Flow cytometry was prepared for assessing THP-1-derived macrophage apoptosis. The protein and expression levels of miR-34a-5p and MDM4 were examined by Western blot and RT-qPCR, respectively. They also measured the levels of total cholesterol (TC) and triglyceride to determine the lipid accumulation. Subsequently, the activities of superoxide dismutase, malondialdehyde, and reactive oxygen species revealed the level of oxidative stress injury after miR-34a-5p and MDM4 knockdown.

After ox-LDL treatment, cell apoptosis of macrophages increased in a dose-dependent and time-dependent manner. With the increase of ox-LDL treatment and the prolongation of treatment time, the expression level of miR-34a-5p was upregulated. Next, interfering with miR-34a-5p inhibited lipid accumulation and oxidative stress injury in ox-LDL-stimulated macrophages. MDM4 was a target gene of miR-34a-5p and was upregulated in ox-LDL-stimulated macrophages. With the increase of ox-LDL treatment and the prolongation of treatment time, the expression level of MDM4 was downregulated. Importantly, MDM4 knockdown partially counteracted the inhibitory effect of miR-34a-5p on oxidative stress injury.

MicroRNA miR-34a-5pknockdown suppressed oxidative stress injury via MDM4 in ox-LDL-treated macrophages [1].

1)
Kong J, Liu L, Song L, Zhao R, Feng Y. MicroRNA miR-34a-5p inhibition restrains oxidative stress injury of macrophages by targeting MDM4. Vascular. 2022 Feb 28:17085381211069447. doi: 10.1177/17085381211069447. Epub ahead of print. PMID: 35226569.

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