Popular Article

TRENDS IN AGRICULTURE SCIENCE

Vol.3 Issue 11 November 2024, Page: 2288-2290

Genetic control of low arsenic accumulation in rice

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https://doi.org/10.5281/zenodo.14207841

Arsenic contamination in groundwater and the food chain is a major global health concern, particularly in South East Asia, where rice is a dietary staple (Begum et al., 2016). In regions like West Bengal and Bangladesh, rice contributes up to 50% of total dietary arsenic intake, while in China, this figure rises to 60%. Asian adults consume 400–600 grams of rice daily, making them vulnerable to arsenic exposure. Even trace amounts of arsenic (0.2 mg/kg) in rice pose significant health risks, as arsenic is a non-threshold carcinogen (Zhao et al., 2010). Arsenic levels in rice often exceed the safe limit of 0.2 mg/kg recommended by the Codex Alimentarius Commission, with some varieties containing up to 0.5–0.6 mg/kg depending on the cultivar and environmental conditions. Climate change is expected to worsen this issue by increasing the levels of arsenite (AsIII) in the rhizosphere of rice, leading to higher arsenic accumulation in grains and potentially reducing productivity. Rice is particularly prone to arsenic accumulation due to its traditional cultivation under submerged conditions. Flooding creates anaerobic soil environments that convert arsenate (AsV) into arsenite (AsIII), which is more readily absorbed by rice roots through silicon transporters like Lsi1 and Lsi2, given the similarity in properties between arsenite and silicon below pH 8. Once inside the plant, arsenate is further reduced to arsenite by OsHAC genes, leading to either efflux into the soil or sequestration in vacuoles as complexes with phytochelatins (PCs). The detoxification process involves C-type ATP-binding cassette transporters (ABCC1 and ABCC2) that help store As(III)-PC complexes in vacuoles (Song et al., 2014). Despite these detoxification strategies, a portion of absorbed arsenic is transported from the roots to the shoots and deposited in the grains. Phloem plays a crucial role in this process, accounting for about 80% of the total arsenic content in rice grains. Several genes regulate arsenic uptake and stress tolerance in rice, including phosphate transporters (OsPHT1, OsPHT4, and OsPHT8), the MATE family protein (OsMATE2), and the RING finger E3 ligase (OsAIR2), among others (Shi et al., 2016; Das et al., 2017). However, these genes are predominantly expressed in roots and do not significantly reduce arsenic accumulation in the grains. Additionally, studies have found no direct correlation between shoot silicon content and grain arsenic concentration, complicating breeding efforts. Cooking and rinsing rice with arsenic-free water can

Published: 22 November 2024
ISSN: 2583-7850

lower arsenic levels by up to 40%, but this also results in the loss of essential vitamins and minerals, making it an imperfect solution (Gray et al., 2015). Developing rice varieties with low arsenic accumulation potential is the most promising strategy for reducing dietary arsenic exposure. Genetic modifications targeting key transporters and regulatory genes could help limit arsenic uptake and translocation, ultimately leading to safer rice consumption and improved public health in arsenic-affected regions. This article gives a brief description about the genetic control of As metabolism in rice.

Genes related to as uptake, transport and metabolism in rice

The gene families involved in arsenic (As) uptake, transport, and metabolism in rice plants are crucial for understanding how these plants manage arsenic contamination. Various gene families contribute to different aspects of arsenic handling.

Studies involving mutations and overexpression indicate that OsABCC1 (Os04g52900), a C-type ATPase, is a crucial gene in rice that protects seeds from arsenic toxicity by sequestering As(III)-PC complexes into the vacuoles at the first node of the panicle. The expression of OsABCC1 is most prominent in the diffuse vascular bundle, aiding in the transport of nutrients, including metals and metalloids, into the chalaza end. Mutant lines deficient in ABCC1 show a 10–18 fold increase in grain arsenic content compared to wild-type plants. Consequently, researchers have proposed developing rice lines with lower arsenic accumulation and higher arsenic tolerance by overexpressing OsABCC1. While numerous genes related to arsenic transport and speciation have been identified using gene overexpression and silencing approaches, the allelic variation of these genes, including OsABCC1, remains unclear despite significant genotypic differences in arsenic accumulation in grain and shoot across rice varieties. SNP based markers have also been designed for the favourable allele of it.

Aquaporins, such as Lsi1 and Lsi2, located in the plasma membrane, have been shown to reduce As accumulation in roots and straw when knocked out, indicating their roles in AsIII transport. Similarly, phosphate transporters like OsPT1 and OsPT4 also play significant roles; their knockout leads to decreased AsV content in roots and grains. The arsenate reductase genes OsHAC1;1 and OsHAC1;2, when overexpressed, increase AsIII efflux and enhance As(V) tolerance. Other important genes include CRT-like transporters like OsCLT1, which reduce AsIII accumulation in roots, and O-acetylserine (thiol) lyase (OASTL-A1), which affects As accumulation differently in roots and shoots. Additionally, phytochelatin synthases such as OsPCS1 help decrease As levels in rice grains through overexpression. Regulatory genes like OsPHF1 and OsWRKY28 are involved in transcriptional regulation of As transporters, influencing overall As resistance. Finally, the arsenite antiporter PvACR3;1 contributes to altering total As accumulation in unhusked rice grain through

Published: 22 November 2024
ISSN: 2583-7850

heterogeneous expression. Understanding these gene families provides insight into improving rice's resilience against arsenic contamination, which is vital for food safety and agricultural sustainability.

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