Letter to the Editor: Increased uric acid levels following fructose consumption: a biochemical perspective

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Dear Editor

I am writing to address a significant concern regarding the article titled "Acute effect of fructose, sucrose, and isomaltulose on uric acid metabolism in healthy participants". Published in first issue of volume 72 in January 2023.[1] As a Biochemistry Faculty member deeply engrossed in unravelling the intricate mechanisms of metabolic pathways, I find it imperative to shed light on the biochemical intricacies underlying this phenomenon. This study has illuminated the biochemical link between fructose consumption and increased uric acid production.

I would like to mention bias regarding sample selection. This study was not excluded the patient those are having glycogen storage disorders (GSDs) and taking high amount of animal diet. GSDs are major cause of hyperurecimia as there is deficiency of glucose 6 phosphatase, flux of glucose is entered in hexose monophosphate shunt which produces more purine which further breakdown and converted into uric acid. Hyperuricemia remains asymptomatic but gradually damages kidney and joint. Also in people of Ashkenazi Jewish, Mexican, Chinese, and Japanese descent GSDs is more common.[2]

In present study the samples were analysed after 240 mins of fructose intake and it's very difficult to make conclusion increased production of uric acid immediately. Different genes have varying expression kinetics. If the gene is involved in a complex metabolic pathway, it might take more time for changes to become noticeable as the entire pathway adjusts to the dietary shift. Short-term dietary changes might result in transient gene expression changes that reverse once the diet is reverted. Long-term dietary habits could lead to more sustained changes in gene expression.[3]

The fructose metabolized by fructokinase pathway which leads to the generation of adenosine triphosphate (ATP), depleting inorganic phosphate and triggering the release of purines. The purine degradation results in the production of uric acid. The fructose consumption increases synthesis of triglycerides in liver through upregulation of de novo lipogenesis. Increased triglyceride levels lead to increased fatty acid breakdown, which further enhance the production of uric acid. It is essential to raise awareness about the potential health implications of excessive intake.

In conclusion, the surge in uric acid levels subsequent to fructose consumption is a subject of paramount concern from a biochemical perspective. As a devoted Biochemistry Faculty member, I strongly urge for continued research into this area.

Thank you for your attention to this matter.

***Key Words*:** **fructose, hyperuricemia, gene expression and GSDs**

References

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2 Lee G, Schafer AI. *Goldman's Cecil Medicine (24th ed.)*. Philadelphia: Elsevier Saunders, 2011: 1356.

3 Zhang P, Sun H, Cheng X, *et al*. Dietary intake of fructose increases purine *de novo* synthesis: a crucial mechanism for hyperuricemia. *Front Nutr* 2022; **9**: 1045805.

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