

An In-Depth Look at the Importance of Arsenic Speciation in Rice Products

Irina Agro - International Territory Manager, SPEX CertiPrep



Rice, like many of its various grain brethren, is a basic staple food, commonly consumed in fairly large quantities by many different cultures across the globe. It is a relatively cheap crop whose seeds (grain) can be cooked and eaten or processed to make a huge variety of products like flours, cereals and sweeteners. However, in recent years there have been ongoing studies pointing to high levels of natural arsenic accumulation in rice. Rice is unique among the grains to exhibit this kind of contamination and the reasons for it are as interesting as they are necessary to understand in order to ensure a safe food supply.

The biological reasons behind rice's bioaccumulation of arsenic are just now beginning to be explored and understood. All rice cultivators exhibit a natural silicon uptake pathway which allows them to absorb large amounts of the element from the soil. The silicon is then used by the rice in order to create observable "silicon bodies" to strengthen the leaves, stems and husks against various pest attacks (an adaptation that has allowed it to become a fairly reliable, disease resistant, cash crop). However, because of the chemical similarities between arsenic and silicone in flooded soil, the same pathways also readily take up inorganic arsenic which is then deposited in the same locations as the silicone - the husks of the rice grain being of acute interest in terms of human consumption. This also can create some concern that requires further study, however, it is only one of the contributing factors.

Ecologically speaking, while arsenic is naturally present in the environment, the use of arsenic based pesticides has greatly contributed to increased soil contamination. As with many metal and metalloid contaminants, arsenic tends to stay in the soil for long periods of time; even though many of these pesticides have been banned for 35+ years, the inorganic contaminants still remain. The most obvious example of this legacy can be demonstrated by sample analysis of rice grown in different regions of the USA. Comparison studies, done in 2007 by Dr. Andrew Meharg and his team at the University of Aberdeen, found that rice originating in states like Arkansas and Louisiana where former cotton fields, now used as rice paddies, were once doused in lead arsenate, contained two to three time mores arsenic than rice grown in California, in mostly virgin soil.

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An In-Depth Look at the Importance of Arsenic Speciation in Rice Products (Pgs. 1-3)

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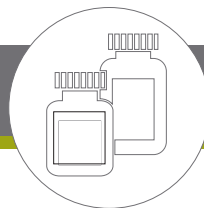
ABSTRACT: Rice is an agricultural commodity of high economic and nutritional importance around the world. Rice is a staple for nearly half of the world's seven billion people with between 500 - 8000 million metric tons consumed each year. Rice consists as a staple in more than 80% of Asian diets. In recent years, there have been ongoing studies pointing to high levels of natural arsenic accumulation in rice. Rice is unique among the grains to exhibit this kind of contamination and the reasons for it are as interesting as they are necessary to understand in order to ensure a safe food supply. Take a look at our article on rice and arsenic speciation in this issue!

A Case of Methylmercury Poisoning (Pgs. 3-4)

Patricia Atkins, Applications Scientist, SPEX CertiPrep

ABSTRACT: Mercury is a common environmental and aquatic contaminant this found as both natural chemical by-products and persistent pollutants from agricultural and industrial processes. In the 1940's and 1950's, Japanese citizens were plagued by mysterious neurological symptoms which later would be linked to industrial chemical manufacturing discharges into bays and rivers. Our article on a historical methyl mercury poisoning event is featured in this issue's discussion of speciation in our world.

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Arsenic is a well known carcinogen and immunosuppressant whose ill-effects on human health have been very well documented. While all forms of arsenic are considered toxic to humans, this element is unique in that its inorganic species presents significantly higher levels of toxicity. It was believed that human over-exposure was mostly due to contaminated ground water and, to that effect, the WHO and US EPA have promoted environmental regulatory limits of arsenic in water, including the need to speciate the arsenic contamination. Up until very recently, no such considerations were given food-derived arsenic consumption. With new research showing surprisingly high levels of arsenic in rice, and adverse effects consistently being reported at lower level exposure to inorganic arsenic than previously suspected, the regulatory landscape is changing, especially when it comes to the most at-risk population: infants and children under the age of three.

Fortified rice products such as cereals, crackers, wafers, and puffs are often referred to as “first foods” and, until recently, have been promoted by pediatricians as a safe introduction of solid foods to infants. Moreover, since the “blacklisting” of high-fructose corn syrup, organic brown rice syrup (OBRS) has become the sweetener of choice for baby formula, cereals, and other “child friendly” food products. Of the two pathways of possible arsenic exposure, OBRS is, perhaps, more problematic; brown rice usually contains much higher levels of inorganic arsenic than white rice (Sun et al. 2008) due to inorganic arsenic accumulation disproportionately occurring in the “husk” portion of the grain, which is removed during the polishing process used to manufacture white rice and its derivative products. Effectively, this results in children under the age of three consuming a higher than average amount of rice and rice-based products, raising significant cause for concern.

