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Chinese Medicine

Translational metabolomics in drug metabolism and gastrointestinal carcinogenesis

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Abstract

Despite the increasingly recognized associations among central obesity, hyperinsulinemia and dyslipidemia, microbial dysbiosis, and cancer epidemic, the exact molecular mechanisms that integrate these events have remained largely unclear. Human beings are colonized by a diverse range of symbiotic gut bacteria and other microorganisms collectively known as the gut microbiota. Here we show, through a unique liver fibrosis and HCC mouse model, that the complex metabolic communications between the microbiota and the host change dynamically at different stages of liver conditions, affecting disease risk factors and therapeutic responses. Together these complex interactions comprise a series of host-microbiota metabolic axes which produce a large array of metabolites that serve as important signaling factors and energy substrates, such as bile acids (BAs), choline, and short-chain fatty acids. A critical component of the host-microbiota metabolic axes, the liver- BA - microbiota axis, is emerging, in which the BAs modulate metabolic phenotypes and participate in the genesis of drug toxicity and a substantial number of malignancies, particularly in gastrointestinal carcinogenesis.

Given the complexity of the BA signaling processes as well as the direct chemical interactions between the microbes and host, a systems perspective is needed to help understand liver - BA - microbiota axis and its implication in gastrointestinal carcinogenesis so that microbiota-mediated alterations in BA metabolism in disease states can be reversed. Based on our combined metabolomics and microbiome findings in human, rodents, and cell models, we believe that gut microbiome-derived metabolites and factors, resulting from dysbiosis, may constitutes a critical mechanism of drug induced liver injury, or sustained cellular injury which progresses to malignancy.

~ ALL ARE WELCOME ~