions, which involve the microbial reduction of insoluble Fe(III) into soluble Fe(II) (*Ponnamperuma*, 1972). A wide range of soil types can be iron-toxic, including acid sulfate soils (*Tinh*, 1999), acid clay soils (*Alaily*, 1998), peat soils (*Deturck*, 1994), and valley-bottom soils receiving interflow water from adjacent slopes (*Sahrawat* and *Diatta*, 1995). The Fe^{2+} concentrations in the soil solution that reportedly affect lowland-rice yields can range from 10 to >2000 mg L^{-1}. Iron-induced yield reduction is frequently associated with a poor nutrient status of the soil (*Benckiser* et al., 1983) or with accumulation of respiration inhibitors (*Tanaka* et al., 1966). Hence, iron toxicity may be described as a multiple nutritional disorder hastened by, but also increasing conditions of P, K, and Zn deficiency and H_2S toxicity (*Ottow* et al., 1983).

The expression of iron-toxicity symptoms in rice requires excessive uptake of Fe^{2+} by roots and its acropetal translocation *via* xylem flow into the leaves. Inside the leaf, excess amounts of Fe^{2+} cause an elevated production of radicals which can irreversibly damage cell structural components (*Thompson* and *Ledge*, 1987) and lead to an accumulation of oxidized polyphenols (*Yamauchi* and *Peng*, 1993). The typical visual symptom associated with those processes is the "bronzing" of the rice leaves (*Howeler*, 1973). Rice-yield losses associated with the appearance of bronzing symptoms commonly range from 15% to 30%. However, in the case of severe toxicity, complete crop failure can occur (*Audebert* and *Sahrawat*, 2000). Overcoming rice-yield reductions caused by iron toxicity requires both adapted and tolerant varieties as well as appropriate management interventions.

Various water-, crop-, and nutrient-management options have been reported to alleviate the negative effects of Fe toxicity on lowland-rice performance. The most prominent and easily adoptable strategy to address Fe toxicity at field level is the development and use of tolerant rice germplasm. Breeders have proposed a wide array of genotypes with various degrees of adaptation. However, depending on the region, the soil type, the cropping season, and the severity and duration of Fe-toxicity occurrence, genotypes strongly differ in their response patterns and their ability to cope with excess amounts of Fe^{2+}. As a result, "adapted" cultivars frequently succomb to iron toxicity, and agronomic intervention strategies are often not adoptable and may not always produce the desired effect. A systematic approach and a classification of Fe-toxicity types and the required adaptation mechanisms in rice genotypes need to be matched with agronomic management interventions to efficiently address the iron-toxicity problem at field level. The following sections discuss the processes and the conditions involved in the occurrence of Fe toxicity and propose a step-wise approach for intervention strategies in view of shaping the agenda of future iron-toxicity research.

2 Iron in soil

2.1 Conditions for Fe reduction

Iron toxicity is a syndrome expressed in rice plants that is associated with an excessive amount of Fe^{2+} in the soil. While it may occur in a wide range of soil types, general characteristics shared by most Fe-toxic soils are high amounts of reducible Fe, low pH, and low CEC and exchangeable K content (*Ottow* et al., *1982*). These may be associated with P and Zn deficiency and H_2S toxicity (*Kirk*, 2004). Most importantly, Fe toxicity is linked to water logging and only occurs under anoxic soil conditions (*Ponnamperuma* et al., 1967). Paddy rice soils are subjected to periodic changes between oxic and anoxic conditions. Since oxygen diffuses in air about 10^3–10^4 times faster than in water or in water-saturated soils (*Armstrong*, 1979), oxygen is depleted rapidly by the respiration of soil microorganisms and plant roots in waterlogged soils (*Prade* et al., 1990). With the depletion in oxygen, NO_3^-, Mn^{4+}, Fe^{3+}, and SO_4^{2-} can act as electron acceptors for microbial respiration and sequentially become reduced in a flooded rice field. Oxygen and nitrate are used within the first hours or days of inundation. The subsequent reduction of Mn^{4+} also occurs rapidly, since the Mn content of soils is usually low (*Ponnamperuma*, 1977). Within a few days after inundation or at a redox potential of <180–150 mV, the reduction of Fe^{3+} begins (*Patrick* and *Reddy*, 1978).

The amount of extractable Fe^{2+} increases with the quantity of decomposable organic matter, the temperature, and the amount of available redox buffers (*Ponnamperuma* et al., 1967). It is enhanced by a low initial soil pH, a sustained supply of organic matter (*Prade*, 1987), and the absence of compounds with a higher oxidation state than Fe(III)-oxide (*Ponnamperuma*, 1972). It increases with the duration of submergence, potentially reaching peak values 2–8 weeks after soil flooding (*Swarup*, 1988; *Patra* and *Mohany*, 1994) and remains constant thereafter (*Sadana* et al., 1995). Before the harvest of rice, fields are usually drained which results in a re-oxidation (detoxification) of Fe^{2+} to Fe^{3+} (*Sahrawat*, 1979). This re-oxidation has been shown to be either an enzymatic process, involving aerobic microorganisms (*Emerson* and *Moyer*, 1997; *Blake* et al., 1993) or an anaerobic microbial Fe^{2+} oxidation, involving both phototrophic and nitrate-reducing bacteria (*Straub* et al., 1996). Other than that, a strictly chemical (nonenzymatic) oxidation can occur in the presence of molecular oxygen or of Mn(IV) oxide and nitrite (*Schwertmann*, 1985).

2.2 Distribution of Fe in soils

Apart from the seasonal changes due to the rainfall pattern (rainfed rice) or drainage and irrigation (irrigated rice), changes in Eh and Fe^{2+} concentrations are found on a small scale horizontally in the soil profile and vertically between the bulk soil and the rhizosphere (*Howler* and *Bouldin*, 1971). Three compartments have been differentiated and include a thin oxic surface layer, the reduced puddled bulk soil and the oxic rhizosphere soil and rhizoplane (*Liesack* et al., 2000). The horizontal distribution of Eh and reduced Fe in the profile has been described by *Ratering* and *Schnell* (2000). The extent

of the oxic surface layer can vary between 2 and 10 mm and is partially determined by a nitrate-dependent microbial re-oxidation of Fe^{2+}. Highest Fe^{2+} concentrations are found at 2–15 cm soil depth. They can again decline in deeper layers or below the plough pan where the soil contains less organic matter than in the puddled soil layer (*Revsbech* et al., 1980). Horizontal variations in Fe^{2+} are linked to the oxic rhizosphere soil, which is the result of oxygen release from rice roots (*Yamanouchi* et al., 1989). Its extent is determined by the formation of aerenchyma (oxidation power of the rice root) and the root density (*Frenzel* and *Bosse*, 1999).

While the rhizosphere of rice is a potential site of Fe^{2+} oxidation, it can also act as a site of Fe reduction (*Prade*, 1987). Facultative anaerobic chemo-organotrophic microorganisms (*e.g.*, *Geobacter, Pseudomonas*, *Clostridium*, or *Bacillus* sp.*)* play a key role in the reduction and mobilization of Fe-oxides (*Munch* and *Ottow*, 1977; *Trolldenier*, 1988). Some fungi are also hypothesized to be capable of enzymatically reducing Fe(III)-oxides (*Schwertmann*, 1985). As the use of oxidized Fe as an alternative electron acceptor for respiration requires energy-rich electron donors (*e.g.*, easily mineralizable organic root exudates), the abundance of iron-reducing microorganisms can be higher in the rhizosphere than in the bulk soil (*Benckiser* et al., 1983; *Wang* and *Liu*, 1992). However, processes of iron re-oxidation dominate over the Fe reduction in the rhizosphere of most *indica*-type rices, leading to a considerable Fe^{3+} accumulation and the formation of iron plaque around rice roots (*Kirk* et al., 1990). The distribution of reduced iron in a paddy soil profile is conceptually depicted in Fig. 1.