Compounding the problem of a higher frequency of rice and rice product consumption is the issue of increased relative exposure levels. Inorganic arsenic exposure limits are set based on the amount of arsenic consumed per kilogram of body weight (currently set as 0.17 µg/kg, per the EPA drinking water regulation). However, due to their small size, infants and young children, eating the same portions as adults, are exposed to levels two to three times higher than adults (EFSA 2009).

The hazards of arsenic over-exposure in young children have been well characterized; physical and mental developmental delays (Wasserman et al. 2004), suppressed immune response (Nadeau et al. 2014), a higher instance of childhood cancers (Moore et al. 2002), as well as a higher than average likelihood of developing certain types of cancers (specifically bladder, kidney, lung, and skin) later in life (Smith et al. 1998). The levels of exposure required to cause harm have been a point of contention and much discussion. In Europe, the Joint FAO/WHO Expert Committee on Food and Additives (JECFA) has established the “acceptable” levels of exposure as 15 µg/kg **total** arsenic, however this number was based on studies of adult consumption habits and total (inorganic and organic) arsenic intake and has recently been challenged.

The US FDA is still reviewing the effects of various arsenic concentrations in rice and rice products and has yet to publish any regulations or recommendations. A “proposed action level”, or maximum limit, of inorganic arsenic in baby food was proposed in April 2016, however, any real regulatory steps are still pending. However, effective June 25, 2015, the European Union passed and ratified Commission Regulation 2015/1006, which amends the existing EC regulation 1881/2006 regarding contaminant limits in food. The amended regulation took effect on January 1st, 2016 and, as of this writing, all food manufacturers, distributors and resellers must meet the requirements in order to be able to sell within the EU member states.

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The amendment itself is quite small and straightforward; while the previous regulation controlled for many different types of contaminants in foodstuffs, arsenic was not part of these regulated adulterants. The new regulation establishes the need for arsenic testing (total and inorganic) of rice products, breaks down the overarching “rice products” category into four sub-categories - polished rice, husked rice, rice cakes, wafer or cracker, and rice destined for the production of food for infants and young children - and institutes maximum allowable inorganic arsenic limits for each category (see Table 1).

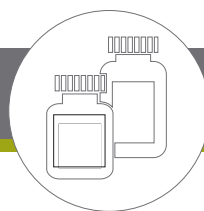


Table 1. Maximum Allowable Inorganic Arsenic Limits.

Sub-Section	Foodstuffs	Maximum Allowable Levels
3.5	Arsenic (inorganic) ⁽⁵⁰⁾ ⁽⁵¹⁾	N/A
3.5.1	Non-parboiled milled rice (polished or white rice)	0.20 µg/kg
3.5.2	Parboiled rice and husked rice	0.25 µg/kg
3.5.3	Rice waffles, rice wafers, rice crackers, and rice cakes	0.30 µg/kg
3.5.4	Rice destined for the production of food for infants and young children ⁽³⁾	0.10 µg/kg

(2) the following endnotes (50) and (51) are added:

⁽⁵⁰⁾ Sum of As(III) and As(V)

⁽⁵¹⁾ Rice, husked rice, milled rice, and parboiled rice as defined in Codex Standard 198-1995

Prior to the enactment of this amendment, there were no formal rules or regulations regarding the presence of arsenic in any food types. Companies wanting to observe due diligence referenced the JECFA set limit of 15 µg/kg total arsenic. With the new regulation in place, these same companies now have a guide for testing procedures and new regulatory limits, including the requirement for speciation and reporting of inorganic arsenic levels (as the sum of As(III) and As(V)).

The new limits and testing guidelines will prove especially valuable in the analysis of food destined for infant consumption. A study specifically examining arsenic concentration in infant formulas found that, while the total concentration of arsenic was not very high across most of the brands surveyed (all but 3 would have met the previous testing limit), the brands that did contain arsenic, when speciated, produced a result of 3:7 organic to inorganic arsenic, and in some cases that ratio was closer to 1:9 (Jackson et al. 2012). In other words, most of these brands would not be suitable for distribution under the new regulations.

It is inevitable that as we develop more accurate and sensitive testing methods, our knowledge and understanding of various substances' health effects expands. Fast and easy speciation by LC/ICP-MS and IC/ICP-MS has allowed us to look past the total arsenic mantra and allowed us to focus on the truly harmful and dangerous factors present in our food and water. Manufacturers will be rewarded for their investment in these new testing methods by higher consumer confidence and less waste, as products that may have previously been deemed inappropriate for sale under the "total arsenic" regulations will now be considered safe because of speciation analysis. As consumers, we can all breathe a sigh of relief as well, knowing that the food we are buying and feeding to our children meets all of the latest safety standards and reflects the latest in our scientific understanding.

A Case History of Methylmercury Poisoning

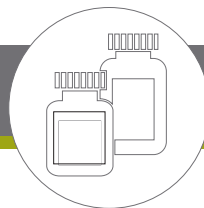
Patricia Atkins - Applications Scientist, SPEX CertiPrep

On the western coast of the southern Japanese island of Kyushu, Minamata village was officially designated by the Japanese government in 1889 with just over 12,000 residents. Less than 20 years later, in 1908, the Nippon Nitrogen Fertilizer Company (later the Chisso Corporation) built its factory in Minamata. The plant began manufacturing acetaldehyde in 1932 using mercury sulfate as a catalyst. By the end of World War II in 1949, Minamata Village had grown into a city of over 40,000 people. A large part of the economy of the city (over half its tax revenue) was the chemical factory and its acetaldehyde product which was an important component in the manufacture of plastics. The plastic boom saw the increase of acetaldehyde product grow from over 200 tons to more than 45,000 tons by 1960.

