THE ADMINISTRATION OF MM-MIR-378A-3P EXACERBATES CHRONIC INFLAMMATION AND FIBROSIS IN A MURINE INTESTINAL MODEL

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Exogenous administration of miR-378a-3p, which is diminished in DSS-induced murine intestinal fibrosis, increases inflammatory response, fibrosis markers, and antiapoptotic molecule expression in a murine colitis model and human intestinal fibroblasts.

KEY POINTS

- In DSS-treated mice, colon tissue showed significantly diminished expression of mm-miR-378a-3p compared with naïve samples, and intravenous administration of miR-378a-3p mimic significantly increased its levels in the colon.
- Mice receiving miR-378a-3p mimic compared with negative control showed increased neutrophils, heightened glandular epithelia changes and architectural distortion, decreased lymphoid follicles, and slightly increased collagen deposition.
- In DSS-treated mice, miR-378a-3p mimic administration significantly increased mRNA expression of Tgfb1, Il1b, and Mmp2.
- In human intestinal fibroblasts, miR-378a-3p mimic treatment significantly increased mRNA expression of antiapoptotic molecules BCL2 and MCL1 but did not significantly modify fibrosis marker expression.
- This study is relevant to researchers investigating microRNA roles in intestinal inflammation and fibrosis in Crohn's disease.

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