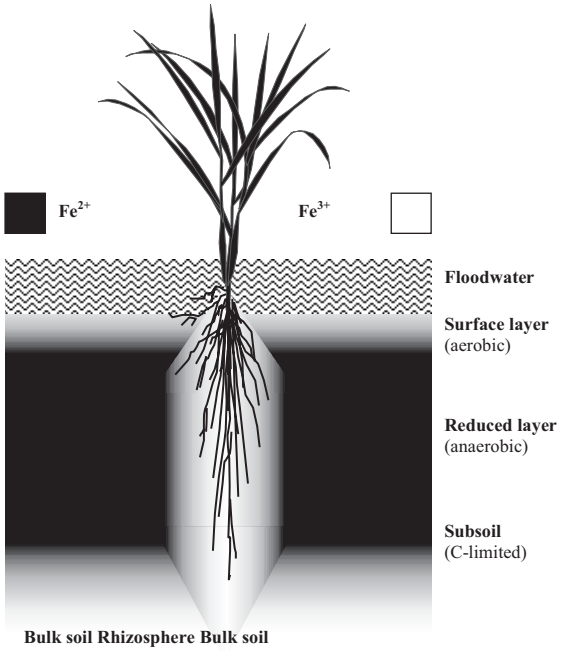

Figure 1: Conceptual horizontal and vertical distribution of reduced iron (Fe^{2+}) in a paddy soil.
Abbildung 1: Konzeptionelle Darstellung der horizontalen und vertikalen Verteilung von Fe^{2+} im Nassreisboden.

2.3 Conditions enhancing iron toxicity

The severity of iron-toxicity expression in rice has been related to a number of soil factors. These involve most prominently (1) the content and type of the clay minerals, (2) the amount of exchangeable soil Fe, (3) the soil pH, and (4) the presence of "stress factors". The concentration of soil Fe^{2+} is reportedly less in clay than in sandy soils (*Das* et al., 1997). Clay was found to control the content and distribution of iron in both Alfisols and Vertisols (*Rajkumar* et al., 1997). Clay content affected the Fe dynamics primarily *via* Fe retention on clay-mineral surfaces (cation-exchange capacity—CEC), explaining why in iron-toxic lowland soils with kaolinite (low CEC) as predominant clay mineral, toxicity symptoms in rice occur more frequently than in those having predominantly smectites (high CEC) (*Prade* et al., 1990). High amounts of soluble Fe^{2+} (100–1000 mg L^{-1}) may be found in acid soils (*Ponnamperuma*, 1972). In acid sulfate soils, concentrations of up to 5000 mg kg^{-1} Fe have been reported (*Harmsen* and *Van Breemen*, 1975). However, toxicity symptoms reportedly also occur in near neutral soils (*Tadano*, 1976) and not in all acid sulfate soils rice expresses toxicity symptoms (*Tinh* et al., 2000). A soil solution concentration of 300 mg water-soluble Fe L^{-1} is generally considered the critical limit for the cultivation of lowland rice (*Lantin* and *Neue*, 1989). However, depending on the site and the cultivars used, reported critical concentrations can range from 20 to 2500 mg kg^{-1}, indicating that factors other than pH and Fe concentration influence the occurrence of Fe toxicity symptoms. Such factors may include the accumulation of hydrogen sulfide and of organic acids (*Tadano* and *Yoshida*, 1978) and the availability of (nutrient) elements (*Ottow* et al., 1982). Sulfide acts as a respiratory inhibitor and strongly affects root metabolism (*Tanaka* et al., 1966). Organic acids may exert their toxic effects by chelating iron and thus increasing its plant availability (*Marschner*, 1995). As plants develop, the toxic effect of both respiratory inhibitors and organic acids decreases (*Jaqc*, 1977). *Ottow* et al. (1982) described iron toxicity as a multiple nutritional disorder, which also involves other (nutrient) elements. Their chemical availability can be affected as a result of iron reduction (P, Mo, Al), or their uptake by plants can be impaired as a result of iron accumulation in the rhizosphere (K, Zn, Mn).

In the process of the reduction of Fe(III)-oxides, essential nutrients such as P and to a lesser extent Mo can be released (*Ponnamperuma*, 1977), but the availability of toxic aluminum may also increase (*Fischer*, 1983). Consequently, large quantities of Al^{3+} can also be taken up along with iron by the rice plant (*Prade*, 1987). The amount of water-soluble P in the soil is related to the reduction of iron (*Navarro* et al., 1988a; *Krairapanond* et al., 1993) and increases with declining Eh (*Satawathananont* et al., 1991). In the absence of mineral-fertilizer-P application, the P released from iron phosphates upon Fe^{3+} reduction is the dominant source of P for rice (*Liao* et al., 1994). There is also some evidence that P- and K-deficient plants exude elevated amounts of organic metabolites, thereby stimulating the microbial reduction of iron and the subsequent P release (*Prade*, 1987).

Apart from increasing the availability of P, the Fe^{2+} formed by the reduction of Fe oxides shows antagonistic interactions with other cationic nutrients, mainly manganese, zinc, and potassium. Consequently, with increasing Fe concentrations, Mn, Zn, and K deficiencies are likely to occur (*Fageria*, 1988). Thus, large amounts of reduced Fe adversely affected the uptake of Mn. *Vice versa*, Mn application can suppress iron desorption, delay the reduction of Fe oxides, and subsequently reduce Fe uptake by rice (*Tanaka* and *Navasero*, 1966; *Howeler*, 1973). Iron oxides are known to have a strong zinc-binding capacity. When soils are flooded, Zn becomes available in the process of iron oxide reduction (*Mandal* et al., 1992). On the other hand, reduced Fe can also exert a direct antagonistic effect on Zn uptake (*Sajwan* and *Lindsay*, 1988). Furthermore, the plaque formation resulting from Fe re-oxidation around the rice root can reduce the concentration of soluble Zn in the rhizosphere by forming sparingly soluble $ZnFe_2O_4$ (*Sajwan* and *Lindsay*, 1988). When the iron plaque exceeds 25 g (kg root dry weight)$^{-1}$, the plaque becomes a physical barrier for the uptake of Zn (*Zhang* et al., 1998) and of Mn (*Wang* and *Shuman*, 1994). Similar to Zn, the uptake of K is affected by excess Fe^{2+} in the soil solution (*Jugsujinda* and *Patrick*, 1993). A large body of literature supports the ameliorative effects of fertilizer (mainly P, K, and Zn) application on the performance of lowland rice grown under iron-toxic conditions. Given the large diversity of soil and environmental conditions that do affect the rate of reduction and the amount of Fe^{2+} in the soil and the time of occurrence and severity of Fe stress, a systematic categorization of iron-toxic environments is required to improve the targeting of intervention strategies.

2.4 Typology of iron-toxic environments

There is a large variability within the published literature on iron toxicity with regard to soil Fe content (20–5000 mg kg^{-1}), leaf Fe concentrations (300–2000 mg kg^{-1}), time of toxicity occurrence (from 2 weeks after soil flooding or rice transplanting to the late reproductive phase), the distribution of toxicity symptoms in the field (whole plot, patches, isolated plants), and the observed yield losses (10%–100%). An analysis across these factors and site descriptors allows to separate the information into three clusters of conditions associated with iron toxicity (Tab. 1).

Cluster 1 refers to acid sulfate soils, in which extremely large concentrations of Fe^{2+} in the soil solution arise as a result of the soils' peculiar mineralogy. The soil Fe^{2+} can range from 500 to over 5000 mg kg^{-1}, and tissue concentrations are generally between 500 and 2000 mg kg^{-1}. The symptoms on plants can occur throughout the rice-growing period, and yield losses reportedly range from 40%–100%. Plants suffering from iron toxicity may cover large contiguous areas such as in the Mekong Delta in Vietnam or in the coastal plains of West Africa (*e.g.*, Liberia, Sierra Leone, Senegal) and of Thailand. **Cluster 2** is associated with more clayey acid, iron-rich soils in sediments derived from highly weathered soils. Iron concentrations of 300–1000 mg kg^{-1} occur usually one month or more after transplanting, and tissue Fe concentrations range from 300–800 mg kg^{-1}. Symptoms on rice appear during the late vegetative growth stage, and yield losses rarely exceed 30%. However, yield losses of 35%–50% can occur when rice is grown during the dry season. In this situa-

Table 1: Characteristics of site and crop parameters of major iron-toxic environments.
Tabelle 1: Bestimmungsgrößen und Merkmale der wichtigsten Kategorien von eisentoxischen Standorten.