During this post WWII plastic production boom, a curious phenomenon was observed in Minamata. Cats began to display odd behavior which resulted in them falling into the water and dying. The locals dubbed the curious disease 'cat suicides' or 'dancing cat fever'. Less than a decade later, in the 1950s, a strange



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disease of unknown cause started plaguing the city inhabitants. Just like the cats, people, especially children, would start to stumble or have trouble controlling fine motor skills. In April 1956, a five-year old girl was hospitalized at the Chisso Minamata Plant Hospital. She complained of numbness in her limbs and inability to speak or eat. In May 1956, four patients were admitted to the local hospital all suffering from the same disease characterized by very high fevers, convulsions, psychosis, unconsciousness, coma, and then finally death. The disease was believed to be infectious and patients were quarantined and disinfected. Families were ostracized in fear of the disease which was called the Minamata disease.

By 1957, the medical and scientific community was starting to believe that the disease was not an infectious agent but a result of poisoning. The main culprit was believed to be seafood caught in the Minamata Bay. The local fishermen voluntarily stopped fishing the bay and the government banned fishing in the bay. During this time in the fall of 1958, the corporation changed its discharge system for plant effluent. Prior to this change, the plant effluent was directly discharged into the Minamata Bay. The new system stored the effluent in a pool near the Minamata River where it was later discharged. Suddenly, new cases of Minamata disease were identified near the river. During the course of the effluent discharges from the plant, over 60 tons of methyl mercury was released into the waterways.

Almost three years after the start of the outbreak of the disease, the majority of university researchers were able to conclude that the origin of the disease was most likely an organomercury compound. Researchers investigated the mercury distribution in Minamata Bay in February 1959 and discovered that the concentrations of mercury at the mouth of the plant's wastewater canal were shockingly high. Levels were detected in the sediment of 2 kg/ton which would be a concentration considered high enough to mine or refine. The company later created a division to reclaim and sell the mercury recovered from the sludge.

In November of 1959, a researcher from the University of Kumamoto reported his belief that the plant effluent was the cause of the poisonings. Citing lack of proof, the investigating council ruled the poisoning was probably due to an unknown source of organomercury pollutants. The findings of the University of Kumamoto created public protests by patients and fishermen in Minamata calling for compensation and for plant effluent treatment systems to be installed at the plant. By the end of 1959, agreements had been reached for sympathy compensation. Living patients, who were certified to have Minamata disease, were given between the equivalents of approximately \$275 to \$925 per year. Families of patients who died from the disease were given a one time payment of about \$3000. Minamata disease started to fade from the public consciousness until a similar outbreak of the disease occurred in 1965 in the Niigata Prefecture along the banks of the Agano River.

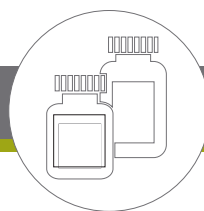
A different chemical factory used a similar mercury catalyst that was thought to be responsible for the previous illnesses. From the fall of 1964 to the spring of 1965, cats in Niigata were observed experiencing 'dancing cat fever'. Shortly afterwards, patients living along the Shiranui Sea began to appear with symptoms of Minamata disease. As a result of the events in Minamata, lawsuits were quickly filed against the company and investigations were reopened into Minamata's pollution. In 1968, twelve years after the discovery of Minamata disease and four months after the discontinuation of the production of acetaldehyde using its mercury catalyst, the government issued its final conclusions: "Minamata disease is a disease of the central nervous system, a poisoning caused by long-term consumption, in large amounts, of fish and shellfish from the Minamata Bay. The causative agent is methylmercury. Methylmercury produced in the acetaldehyde acetic acid facility in Shin Nihon Chisso's Minamata factory was discharged in factory wastewater...".

In light of the governments findings, patient advocate societies asked for new compensation agreements with the company. Meetings and arbitrations were negotiated but, in the end, many sought to bring their grievances to trial. During those trials, dramatic testimony was given by plant employees and managers who testified to the falsification of safety studies done by the plant during the outbreaks. Many employees admitted the company put profit ahead of safety. As of 2001, 2,265 victims were officially certified, 10,000 people received compensation from the company, and 1,784 patients had died. During the course of certification, over 17,000 people applied for certification with the council. Enormous social and economic pressure was put on citizens not to declare their symptoms and apply for compensation. The certification council was pressured to reject claimants and minimize the economic impact on the company.

Minamata disease is an important issue to this day in Japan. Lawsuits still continue. Most of the congenital patients exposed to the Minamata pollutants during the 1950s and 1960s are now in their fifties or older and are reporting severe changes to their health.

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