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An improved understanding of soil Cd risk to humans and low cost methods to phytoextract Cd from contaminated soils to prevent soil Cd risks

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Abstract

We believe greater consideration should be given the agronomic and nutritional/bioavailability factors that influence risk from Cd-contaminated soils. We have argued that the ability of rice to accumulate soil Cd in grain while excluding Fe, Zn and Ca (even though the soil contains 100-times more Zn than Cd) was important in adverse effects of soil Cd is farm families in Asia. Further, polished rice grain is deficient in Fe, Zn and Ca for humans, which promotes Cd absorption into duodenal cells. New kinetic studies clarified that dietary Cd is absorbed into duodenum enterocytes; ^{109}Cd from a single meal remained in the duodenum for up to 16 days; part of the turnover pool ^{109}Cd moved to the liver and kidneys by the end of the 64-day 'chase' period. Thus malnutrition induced by subsistence rice diets caused a higher absorption of dietary Cd and much higher potential risk from soil Cd than other crops. Because rice-induced Fe-Zn-Ca-malnutrition is so important in soil Cd risk, it seems evident that providing nutritional supplements to populations of exposed subsistence rice farmers could protect them against soil Cd during a period of soil remediation. In the long term, high Cd rice soils need to be remediated. Remediation by removal and replacement of contaminated soil is very expensive (on the order of $3 million/ha); while phytoextraction using the high Cd accumulating ecotypes of the Zn-Cd hyperaccumulator, *Thlaspi caerulescens*, should provide low cost soil Cd remediation.

Introduction

It is well established that long-term consumption of rice grown in paddy soils that are contaminated by geogenic Zn+Cd from mining or smelting wastes can cause a high prevalence of renal proximal tubular dysfunction (e.g., Kobayashi 1978; Cai *et al.* 1995; Kobayashi *et al.* 2002). However, consumption of home garden crops (e.g., lettuce with 4 mg Cd and 500 mg Zn kg^{-1} dry weight) grown on aerobic soils with as high or higher Cd+Zn as found at Shipham, UK (Strehlow & Barltrop 1988), Palmerton, Pennsylvania, USA (Sarasua *et al.* 1995), and Stolberg, Germany (Ewers *et al.* 1993) [some garden soils exceeding 100 mg Cd and 10,000 mg Zn kg^{-1}], has not caused Cd disease in humans. Also, no apparent Cd disease was found in humans who consumed high levels of Cd in oysters (Sharma *et al.* 1983). These quite different outcomes from rice vs. garden foods grown on soils with geogenic Cd+Zn contamination suggests to us that agronomic and/or nutritional-bioavailability factors related to subsistence rice diets must play a key role in soil Cd risk from rice paddy soils.

Rice diets promote risks to humans from soil Cd

We have discussed two ways that subsistence rice diets may increase soil Cd risk: (1) rice grain Cd but not Zn is increased when rice is grown on geogenic Cd+Zn contaminated paddy soils resulting in high rice grain

Cd:Zn and Cd:Fe ratios (Simmons *et al.* 2003); and (2) the very low total and bioavailable levels of Fe, Zn and Ca in subsistence rice diets cause deficiencies in these nutrients that might substantially increase net Cd absorption by humans. Further, our studies of the effect of consuming rice diets (0.2 mg Cd kg^{-1} diet) with marginal vs. adequate (meet NRC diet recommendation for rats) Fe-Zn-Ca showed 10-fold higher net Cd absorption in rats on marginal diets, and emphasized an increased intestinal Cd turnover pool as likely causing the difference (Reeves & Chaney 2001, 2002).

Plant species and cultivars affect crop accumulation of Cd and Zn (Chaney *et al.* 1999). Breeding crop cultivars that accumulate lower levels of Cd in edible crop tissues is one method to reduce food-chain transfer of Cd from contaminated soils. Lower Cd durum wheat and sunflower cultivars have been introduced in the last few years; improving cultivars takes time and money, but gives persistent benefit of lower dietary Cd.