	Cluster 1	**Cluster 2**	**Cluster 3**
Land form	Coastal planes, river deltas	Marches, highland swamps	Inland valley swamps
Soil	Young acid sulfate soils	Clayey Ulti- and Histosols	Sandy valley-bottom soils
Soil iron (mg Fe^{2+} L^{-1})	500–2500	300–900	20–600
Leaf tissue iron (mg (kg dry matter)$^{-1}$)	500–2000	300–800	100–1500
Cropping season (most symptoms)	Dry and wet season	Mainly dry season	Mainly wet season
Rice growth stage (most symptoms)	Seedling to maturity stages	Late vegetative to reproductive stages	Early to late vegetative stages
Spatial distribution of symptoms	Whole field	Isolated patches (org. matter, H_2S)	Valley fringe (zone of upwelling)
Maximum yield losses (%)	40–100	15–50	30–70
Country examples	Vietnam (MD), Liberia, Senegal, Thailand	Philippines, Indonesia (Java), Burundi, Madagascar	Guinea, Madagascar, Côte d'Ivoire, SriLanka

Based on: *Abifarin* (1988, 1990), *Abu* et al. (1989), *Alaily* et al. (1998), *Audebert* and *Sahrawat* (2000), *Ayotade* (1979), *Baggie* and *Bah* (2001), *Balasubramanian* et al. (1995), *Baruah* and *Nath* (1996), *Benckiser* et al. (1982, 1983), *Das* et al. (1997), *De Giudici* (1991), *Deturck* (1994), *Genon* et al. (1995), *Gunatilaka* (1994), *Howeler* (1973), *Jugsujinda* and *Patrick* (1993), *Kirk* (2004), *Lantin* and *Neue* (1989), *Luo* et al. (1997), *Mahadevappa* et al. (1991), *Makarim* et al. (1989), *Mathew* and *Sankaran* (1993), *Mitra* et al. (1993), *Moore* et al. (1990), *Nguyen* (1991), *Olumo* et al. (1973), *Ottow* et al. (1993), *Patra* and *Mohany* (1994), *Prade* et al. (1993), *Rajkumar* et al. (1997), *Rathaiah* et al. (1988), *Sadana* et al. (1995), *Sahrawat* (1979, 2000), *Sahrawat* and *Diatta* (1995), *Sahrawat* et al. (1996), *Singh* et al. (1992, 1993), *Van Breemen* and *Moormann* (1978), *Virmani* (1977), *Vizier* et al. (1990), *WARDA* (2002), *Winslow* et al. (1989), *Yamauchi* (1989).

tion, plants suffering from Fe toxicity are found in localized spots within a landscape or a field (*e.g.*, The Philippines, Java) and are frequently associated with an extremely low redox potential, relatively high organic-matter content (*e.g.*, peat soils in Burundi and Madagascar), and high concentrations of respiration inhibitors (*e.g.*, H_2S). **Cluster 3** refers to poorly drained sandy soils in valleys receiving interflow water from adjacent slopes with highly weathered sediments. The lowland soils tend to be kaolinitic with a low CEC and little available P. The concentrations of Fe^{2+} range from 20 to 600 mg kg^{-1} and are largely restricted to the wet season (interflow coincides with the onset of the rainy season). Leaf tissue Fe at harvest is highly variable, but often below the supposedly critical level of 300 mg kg^{-1}. Symptoms usually occur very early in the rice plant's development and are associated with the onset of interflow from the slopes. The occurrence of toxicity symptoms is patchy and localized in the zone of upwelling of the interflow in the valley fringe. As toxicity symptoms may be observed at relatively low soil Fe^{2+} concentration and are frequently associated with a relatively low tissue Fe content, it has been hypothesized that interflow is mainly aggravating a low level of *in-situ* toxicity by mechanisms other than bringing in Fe^{2+}, possibly involving the depletion of nutrients and upsetting the rice plants' ability to exclude Fe^{2+} (*Kirk*, 2004). Yield losses can range from 30% to 70%. However, when the peak of iron interflow coincides with the transplanting of rice, severe toxicity in seedling leaves can result in complete crop failure. This situation is prototypic for the highland valleys in Madagascar and Sri Lanka as well as for much of the inland valley landscape in the humid zone of West Africa.

In terms of rice-growing area, the situations described by Clusters 1 and 3 are the most important. Thus, environments where Fe toxicity is associated with interflow reportedly affect some 5 Mha globally, comprising an estimated 70% of the area in the inland valley swamps in the humid zone of West Africa and much of the highlands in Madagascar. Rice is grown on an estimated 4 Mha of acid sulfate soils, much of it in the Mekong Delta and in coastal areas of Africa (Senegal, the Gambia, Guinea Bissau, Sierra Leone, Liberia), Southeast Asia (Indonesia, Thailand), and South America (Venezuela, the Guyanas) (*FAO*, 2005).

3 Iron in the plant

3.1 Uptake and transport of iron

Rice plants have the tendency of taking up more iron than most other plants, and Fe^{2+} is the iron species prevailing in paddy fields. Since Fe^{2+} is easily taken up, the uptake mechanism of Fe^{2+} is probably of less importance in flooded environments (*Mengel*, 1972). After uptake into the root cortex, the reduced Fe^{2+} can enter the xylem after a symplastic passage through the Casparian strip. However, a large share of Fe^{2+} may enter the xylem directly *via* an apoplastic bypass (shown for sodium by *Yeo* et al., 1987; *Tsuchiya* et al., 1995; *Asch*, 1997) or after root injury resulting from the pulling and transplanting of the seedling. In the xylem, Fe^{2+} follows the transpiration stream in the acropetal long-distance transport. This is in contrast to the iron transport in upland crops or in plants that do not grow under iron-toxic conditions, where the long distance transport is dominated by Fe^{3+} complexes with citrate (*Clark* et al., 1973; *Schmidt*, 1999) or peptide carbohydrate compounds (*Höfner*, 1970). Reaching the leaf apoplastic space, Fe may re-enter the symplast. The exact mechanism by which Fe is taken up by leaf cells is not yet well understood. In rice, this uptake is likely to occur in the form of Fe^{2+}, being the physiologically active form of iron in the symplast (*Machold* et al., 1968).

Within the cell, excess amounts of Fe^{2+} may catalyze the generation of active oxygen species such as superoxide, hydroxyl-radical, and H_2O_2 as follows: $O_2 + Fe^{2+} \rightarrow O^{2-} + Fe^{3+}$ or $H_2O_2 + Fe^{2+} \rightarrow {}^{-}Fe^{3+} + OH^{-} + OH^{\circ}$ (*Marschner*, 1995). These reactions are greatly enhanced when iron is abundant, and iron itself can be part of highly reactive perferryl-radicals (*Halliwell* and *Gutteridge*, 1984), or in combination with fatty acids, Fe^{2+} can form peroxy fatty acids (*Peterson*, 1991). Free radicals are responsible for the damage caused by iron toxicity (*Thongbai* and *Goodman*, 2000). They irreversibly damaged membrane lipids (*Thompson* and *Legge*, 1987), proteins (*Chevrier* et al., 1988; *Miyao* et al., 1995), and nucleic acids (*Elstner*, 1982) and affect the membrane charge (*Vladimirov*, 1980). The activity of phenol oxidases increases and oxidized polyphenols can accumulate (*Yamauchi* and *Peng*, 1993). Free radicals will eventually oxidize chlorophyll and subsequently lead to a decrease in chlorophyll content (*Monteiro* and *Winterbourn*, 1988).

3.2 Symptoms of iron toxicity

The typical visual symptom associated with above described processes, and particularly with the accumulation of oxidized polyphenols, is the "bronzing" of the rice leaves. The bronzing symptoms start in fully developed older source leaves with the occurrence of tiny brown spots that spread from the leaf tip to the base. In the further development of the symptom, the leaf tips become orange-yellow and dry up in some rice varieties. These symptoms are particularly developed in older leaves having higher transpiration rates (*Yamanouchi* and *Yoshida*, 1981). Eventually, the entire transpiring leaf becomes orange to rusty brown, or purple brown when toxicity is extremely severe (*Fairhurst* and *Witt*, 2002).

