An Orchestrator on Xenobiotic Metabolism is the Human Intestine Biota

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Introduction

e human digestive tract is a location for xenobiotic metabolism, and microbes that live there play a role. e gut microbiome, which is a collection of microorganisms found in the gastrointestinal system, can change the pharmacokinetics of medications, environmental toxins, and heavy metals by altering their metabolic result Depending on the enzymatic activity within the microbial niche, direct chemical alteration of xenobiotic by the gut microbiome, either through the intestinal tract or via enter hepatic circulation, might result in enhanced metabolism or bioactivation. [1-15] ose that reverse the alterations given by host detoxi cation pathways are among the unique enzymes encoded in the microbiome.

Disruptions to the composition and activity of the gut microbiome contribute to a variety of human diseases. An indicator of microbiome health is community diversity, as redundancy in functional pathways supports the maintenance of essential functions upon perturbation. Such imbalances can contribute to a variety of conditions throughout the body, including in ammation, muscle mass, depression, and blood pressure in the elderly, suppressed infant weight gain, perturbed immune and endocrine system development, increased allergic responses and behavioral and neurochemical alterations. However, the most notable and well-understood examples are in relation to metabolism. Disruptions to the generally consistent metabolic activity of the microbiome can contribute to obesity and metabolic disease through the dysregulation of lipid and carbohydrate metabolism.

Subjective heading

e AZT uptake by zebra sh was assessed according to the methodology adopted by Keskar and Jugade with little modi cations. It was used 8 animals/group, weighing approximately 350 mg/animal, which were euthanized (immersion in ice-slurry) and subsequently macerated in 1 mL of phosphate bu ered saline (PBS), and centrifuged at 13,000 rpm for 5 min (at 4°C). Aliquots of 30 µL of the sample supernatant were transferred to test tubes (previously sanitized) and mixed with 470 µL of acetonitrile solution (0.01 M), 500 µL of bromocresol green solution

Discussion

e gut microbiome directly metabolizes xenobiotics Inhaled xenobiotics interact with the many microbial populations that populate the small intestine and colon, which can o en modify them

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other organs and tissues. Hence, it is also termed a “stealth pathogen. How *T.* pallidum overcomes the immune response and damages

of In addition, to assess whether these drugs were able to alter the mechanosensory system of the sh, we performed the count of super cial neuromasts in exposed individuals. For this, we adopted the procedures described in, in which, brie y, the live animals (n = 8/each group) were placed (for 30 min) in a beaker containing 400 mL of water (with constant aeration) reconstituted with 5 mM of the uorescent dye 4-(4-Diethylaminostyryl)-1-methylpyridinium iodide (4-Di-2-ASP), from stock solution (40 mg of 4-Di-2-ASP) diluted in 10 mL of dimethyl sulfoxide P.A. en, the animals were carefully removed and transferred to a beaker containing dechlorinated water (without dye), and remained for 30 min, to remove excess of dye in the body. A er that, the animals were euthanized (immersion in ice-slurry) and positioned horizontally on glass slides for later observation under a uorescence microscope.

e e ects of exposure to AZT e HCQ (alone or in combination) on oxidative stress reactions were evaluated based on (i) indirect nitric oxide (NO) (via nitrite measurement; NO_2^-) (ii) thiobarbituric acid reactive substances (TBARS) [predictive of lipid peroxidation)]; (iii) production of reactive oxygen species (ROS), and (iv) hydrogen peroxide (H_2O_2) [which plays an essential role in responses to oxidative stress in di erent cell types e Griess colorimetric reaction [as described in was used to measure NO_2^- and the TBARS levels were determined based on procedures described by , respectively., with some modi cations.

e supernatant of the same 8 animals/group mentioned above was used. In that case, 200 µL aliquot of supernatant from each sample was transferred to previously cleaned hygienic conical bottom microtubes and, sequentially, 400 µL of the bromothymol blue solution (0.65 mmol/L) and 600 µL of dichloromethane P.A. were added sequentially.

en, the solutions were homogenized in a vortex mixer (for 30 s) and centrifuged at 1500 rpm, for 5 min, at 23°C. Subsequently, the aqueous phase of the mixture was discarded and 200 µL of the organic phase was transferred to a 96-well microplate, for later reading at 405 nm, in an ELISA reader. e concentrations of HCQ in the samples were determined from the equation of the straight line obtained by making a standard curve, using known concentrations of HCQ (0, 0.00625, 0.0125, 0.025, 0.05, 0.1, 0.2, 0.4 and 0.8 mg/mL). e background uorescence of the control samples was also determined and su

Conclusion

e complicated interplay between the gut microbiome, host factors, and xenobiotic metabolism is di cult to understand. e microbial world's variety of enzyme responses has broadened our understanding of how xenobiotics are digested. ese microbial enzymes' products can perform new functions, whether they are distinct from host metabolites or complement those that are currently present. Microbial xenobiotic metabolism can result in bioactivation, detoxi cation, or even reverse host detoxi cation in some situations, such as with glucuronidases. By binding or importing xenobiotics or reinforcing the intestinal mucosal barrier, the microbiome atop enterocytes can inhibit absorption. Finally, we're starting to understand how the microbiome a ects the host's xenobiotic metabolism enzymes, changing the fate of endobiotics and xenobiotics.

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Con ict of Interest

e authors declare that they are no con ict of interest.

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