Recent field samples of soils and crops at a geogenic Zn-contaminated site in Thailand showed the remarkable difference in Zn and Cd accumulation by rice and soybeans (Simmons *et al.* 2003). Rice growing on soil with up to 7,000 mg Zn kg^{-1} had no increase in grain Zn; but up to 200 mg Cd kg^{-1} in the same fields caused a large increase in grain Cd (Figure 1A). Soybean grain had a relatively greater increase in Zn than in Cd when grown in the same fields in different years (Figure 1B). Similarly, Chinese cabbage accumulated 643 mg Zn^{-1} when leaves contained 5.8 mg Cd kg^{-1}. Most fruit, grain and tuber crops exclude Cd from edible crop relative to Zn. If soil pH is acidic and promotes Cd uptake to plant leaves, co-contaminating Zn can serve as a limit on dietary Cd by killing the crop when leaf Zn exceeds about 400–500 mg kg^{-1} dry weight. Using Cd uptake by lettuce to predict increased dietary Cd from 100% home grown garden crops in Palmerton, PA, Baker and Bowers (1988) found that Zn phytotoxicity reduced lettuce yields and kept gardeners from ingesting as high as 490 μg Cd/week, the WHO Provisional Tolerable Weekly Intake for Cd with a safety factor.

Independent of our concern about nutritional interactions affecting Cd absorption, it has become widely recognized that rice grain contains inadequate bioavailable concentrations of Fe, Zn, and Ca to supply human requirements (Welch and Graham 2002; Bouis 2002). Subsistence rice diet-induced Fe and Zn malnutrition is now recognized as an important international public health problem. An international program has been developed to breed rice and other staple foods that contain higher densities of bioavailable Fe and Zn (Bouis, 2002) which may also reduce risks from soil Cd.

It is well known that Fe, Zn and Ca deficiencies cause increased Cd absorption in tissues of animals consuming such diets. Fe deficiency is known to be especially important in Cd absorption by humans (Flanagan *et al.* 1978; Fox 1988; Vahter *et al.* 1996). Zinc deficiency promotes Cd absorption, but high Zn in a similar meal decreases Cd absorption (Fox 1988). These patterns have been confirmed in studies in which crops grown on biosolids amended soils were fed to test animals to characterize changes in Cd and Zn caused by biosolids use; even when Swiss chard Cd was increased 5-fold by growing it on an acidic biosolids amended soil, there was no increase in Cd in liver or kidney of guinea pigs fed the chard for 89 days at 28% of diet (Chaney *et al.* 1978). Furthermore, when livestock were fed forage crops with increased Cd and Zn from biosolids with the geogenic ratio of Cd and Zn, there were no increases in Cd in kidney or liver of cattle, sheep or goats. Livestock production strongly reduces soil Cd flow to human food; in several studies, less than 0.01% of total ingested feed Cd remained in the carcass at slaughter (e.g., Bray *et al.* 1985).

Kinetics of dietary Cd metabolism

Reeves & Chaney (2002) reported the absorption and distribution of dietary Cd in rats when the animals were raised on rice-based diets with marginal or adequate levels of Zn-Fe-Ca. Because rice supplies very little bioavailable Fe, Zn, or Ca, animals fed these marginal diets retained 10 times higher ^{109}Cd than rats given adequate Fe-Zn-Ca diets. The ‘marginal’ diets were designed to avoid reducing the rat growth rates, so the deficiency-stress imposed was very mild. Clearly humans suffer frank deficiency of Fe or Zn when consuming non-supplemented rice diets (Welch & Graham 2002), so our test likely underestimates the potential effects on humans of the low bioavailable Fe, Zn, and Ca from polished rice.

One of our recent experiments used the same protocol for pre-feeding Fe-Zn-Ca marginal and adequate diets and then feeding ^{109}Cd-labeled meals, but then animals were sampled over time in order to follow the movement of ^{109}Cd in a ‘pulse-chase’ experiment. Figure 2 shows the fraction of the dietary ^{109}Cd dose