These symptoms can occur at different growth stages and may affect rice at the seedling stage, during the vegetative growth, and at the early and late reproductive stages. Depending on the growth stage leaf bronzing occurs, other symptoms and growth effects may be associated. In the case of toxicity occurring during seedling stage, the rice plants remain stunted with extremely limited tillering (*Abraham* and *Pandey*, 1989). Toxicity during the vegetative stages is associated with reduced plant height and dry-matter accumulation (*Abu* et al., 1989), with the shoot being more affected than the root biomass (*Fageria*, 1988). Both the tiller formation and the share of productive tillers can be severely reduced (*Cheema* et al., 1990). When iron toxicity occurs during the late vegetative or early reproductive growth phases, it is associated with fewer panicles per hill (*Singh* et al., 1992), an increase in spikelet sterility (*Virmani*, 1977), and delayed

flowering and maturity by up to 20–25 d. In highly susceptible cultivars, no flowering at all will occur (*Ayotade*, 1979). After booting, root growth stops, and the aerenchyma starts to senesce and decay. As a result, the oxidation power of the root breaks down, and the root surface is coated with dark brown to black precipitates of $Fe(OH)_3$, and many roots die (*Morel* and *Machado*, 1981).

There is some correlation between the severity of iron-toxicity symptom expression and yield. This relationship can vary within the cropping season as well as between seasons and years. Average reported yield losses due to iron toxicity are in the range of 12%–35% (*Lantin* and *Neue*, 1989). However, toxicity at seedling and early vegetative stages can strongly affect plant growth and result in a complete yield loss (*Abifarin*, 1988). Seasonal and interseasonal variation in the relationship between symptom expression and yield loss are mainly related to transpiration and differences in acropetal Fe translocation. Hence, bronzing symptoms and Fe uptake were increased by a high vapor-pressure deficit in a phytotron study (*Kpongor* et al., 2003), and iron-induced yield losses and leaf bronzing were more pronounced in a dry as compared to a wet season crop (*Sahrawat* and *Diatta*, 1995). On the other hand, yield reductions of up to 30% have also been reported to occur without any foliar symptoms (*Abifarin*, 1988).

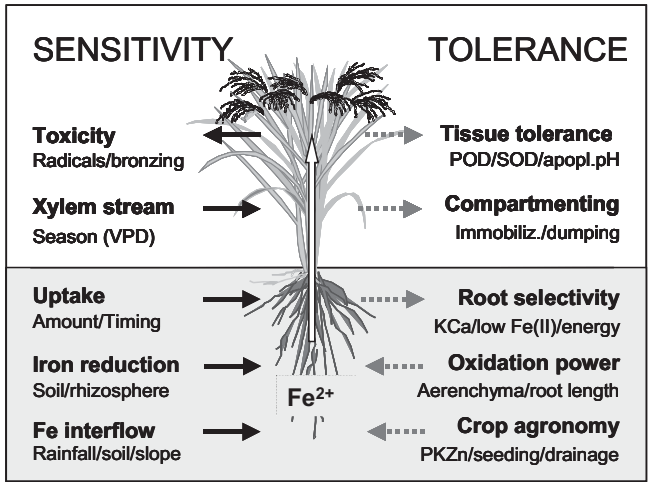

Figure 2: Factors affecting iron-toxicity occurrence and iron tolerance of lowland rice.
Abbildung 2: Bestimmungsfaktoren für das Auftreten von Eisentoxizität und für die Eisentoleranz von Nassreis.

3.3 Adaptation strategies

Evidently, rice plants have developed morphological and physiological avoidance and/or tolerance mechanisms to cope with and survive adverse iron-toxic soil conditions and large amounts of iron in the plant. These mechanisms are important in the selection of tolerant or adapted rice genotypes. Three major types of adaptation strategies can be differentiated and comprise "includer" and "excluder" strategies as well as "avoidance" and "tolerance" mechanisms. Plants employing strategy I (exclusion/avoidance) exclude Fe^{2+} at the root level and hence avoid of Fe^{2+} damage to the shoot tissue (rhizospheric oxidation and root ion selectivity). With strategy II (inclusion/avoidance), Fe^{2+} is taken up into the rice root, but tissue damage may be avoided by either compartmentation (immobilization of active iron in "dumping sites", *e.g.*, old leaves or photosynthetically less active leaf sheath tissue) or exclusion from the symplast (immobilization in the leaf apoplast). Strategy III plants (inclusion/tolerance) actually tolerate elevated levels of Fe^{2+} within leaf cells, probably *via* enzymatic "detoxification" in the symplast. Whereas Fe^{2+} exclusion by oxidation in the rhizosphere and the detoxification of radicals in leaf cells are well established Fe-tolerance mechanisms of rice, the other mechanisms are not yet well understood and to date are not considered in rice breeding or screening for iron tolerance. The factors leading to iron toxicity, and those enhancing the rice crops' ability to cope with elevetaed Fe^{2+} concentrations, are conceptually summarized in Fig. 2.

Strategy Ia: Oxidation of iron at the root surface. Fe^{2+}, formed either *in situ* in the soil or imported *via* interflow, must first pass the oxidation barrier in the rhizosphere before being absorbed by the root. To establish this barrier, molecular oxygen is channeled from the atmosphere through the stems into the roots *via* a gas-conducting tissue or aerenchyma. This aerenchyma forms upon the establishment of anoxic conditions induced by an increased production of ethylene (*Kawase*, 1981). It involves the degeneration of cortex cells and the subsequent formation of large intercellular lumina (*Ando*, 1983). The aerenchyma may occupy 20%–50% of the total root volume of flooded rice (*Armstrong*, 1979), whereby genotypic differences in the extent of aerenchyma formation appear to be correlated with the rate of ethylene formation in root tissues (*Blake* and *Reid*, 1981). The rate of iron exclusion by oxidation in the root zone *via* the aerenchyma depends on the phenological stage of the rice plant and on the growth stage of the root system. Young secondary roots and root tips diffuse oxygen at the highest rates into the rhizosphere (*Chen* et al., 1980a). At these growing zones, large quantities of Fe^{2+} can be microbially or chemically oxidized, resulting in the formation and accumulation of immobile $Fe(OH)_3$ deposits or iron plaque. Iron plaque was calculated to amount to some 500 kg FeOOH ha^{-1} in a crop of the wetland rice cultivar "Brazos", amounting to as much as 10% of the total root dry weight (*Chen* et al., 1980b). The formation of iron plaque on rice roots is not only reducing the Fe^{2+} concentration in the soil solution, but is also thought to form a physical barrier for further influx of reduced iron (*Tanaka* et al., 1966). Flood-/ethylene-induced aerenchyma formation starts in 2–4-week-old plants, and the oxidation power of the root is highest at the maximum tillering stage (*Tadano*, 1975). As roots senesce, the aerenchyma starts to disintegrate, thus losing its capacity for gas transport, and little Fe oxidation occurs in the root zone after the flowering stage of rice. Consequently, late-season Fe-toxicity symptoms in flag leaves of rice, grown in an acid sulfate soil were primarily associated with a breakdown of the root oxidation power (*Tinh*, 1999). Given the importance of the oxidation power of the root in excluding iron, vigorous and early development as well as longevity of aerenchyma are desired traits for Fe tolerance (*Jayawardena* et al., 1977).

