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SET REDUCTION BY MIR-199B-5P ATTENUATES TRICHLOROETHYLENE-INDUCED HEPATOCYTE APOPTOSIS

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Trichloroethylene (TCE) is a commonly used industrial solvent and widespread environmental pollutant, exposure to which can cause severe liver damage. Previously we have observed in a model of TCE-induced apoptosis of cultured human hepatocytes (L-02 cell line) that SET (a protein encoded by the SET gene in humans) is abnormally elevated which acts as a key mediator of the induction of apoptosis, however, the potential molecular mechanisms modulating SET in that model remain unclear. In this study, through a screening of various databases six microRNAs were predicted to potentially regulate SET. Subsequent experiment indicated that three (miR-199b, miR-21, and miR-23a) of them were decreased in TCE-treated L-02 cells. Further analysis using a dual luciferase reporter assay and miR-199b-5p knockdown/overexpression in L-02 cells revealed that only miR-199b-5p could suppress SET, through direct binding to its 3'-UTR. Functional studies indicated that miR-199b-5p attenuated TCE-induced apoptosis of L-02 cells through the inhibition of SET. In summary, the present study suggests that in TCE-induced cytotoxicity in cultured hepatocytes miR-199b-5p may down-regulate SET, thus further attenuating the toxic response to TCE.

BIOGRAPHY

Xiaohu Ren has completed his PhD in 2015 from Southern Medical University, and now, he is post-doctor of Key Laboratory of Modern Toxicology of Shenzhen. His major research interests include molecular mechanisms of environmental chemical-induced liver injury, proteomic profiling of potential biomarkers for the diagnosis of environmental chemical exposure. He has published more than 20 research papers. Additionally, in the past few years, he has received over six grants, such as Science and Technology Plan Projects of Guangdong, National Science Foundation for Young Scholars of China, National Science Foundation of China, and so on.

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