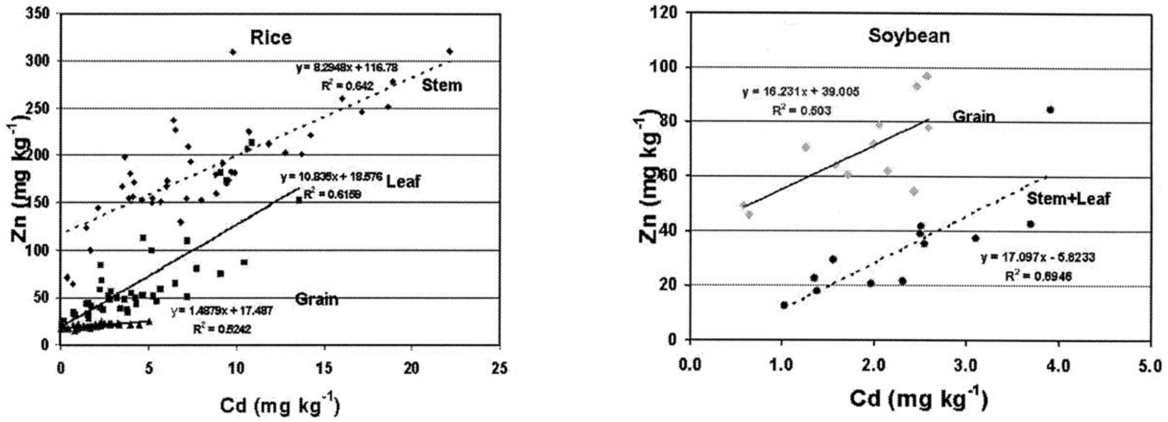

Fig. 1. Cd vs. Zn accumulation in brown rice grain or soybeans in rice and soybean; note Zn exclusion from rice grain but accumulation in soybean grain. Soils were contaminated by Zn-mine wastes with geogenic Cd:Zn ratio (Simmons *et al.* 2003).

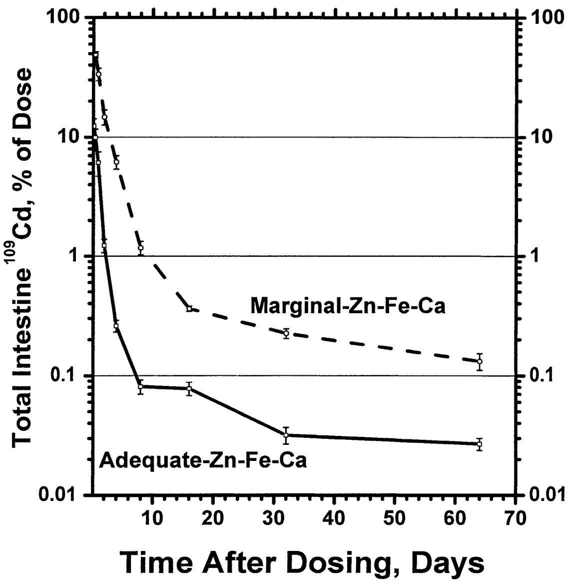

Fig. 2. Effect of marginal vs. adequate supply of Fe-Zn-Ca in rice-based diet on time course of intestinal ^{109}Cd. Rats were fed the marginal or adequate diets for 36 days before the ^{109}Cd-labeled test meal, then up to 64 days post feeding to chase absorbed Cd thru tissues (Reeves and Chaney 2004).

that remained in the intestine up to 64 days after feeding the test meal. The ^{109}Cd in the intestine was always much higher for the 'marginal' diet animals than for the 'adequate'. Although intestinal ^{109}Cd declined over time due to true absorption or excretion from this turnover pool, intestinal Cd remained largely in the duodenum (Reeves & Chaney 2004). We believe that the use of only food levels of Cd, and of diets that do not cause growth differences due to Fe-Zn-Ca deficiency stress, our results may be more relevant to food-chain Cd risk than when toxicological study methods are used. A recent demonstration that the Fe^{2+} transporter in duodenum, DMT1, also transports Cd but not Zn, and the large up-regulation of DMT1 in Fe-stressed animals, seem to explain the increased duodenal ^{109}Cd turnover pool in animals fed Fe-Zn-Ca marginal diets.

Adverse effects of dietary Cd

Diagnosis of adverse effects of dietary Cd is usually achieved through measurement of low molecular weight proteins and Cd in urine. We have raised questions about the diagnosis of renal tubular dysfunction in some European studies because subsistence rice farmers with Cd disease excreted on the order of 100,000 μg β_2-microglobulin (g creatinine)$^{-1}$ in urine while the diagnosis of adverse effects in European studies was based on mean urinary β_2-microglobulin exceeding statistically defined cut-off levels that were not predictive of clinically important renal tubular dysfunction. Furthermore, as persons age, mean concentration and variance of β_2-microglobulin in urine increases, and older citizens are the population who require diagnosis of risk from high dietary Cd. A clarification of the adverse effects of dietary Cd on humans was reported by Ikeda *et al.* (2003) who conducted a meta-analysis of indicator proteins and Cd in urine of subsistence rice populations in Japan with high incidence of renal tubular dysfunction and found that no renal tubular dysfunction occurred until urinary Cd exceeded 10 μg (g creatinine)$^{-1}$. No general population in Europe or the US has exceeded 10 μg

Cd (g creatinine)$^{-1}$ even though urinary Cd may be significantly increased in populations living in Cd contaminated areas. In addition, in a study of over 10,000 middle-aged non-smoking Japanese urban women, no evidence of anyone actually experiencing renal tubular disease from the relatively high dietary intake of Cd commonly found in Japan (2–3 times that in Europe) (Ezaki *et al.* 2003). We believe that this clarification of the indicator proteins vs. Cd in urine supports our model in which paddy rice grown in geogenic Zn contaminated soils plays a special role in food-chain transfer and bioavailability of soil Cd.