Strategy Ib: Root membrane selectivity. Reduced iron having passed the oxidative barrier of the rhizosphere enters the root apoplast. In plants with an undamaged root system, xylem loading requires ions to pass the root cell membranes at the endodermis due to the Casparian strip. Reduced iron can be excluded at the root cell membranes (*Tadano*, 1976), explaining the Fe^{3+} deposition in the apoplast of root parenchymatic cells observed by *Green* and *Etherington* (1977). Xylem-sap analysis of 2-months-old rice plants indicated that up to 87% of the Fe reaching the root apoplast by mass flow was not detected in the xylem and must hence have been "excluded" at the endodermal barrier between cortex and stele (*Yamanouchi* and *Yoshida*, 1981). There was no such reduction found at the seedling stage, and reduction levels remained low throughout the early growth stages of rice (*Tadano* and *Yoshida*, 1978). Iron exclusion by root-cell membranes is strongly affected by respiration inhibitors, indicating an active and probably energy-consuming metabolic process (*Tadano*, 1975). At concentrations larger than 50 mg Fe^{2+} L^{-1}, this reduction process is first impaired and eventually no longer detectable (*Tadano*, 1976). It is also affected by the rice plants' nutritional status with deficiencies in Ca and K (*Yamanouchi* and *Yoshida*, 1981) and P (*Yoshida*, 1981) reducing the ability of root cells to exclude iron. Given the sensitivity of this exclusion process to environmental conditions, the growth stage of rice and the concentration of Fe^{2+}, this process is unlikely to play a significant role under conditions of a sustained severe (Cluster 1) or of seedling toxicity (Cluster 3).

Strategy IIa: Retention in root and stem tissues. The Fe^{2+} that has entered the xylem stream will follow the transpiration-driven acropetal long-distance transport. However, some of this iron may be immobilized and deposited at specific "dumping sites" within the plant. Some "metabolic inactive" Fe has been found inside the root tissue (*Tanaka* et al., 1966). The rice plants' ability to retain iron inside the root reportedly decreases with plant age (*Tadano*, 1976). During the further acropetal transport, Fe^{2+} may be immobilized and deposited in stem/leaf sheath tissues. Iron-tolerant cultivars from West Africa transported less iron from the roots into the leaf blades, whereas the iron content of the leaf sheath substantially increased (*Audebert* and *Sahrawat*, 2000), possibly involving both immobilization or re-oxidation of Fe^{2+}. This "withdrawal" of active Fe may involve the formation of phytoferritin in the xylem and its subsequent storage in stem tissues (*Seckbach*, 1982; *Smith*, 1984). The efficiency of this process is likely to be determined by the rate of acropetal Fe^{2+} transport and may not be sufficient to prevent iron influx into the leaves under conditions of high transpiration such as during the exponential growth phase of rice or under dry-season conditions when a high vapor-pressure deficit greatly enhances crop transpiration rates (*Asch* et al., 2000, 2003). The effectiveness may also decline when the storage capacity of stem tissues is saturated, which may occur towards the end of the growth cycle of rice, when the crop was continuously exposed to large Fe(II) concentrations and no more leaf tissue is produced. There is, however, no empirical evidence of either genotypic variation or the dynamics of the process in time or during the growth cycle.

Strategy IIb: Retention in the apoplast of the leaf. The apoplastic pH is hypothesized to largely determine the mobility of iron in leaves (*Kosegarten* et al., 1999). An acidic apoplastic pH will favor the uptake of Fe^{2+}. A pH increase in the apoplast reduces the mobility of Fe and possibly favors Fe^{2+} oxidation (studies on grape wine by *Mengel* and *Kosegarten*, 2000, and by *Nicolic* and *Römheld*, 2001). This is associated with the formation of nondiffusible polymers that reduce the activity of a plasma-membrane-bound ferric-chelate reductase (*Schmidt*, 1999; *Lucena*, 2000) and involve specific polysaccharides in the cell wall (*Yamauchi* and *Peng*, 1995). In plants unable to regulate apoplastic pH or reduce the activity of the Fe^{3+} reductase, an uncontrolled accumulation or influx of Fe^{2+} in the leaf cell can occur (*Welch* et al., 1993).

Strategy III: Symplastic tissue tolerance. Once it has entered the symplast, Fe^{2+} will catalyze the generation of active oxygen species and of various radicals (see section 3.1). Prevention of oxidation damage and detoxification of these radicals is vital in alleviating the damage caused by Fe^{2+} and is responsible for tissue tolerance (*Yamanouchi* and *Yoshida*, 1981; *Vose*, 1983). Tolerance mechanisms may involve strong binding or incorporation of Fe^{2+} into symplastic structures that allow controlled oxidation/reduction reactions such as phytoferritin (*Bienfait*, 1985). The extent of phytoferritin formation has been hypothesized to be a possible protection mechanism against high concentrations of Fe^{2+} in the symplast (*Landsberg*, 1996). Radical scavengers such as cytosolic ascorbate (*Hu* et al., 1999) or glutathione (*Thongbai* and *Goodman*, 2000) have also been shown to reduce oxidative stress as induced by Fe^{2+} (*Larson*, 1988; *Thompson* and *Legge*, 1987). Much of the radical scavenging inside the leaf cell of rice, however, is associated with superoxide dismutase (SOD) isoenzymes. As zinc is a component of SOD, Zn application can reportedly increase the iron tolerance of rice while Zn deficiency can produce symptoms that resemble those of iron toxicity (*Ottow* et al., 1982). The activity of SOD results in the formation of hydrogen superoxide. While this H_2O_2 is a less reactive oxidizing agent than the radicals, it reduces the activity of SOD (*Cakmak*, 1988). Hence, to effectively prevent oxidative damage, H_2O_2 needs to be further detoxified by catalases and/or peroxidases (POD) (*Gupta* et al., 1993). The combined SOD-POD enzyme activity has been established to be largely responsible for preventing Fe^{2+}-induced oxidative stress in rice leaves. This mechanism may be of particular importance in situations of seedling toxicity (poorly developed aerenchyma) and especially when rice plants have been injured during transplanting and are exposed to an uncontrolled influx of Fe^{2+} (*Yamauchi* and *Peng*, 1995). The genetic variability of SOD-POD activity in the available gene pool is largely unknown and to date, it has not been used as a screening tool by rice breeders.

4 Management concepts for iron toxicity

Management interventions that reduce the influx of Fe^{2+} or that favor the re-oxidation of Fe^{2+} in the soil may be considered at the scale of the landscape or of the lowland field. The mobility of iron both in the rhizosphere and during the acropetal transport may be reduced by management interventions at plot level or through cultivar selection. Finally, the negative

effects of elevated Fe^{2+} concentrations in leaf tissues may be reduced by the application of mineral nutrients and the development or selection of adapted germplasm. On the basis of the clustering of environmental conditions (section 2.4), the transport and the effects of Fe^{2+} in the plant (section 3.1), and the categories of adaptation strategies (section 3.3), the following section proposes a step-wise analysis of possible intervention strategies at different scales (landscape, plot, plant) and following the pathway of reduced iron from the bulk soil over the rhizosphere and the xylem into the leaf tissue.

4.1 Landscape level

In situations where iron toxicity in the lowland is determined or aggravated by the influx of reduced iron from adjacent slopes (Cluster 3), management options must consider factors that determine the extent of downslope water and solute movement. Such factors may include the amount and intensity of rainfall, the parent material and soil texture of the slopes, physical slope characteristics such as length and steepness, and the land use (Fig. 3).

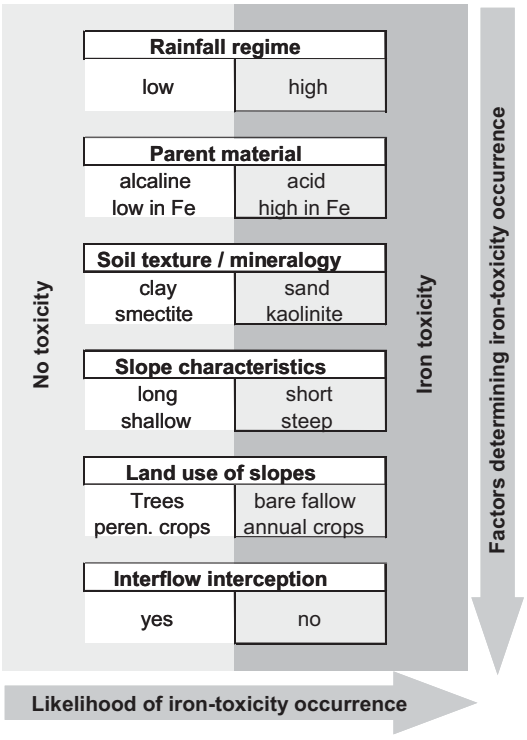

Figure 3: Factors affecting the occurrence of interflow toxicity of iron in lowland rice.
Abbildung 3: Faktoren, die das Auftreten von Eisentoxizität in Nassreis in Zusammenhang mit Hangzugswasser beeinflussen.

