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Effect of miR-29c and miR-129-5p on epithelial-mesenchymal transition in experimental biliary atresia mouse models

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ABSTRACT. Biliary atresia (BA) is a destructive bile duct disease occurring in newborn children within a few weeks after birth. In this study, the effect of miR-29c and miR-129-5p on epithelial-mesenchymal transition (EMT) in experimental BA was explored by constructing BA mouse models via Rhesus rotavirus vaccine infection. miR-29c and miR-129-5p expression was analyzed by real-time quantitative polymerase chain reaction. EMT was established by induction with transforming growth factor (TGF)-β1. miR-29c and miR-129-5p were overexpressed and inhibited, respectively, by Lipofectamine

transfection. EMT-related protein (formin-like 2, FMNL2; E-cadherin; vimentin; and cytokeratin-19, CK-19) expression was analyzed by western blot and immunofluorescent assay. The results indicated that miR-29c and miR-129-5p were downregulated and upregulated in BA mice. TGF-β1 induction caused a time-dependent decrease and increase in miR-29c and miR-129-5p, respectively. Additionally, TGF-β1 induced an increase in FMNL2 and vimentin expression and a decrease in E-cadherin and CK-19 expression ($P < 0.05$). Overexpression or suppression of miRNA-29c or miR-129-5p, respectively, induced the inhibition of FMNL2 and vimentin, and promotion of E-cadherin and CK-19 expression, in the test groups compared to the non-intervention group ($P < 0.05$). However, the FMNL2, vimentin, E-cadherin, and CK-19 expression did not differ between the control and non-intervention groups ($P > 0.05$). Thus, miR-29c upregulation or miR-129-5p downregulation effectively prevented EMT in BA by regulating the expression of EMT pathway-related proteins. Therefore, miR-29c and miR-129-5p could be utilized as therapeutic targets for BA in the future.

Key words: miR-29c; miR-129-5p; Epithelial-mesenchymal transition; Biliary atresia