Prevention of Cd disease and remediation of Cd+Zn contaminated soils

Nearly all Cd-contaminated soils are contaminated with geogenic Zn+Cd whether from Zn, Cu, Pb, or Ag mines or smelters. This low Cd:Zn ratio provides a limit on Cd uptake into plants for most soils. And the presence of such high levels of Zn inhibits Cd uptake of crops. Although chemical leaching of Cd from contaminated soils has even been tested in the field, it is unlikely that leaching of Cd-EDTA will be permitted or will be cost effective. Agronomic management can reduce the level of Cd in grain (raised soil pH; applying certain phosphates or silicates), as can maintaining flooded soil condition until the grain is mature. But such flooding management lowers grain yield and complicates harvest; thus farmers seldom follow this advice.

Providing nutritional supplements (Fe, Zn, Ca) to subsistence rice consumers could prevent further Cd risk during the time required for soil remediation. Furthermore, if an exposed population were provided these supplements in a randomized test, a strong reduction in blood Cd of supplemented individuals would provide additional support for our model of the importance of paddy rice in soil Cd risks to humans, and may aid public decisions based on practical risks from soil Cd.

The remediation of soil Cd is very expensive when the contaminated soil depth is removed and replaced (on the order of \$3 million/ha), the practice used in Japan during the 1980s. Phytoextraction of soil Cd by using newly recognized Cd hyperaccumulator plants appears to offer equally effective reduction of soil Cd risk at 1% of the cost of soil removal and replacement (Chaney *et al.* 2000). Plant breeding is needed to improve the wild *Thlaspi caerulescens* into commercial phytoextraction cultivars before practical soil remediation could begin. We have shown that strains of this species from southern France absorb 10–20 fold higher amounts of Cd than British and Belgian strains (Figure 3), offering high promise for an effective Cd phytoextraction technology for rice soils. Unless plants can selectively accumulate Cd in their shoots relative to Zn, Zn phytotoxicity will limit annual Cd removals. Ae & Arao (2002) reported that Cd-accumulator rice cultivars grown under upland conditions could phytoextract about 50 g of Cd ha^{-1} yr^{-1} while most crop plants remove less than 5 g Cd ha^{-1} yr^{-1}. Growing the Cd phytoextraction cultivar and obtaining 5 t ha^{-1} of *Thlaspi* biomass with 1,000 mg Cd kg^{-1} would yield removal of 5,000 g Cd ha^{-1} yr^{-1}. No other phytoextraction plant has been reported to offer the rapid Cd removal as these southern France *Thlaspi caerulescens* ecotypes.

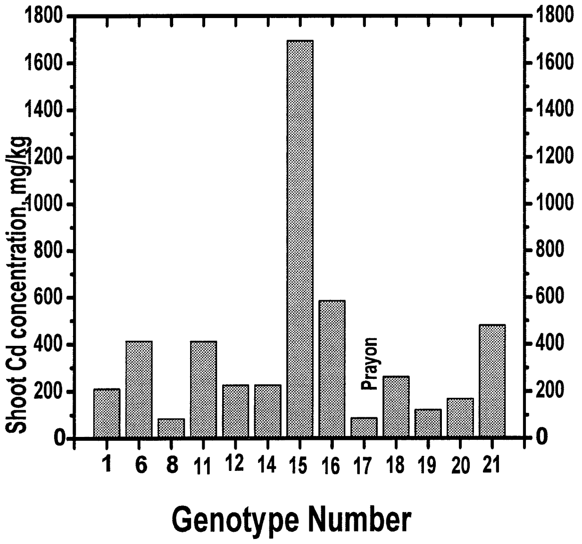

Fig. 3. Variation in Cd in shoots of *Thlaspi caerulescens* ecotypes from different locations in Europe when grown on soil at Palmerton, PA which contained 158 mg Cd and 15,500 mg Zn/kg dry soil and pH 6.0 in water (Chaney et al. 2000); genotypes 15, 16, 21, 6 and 8 were from southern France, while genotype 17 was from Prayon, Belgium, and genotypes 8, 19 and 20 were from the UK.

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