In high-rainfall environments, water infiltration and percolation through the soil profile and up-welling of Fe-rich subsurface flow in the hydromorphic valley fringe is likely to be enhanced. Thus, the vast majority of Fe-toxic lowlands in West Africa is located in the high-rainfall humid-forest zone of Guinea, Liberia, Sierra Leone, and Côte d'Ivoire (*WARDA*, 2002). Also the severity of Fe toxicity in highland valleys in Madagascar appears to be more on the high-rainfall eastern than the moderate-rainfall western side of the island (*Vizier* et al., 1990; *De Giudici*, 1991; *Balasubramanian* et al., 1995). Iron toxicity tends to be more at sites where the parent rock is rich in iron (*Abifarin*, 1990; *Zhao*, 1992). Thus, valley of Ulti- and Alfisols derived from gneiss contained larger amounts of total iron than those derived from granite, and hence more iron is potentially mobilized and translocated into the lowland (*Rajkumar* et al., 1997). Soil texture of the valley slope exerts an influence on water movements and the iron dynamics in the soil as well as the soils' buffering capacity for iron (see section 2.3). High infiltration rates of sandy soils on valley slopes increase the occurrence of (interflow) toxicity in lowlands of West Africa (*Van Breemen* and *Moormann*, 1978). The net transfer of Fe in a soil profile and the positioning of the "oxidation front" depends on the bulk density and the gaseous porosity of soils (*Kirk* and *Solivas*, 1994) and is consequently less in clay than in sand. The quantity of interflow iron may further depend on both physical valley characteristics such as the length and steepness of the slopes and by the land use. The interflow rate (share of interflow water from a given slope area) is higher from short than from long slopes (re-infiltration) and greatly enhanced by slope steepness (*Van de Giesen* et al., 2005). A reduction in both the quantity as well as in the solute charge of interflow water was observed with deep-rooting upland vegetation as compared to bare fallow or maize cropping on the slope (*Bognonkpe* and *Becker*, 2003). Several rows of bananas grown in the hydromorphic fringe of a valley in West Africa have reportedly efficiently intercepted interflow water and the nutrients it contained (*Bognonkpe* and *Becker*, 2000). Finally, a highly efficient technical/mechanical interception of interflow occurs with the construction of irrigation or drainage canals on the valley fringe (*Van Breemen* and *Moormann*, 1978) or of a road in the upper hydromorphic zone (*WARDA*, 1991).

4.2 Plot level

Irrespective of the origin of the reduced iron, a broad range of soil-water and crop management interventions that either avoid the build-up of Fe^{2+} or reduce its negative effects on crop growth may be considered at plot level. These include measures that avoid a rapid drop in Eh in both the bulk soil and the rhizosphere, the removal or re-oxidation of Fe in the bulk soil and the rhizosphere, the avoidance of an uncontrolled Fe influx into the root, and the application of mineral nutrients that strengthen the rice plants' defense mechanisms or act as "competing ions" (Fig. 4).

Measures avoiding a rapid drop in Eh: Iron toxicity is associated with situations where the redox potential declines rapidly upon soil flooding. High levels of reducible iron combined with large amounts of organic matter hasten the occurrence and increase the severity of Fe-toxicity symptoms. Thus, during the initial stages of inundation, up to 60%–80% of the easily decomposable organic matter of rice soils is mineralized by microorganisms using Fe^{3+} as electron acceptor (*Patrick*, 1981). In addition, organic acids can form complexes with Fe^{2+} and >40% of the total Fe^{2+} in the soil

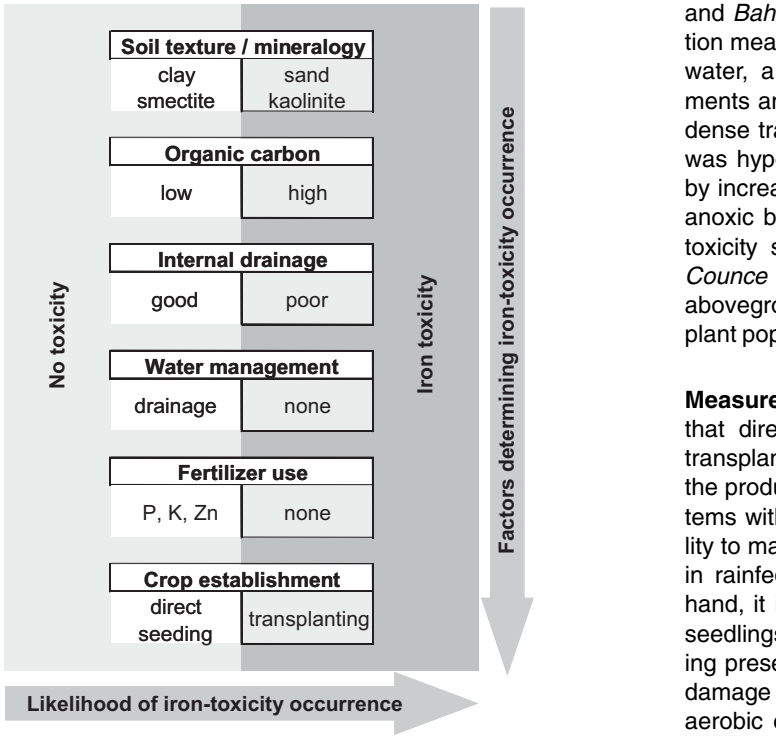

Figure 4: Factors determining iron-toxicity occurrence in lowland rice at plot level.
Abbildung 4: Faktoren, die das Auftreten von Eisentoxizität in Nassreis auf Feldebene beeinflussen.

can be present in chelated form (*Olumo* et al., 1973). Addition of large quantities of easily decomposable organic matter (*e.g.*, green manure) should be avoided, particularly in alluvial clays and acid sulfate soils (Clusters 1 and 2). The metabolic activity of the rhizoflora is regulated by the root membrane, which determines the efflux of organic exudates (*Trolldenier*, 1973). An increased root permeability as a result of K deficiency can lead to an enhanced exudation of low-molecular-weight metabolites, which stimulate the activity of Fe-reducing bacteria in the rhizosphere (*Ottow* et al., 1982). Application of K fertilizer was shown to reduce the uncontrolled exudation of low-molecular-weight substances and hence to limit microbial iron reduction in the rhizosphere (*Prade*, 1987).

Measures enhancing re-oxidation of Fe^{2+}: Various water-, crop-, and nutrient-management options in the field have been reported to alleviate negative effects of Fe toxicity on lowland rice. Flush irrigation reduced the uptake of Fe compared to flood irrigation (*Beyrouty* et al., 1994). Delayed flooding (*Imeokparia*, 1990; *Mathew* and *Sankaran*, 1993) and shortened submergence periods (*Navarro* et al., 1988b) have also been shown to reduce iron toxicity. Mid-season drainage of the field can remove some of the reduced iron together with the floodwater and result in the temporary re-oxidation of the topsoil thus substantially reducing the level of toxic Fe^{2+} and increasing crop yields (*Barbosa* et al., 1983). When such drainage coincides with critical growth stages of rice (*e.g.*, seedling establishment, panicle initiation, flowering), the negative effects of excess Fe on crop yield can be significantly reduced (*Prade*, 1987; *Abu* et al., 1989; *Baggie* and *Bah*, 2001). The timeliness of such drainage and irrigation measures requires a near-perfect control of the irrigation water, a situation usually limited to fully irrigated environments and to the dry-season crop (*Beyrouty* et al., 1994). A dense transplanting of rice with a high root oxidation power was hypothesized to avoid the accumulation of excess iron by increasing the share of the oxic rhizosphere compared to anoxic bulk soil (*Tadano*, 1976). *Ando* (1983) reported less toxicity symptoms on leaves in densely spaced rice, and *Counce* (1987) demonstrated iron concentrations in the aboveground biomass to be reduced with increasing rice-plant population density.

