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Can iron and mitochondrial defects cause diabetes?

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Contributions of iron and mitochondrial defects to the pathogenesis of diabetes have long been debated but unresolved in general population due to the polygenic nature of most diabetes cases. Limited numbers of monogenic diabetes have been identified and allow us to examine the underlying cellular pathways and molecular mechanisms of diabetes. Involvement of iron and mitochondrial defects in the dysfunction and loss of insulin-producing beta-cells have been confirmed in Friedreich's ataxia and animal models that are deficient in TFAM (transcriptional factor A, mitochondrial) and NCB5OR (NADH cytochrome b5 oxidoreductase. My laboratory has been studying how Ncb5or functions to maintain iron homeostasis and mitochondrial function, as well as their relationship to lipid metabolism, in diabetes and neurodegeneration. The new knowledge will provide potential therapeutic target(s) for both diseases.

Biography

Hao Zhu received his PhD degree from the University of Texas at Austin and postdoctoral training at Brigham and Women's Hospital, Harvard Medical School. He has published more than 30 original research papers and review articles in reputed journals, including Science, PNAS, JBC and Biochemical Pharmacology. He has reviewed grant proposals for Diabetes UK and Portuguese Foundation for Science and Technology and various manuscripts for numerous journals.

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