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# RNase L contributes to lipid metabolism

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# ***RNase L contributes to lipid metabolism***

College of Sciences and Health Professions

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## **Abstract**

Macrophage-derived foam cell formation is a milestone of the atherosclerotic lesion initiation and progression, leading to cardiovascular diseases and stroke. Foam cells are formed from the disruption of a homeostatic mechanism that manipulates the uptake, intracellular metabolism and efflux of cholesterol within macrophages. Although studies have yielded much information about the homeostatic mechanism, the molecular basis of foam cell formation remains to be fully understood. We recently found that deficiency of RNase L attenuated macrophage functions including macrophage migration and its endocytic activity. Furthermore, RNase L markedly impacted the expression of certain pro- and anti-foam cell genes in macrophages. Most interestingly we have revealed that lack of RNase L significantly increased the formation of foam cells from bone marrow derived macrophages (BMMs). The increase of foam cell formation was associated with up-regulation of the expression of scavenger receptors such as CD36, SR-A, and PPAR-g. These studies provide new insights into foam cell formation and novel therapeutic strategies for atherosclerosis may be designed through activation/up-regulation of RNase L.