Measures avoiding the influx of Fe^{2+}: There are indications that direct-seeded rice suffers less from Fe toxicity than transplanted rice. This may partially be related to the fact that the production of directly seeded rice is usually linked to systems with good irrigation-water control and thus the possibility to manage Fe by drainage, while transplanting dominates in rainfed agriculture (*Maclean* et al., 2002). On the other hand, it is conceivable that root injury that particularly older seedlings endure in the process of pulling before transplanting presents an opening for uncontrolled Fe influx. This root damage is considerably more severe in seedlings grown in aerobic compared to flooded or water-saturated seedbeds. The uncontrolled Fe^{2+} influx may be particularly severe when the injured root section is planted into the soil layer with highest Fe^{2+} concentrations (2–15 cm). The C content usually decreases with soil depth, thereby depriving the Fe-reducing organisms of their energy source in deeper layers. Transplanting depth could thus be used to avoid high soil-borne iron concentrations during the sensitive seedling stages of rice.

Measures fostering the rice plants' ability to cope with Fe^{2+}: The application of essential plant nutrients can reportedly counteract negative effects of excess amounts of iron by competing with Fe^{2+} for uptake and ion-adsorption sites at the root or by enhancing plants' defense or tolerance mechanisms. Potassium seems to play an important role in regulating Fe in the rice plant, both as competing ion and by reducing root exudation (*Prade*, 1987). In situations where mineral-K application cannot be sustained, the use of rice straw may present a promising alternative (*Sahrawat*, 2000). Zinc application increases the tolerance of rice to an elevated content of reduced iron in the soil. The activity of SOD is essential for the detoxification of superoxide radicals in tissues, and Zn is a component of SOD isoenzymes. Depending on the soil and the season, application of P, K, Ca, Mg, and Zn, solely or in combinations, have been reported to reduce bronzing symptoms and increase rice yield (*Benckiser* et al., 1983; *Ismunadji* and *Ardjasa*, 1989; *Mitra* et al., 1993; *Bhattacharyya* et al., 1995; *Devi* et al., 1996; *Sahrawat* et al., 1996; *Sahrawat*, 2000). A critical analysis of these reports indicates, however, that the nutrient effects strongly vary with site and season. While large positive effects of P and Zn have been reported in soils inherently low in these nutrients, nutrient application was less effective in situations of "induced deficiency" (*e.g.*, high organic-matter content, respiration inhibitors). Furthermore, test cultivars that responded positively to nutrient addition with reduced Fe-toxicity symptoms in the

wet season, failed to show the same response during the dry season (*Audebert* and *Saharawat*, 2000), when a high vapor-pressure deficit enhances the acropetal transport of Fe^{2+}. Thus, the effectiveness of mineral-nutrient application to counteract iron toxicity will depend on the soil type, the time, severity, and the duration of Fe-toxicity occurrence as well as the test cultivars used.

4.3 Plant level

Among the most prominent strategies to address Fe toxicity at field level is the use of tolerant rice cultivars (Fig. 5). Breeders have developed a wide array of cultivars with various degrees of adaptation, using both traditional breeding methods (*Akbar* et al., 1987; *Gunawardena* et al.,1982; *Luo* et al., 1997; *Mahadevappa* et al., 1991) and quantitative trait loci (QTL) analysis combined with marker-assisted breeding (*Bennett*, 2001; *Wan* et al., 2003a, b; *Wissuwa*, 2005). Segregating populations or target genotypes were subsequently subjected to usually nondefined Fe-toxic environments, both in the greenhouse and in the field for cultivar screening. Thus, more than 100 cultivars tested in farmers fields in the Philippines out-yielded farmers' traditional variety by an average of 1.9 Mg ha^{-1} (*Neue* et al., 1998). Greenhouse trials conducted in the Philippines, identified 479 out of 6140 tested rice cultivars to be "relatively tolerant" to excess Fe in soil (*Lantin* and *Neue*, 1989). In West Africa, some 20 cultivars out-yielded the highest-yielding farmers' varieties under Fe-toxic conditions in two inland valleys of Côte d'Ivoire (*WARDA*, 1995; 2002). Based on tissue iron concentrations of >300 mg kg^{-1}, the majority of these cultivars were hypothesized to employ tolerance rather than avoidance or exclusion mechanisms (*Yamanouchi* and *Yoshida*, 1981).

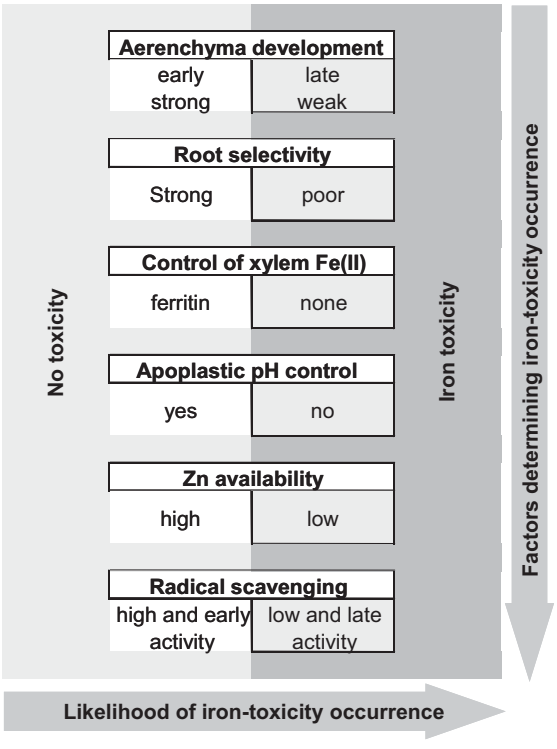

Figure 5: Lowland-rice strategies involved with the adaptation to iron toxicity.
Abbildung 5: Anpassungsstrategien von Nassreis an Eisentoxizität.

However, a high concentration of Fe in the aboveground plant biomass without the expression of the typical damage symptoms (bronzing) does not necessarily indicate symplastic tolerance. It cannot be excluded that such cultivars may have exhibited an efficient mechanism of symplastic exclusion or of stem/leaf sheath retention. In addition, cultivars that showed a high tolerance to Fe^{2+} in greenhouse trials or in one field environment, failed to express the same tolerance level in another environment (*WARDA*, 1993). Hence, some tolerant cultivars from the screening trails in the Philippines succombed to Fe toxicity in West Africa and *vice versa.* The available QTL populations are all based on crosses with the stress adapted cultivars "Niponbare" (*Bennett*, 2001) or "Azucena" (*Wu* et al., 2003), and the sequencing of the genes associated with stress tolerance is ongoing (*Wissuwa*, 2005). However, the projected marker-assisted breeding for Fe-toxicity tolerance is unlikely to advance progress in the development of rice varieties suited to the diverse Fe-toxic environments as long as the mechanisms by which the supposedly tolerant parent cultivars cope with elevated Fe^{2+} concentrations are unknown. The discussion above highlights that problems in the development and selection of adapted rice genotypes for Fe-toxic conditions still relate to the inadequacy of our knowledge on adaptation mechanisms. The following section will summarize the reported findings and propose a framework for future breeding and crop-management research, taking into account the diversity of Fe-toxic environments and crop adaptation strategies.

5 Integrated concepts and research needs

This paper has identified three environmental and soil situations that are typically associated with the occurrence of iron toxicity. These clusters differ in terms of severity and the time of occurrence of excessive amounts of reduced iron in the soil and comprise acid sulfate soils (Cluster 1) with severe and sustained Fe toxicity throughout the rice-cropping period, acid clay and organic soil (Cluster2), showing a gradual increase in toxicity, and valley-bottom soils (Cluster 3) with a distinct Fe-toxicity peak coinciding with the onset of interflow. Crop adaptation mechanisms comprise the exclusion of Fe^{2+} by oxidation or root-membrane selectivity (Strategy 1), the avoidance of elevated concentrations in leaf tissues by stem/sheath tissue retention and leaf apoplastic exclusion (Strategy 2), and the tolerance of reduced Fe in the symplast by detoxification of oxygen radicals (Strategy 3).

