Potential risk of cyclopiazonic acid toxicity in Kodo millet (*Paspalum scrobiculatum* L.) poisoning

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Kodo millet, a staple food in North India, is frequently consumed, but its consumption can lead to intoxication and poisoning. Millet, cultivated in dry and semi-arid regions, is often infected by fungal species rather than bacterial and virus pathogens, causing economic crop loss and adversely affecting grain and fodder yield. They are a rich source of nutrition, anti-oxidant properties, and medicinal properties. Ergot, a parasitic fungal endophyte found in kodo millet ear heads, can cause poisoning when consumed. These kodo grains are frequently infested with *Aspergillus tamarii* Kita, which produces a significant amount of the mycotoxin cyclopiazonic acid (CPA). Cyclopiazonic acid (CPA) is a neurotoxin produced by certain *A. flavus* and *Aspergillus oryzae* strains, which produce aflatoxins. Mycotoxicosis outbreaks in humans are not well characterized, and the direct correlation between mycotoxin and toxic effects in vivo is not well established. CPA, a specific inhibitor of sarcoplasmic reticulum Ca2+-ATPase, can adversely affect broiler chicken health, as demonstrated by toxicological evaluation of aflatoxins and CPA alone or in combination. Most toxins have reported acute and chronic effects in prokaryotic and eukaryotic systems, including humans, despite their unclear specific modes of action. This review explores fungal pathogens, the toxicity of CPA to animals and humans both by itself and in combination with other mycotoxins, biocontrol strategies, and storage methods for better utilisation of kodo grains post-harvest.

Kodo poisoning, cyclopiazonic acid (CPA), mycotoxin, toxicity, *Paspalum scrobiculatum L.*