Agronomic management interventions are geared towards an avoidance of excess amounts of Fe^{2+} in the soil (landscape and water management, crop establishment), an avoidance of a rapid drop in redox potential (application of organic manure and K), and the fostering of the rice crops' ability to cope with elevated concentrations of Fe^{2+} (P and Zn application, seedling management). The extent of Fe-toxicity problems in tropical rice-based systems and the lack of widely adapted and adoptable solutions highlights the need to match crop adaptation mechanisms with site-specific factors associated

with Fe toxicity and, in situations where adaptation is insufficient, the specific targeting of site-specifically appropriate agronomic management practices. In addition, the functioning and the genetic variability of adaptation mechanisms need to be understood for the successful targeting of cultivars to clearly defined environments (severity, time of occurrence, and duration of Fe-toxic stress). The processes and mechanisms will first be examined thereafter, following the pathway of Fe^{2+} from the rhizosphere to the leaf cells. In the next step, we will attempt to match these mechanisms with the time course of the prevailing conditions of Fe toxicity and define supporting agronomic management strategies (Fig. 6).

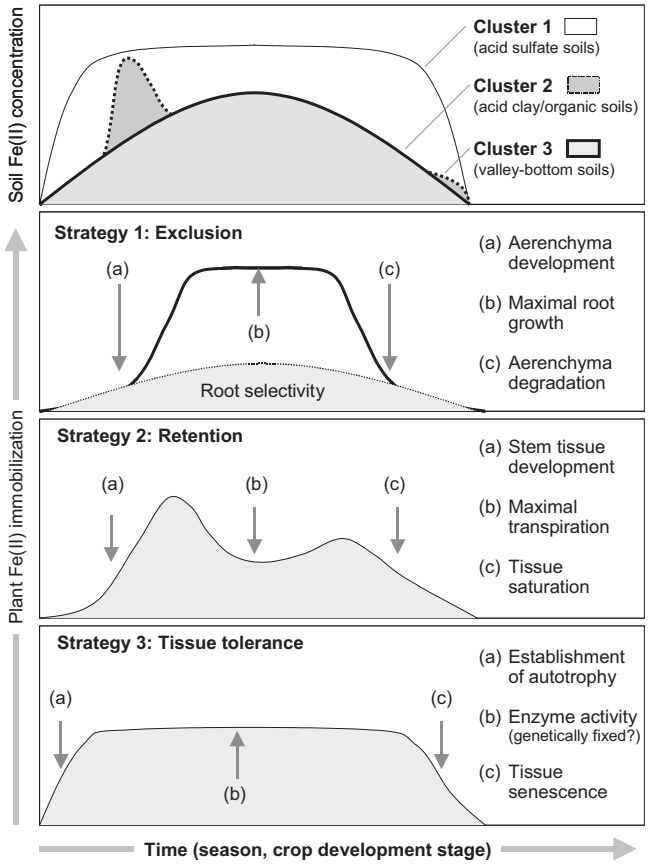

Figure 6: Conceptual dynamics of iron-toxicity occurrence in soils and of crop adaptation mechanisms during the season and the rice-crop development.
Abbildung 6: Konzeptionelle Darstellung des Zeitverlaufs der Eisenbelastung von Böden und der Wirksamkeit pflanzlicher Anpassungsmechanismen während der Reisanbauperiode.

Iron exclusion: The oxidation barrier in the rhizosphere results from the presence of an aerenchyma and determines the amount of Fe^{2+} entering the root. Aerenchyma formation is induced by ethylene. Genotypic differences in the rate of ethylene production in root tissues have been reported. A vigorous and early development of aerenchyma and its longevity may be breeding objectives or potential screening strategies for situations of seedling toxicity (Cluster 3) and late-season toxicity (Cluster 1). At the level of the root apoplast, the selectivity of cell membranes can reduce the uptake of Fe^{2+}. This selectivity is an active metabolic process that is limited to the late vegetative growth stages. It is impaired by nutrient deficiencies, the accumulation of respiration inhibitors and by Fe^{2+} concentration of >50 mg L^{-1}. Thus, it is unlikely to become a promising candidate in future rice-screening strategies for extremely adverse environments. Nevertheless, it may convey an advantage to cultivars used in the clay soils associated with Cluster 2.

Iron retention: The immobilization and deposition of iron at specific "dumping sites" has been associated with iron tolerance. Similar to root selectivity, this mechanism appears to operate during the vegetative growth stages and may thus be relevant for situations described by Clusters 2 and 3. As it appears to involve the formation of phytoferritin in the xylem, sap analysis for ferritin may be envisioned as a possible screening tool.

Tissue tolerance: The apoplastic pH determines the mobility of Fe in leaves. Plants able to increase the pH in the apoplast under Fe stress may avoid an uncontrolled accumulation or influx of Fe^{2+} into the leaf cell. As with the tissue retention mechanism, there is no empirical evidence that genotypic differences exist within the available germplasm. Symplastic tolerance of iron on the other hand is associated with an increased activity of radical-scavenging enzymes, and activities of POD and SOD in leaf cells show strong genotypic variations. This mechanism may be of major relevance in situation where Fe^{2+} exclusion or tissue retention are not yet established (seedling stage) or no longer operational (reproductive stage). To date, it has not been systematically researched or used as a rice-screening strategy for Fe tolerance.

Considering conceptually the time course of Fe^{2+} accumulation in soils and the dynamics of crop adaptation mechanisms, the following conclusions can be derived (Fig. 6):

(1) In acid sulfate soils (Cluster 1), potentially toxic concentrations of Fe establish early in the season and are sustained throughout the growing period of rice. Rhizospheric Fe exclusion (Strategy 1) appears to match the occurrence of Fe^{2+} during most of the vegetative growth phase. However, at the seedling stage and during the late reproductive growth phase of rice, exclusion mechanisms do not fully operate and may need to be supplemented by other measures. Thus, seedling management (direct seeding or young undamaged seedlings) may help to counteract seedling-stage toxicity. Selection for aerenchyma longevity may alleviate the problem of late-season toxicity. In addition, P and Zn deficiencies are frequently associated with acid sulfate soils, and the application of these nutrients may further enhance the capacity of rice to tolerate elevated iron concentrations at tissue level (Strategy 3).

(2) In acid clay and organic soils (Cluster 2), the toxicity establishes gradually. It tends to be less severe than in acid sulfate soils, which may be related to a higher CEC and a better nutrient availability in the "fertile" clay soils. The time course of Fe^{2+} formation in the soil appears to match the dynamics of rice roots' selectivity to avoid uncontrolled Fe^{2+}

influx. As this mechanism is limited to conditions of moderate toxicity and strongly affected by respiration inhibitors, supporting measures may need to include water management (avoid the build-up of Fe^{2+} in the soil) and a dense planting (re-oxidation during the time of maximal soil Fe concentrations). An increased level of tissue tolerance is likely to further improve the adaptation of rice cultivars to this type of environment.

(3) Finally, in valley-bottom soils where Fe toxicity is associated with interflow (Cluster 3), the time course of soil Fe^{2+} closely matches the activity of stem retention mechanisms (Strategy 2). Depending on the environmental conditions, this strategy may need to be complemented by an improved tissue tolerance of the cultivars used and by agronomic management interventions at landscape level (reduction and control of interflow). As valley-bottom soils tend to be sandy-textured with low CEC and nutrient stocks, supplementary agronomic measure may need to include the application of K and where possible Fe^{2+} removal by mid season.

The targeting of the technical options described above needs to be underpinned with progress in rice breeding. The characterization of QTL mapping populations and their subsequent use in marker-assisted breeding is seen to accelerate the process of developing appropriate germplasm, provided the quantitative traits linked with the genetic loci are related to well-understood and clearly described adaptation mechanisms (exclusion, avoidance, tolerance). An improved understanding of crop adaptation for a range of iron-stress situations needs to be translated into repeatable and robust tools for the screening of improved rice cultivars. Finally, these cultivars must be targeted to the iron-toxic environment in which their adaptation mechanism will actually be translated into higher or more robust yields and may need to be supplemented with the appropriate agronomic management interventions both at landscape and plot level. Only such an integrated approach is seen to effectively address the problem of Fe toxicity in lowland rice.

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