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ATORVASTATIN SUPPRESS OXIDISED LOW DENSITY LIPOPROTEIN-INDUCED DENDRITIC CELL-LIKE DIFFERENTIATION OF RAW264.7 CELLS BY INACTIVATION OF THE P38 MAPK PATHWAY

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Dendritic cells (DCs) are professional antigen-presenting cells and have an important role in the pathogenesis of atherosclerosis. It has been confirmed that the optimal oxLDL dose can induce considerable RAW264.7 cells differentiate into dendritic-like cells in our previous work. In this study, we examined whether atorvastatin could inhibit the differentiation of mature macrophages into DCs induced by oxLDL and which pathway could have involved in this process. After 24 h treatment with atorvastatin (20 $\mu\text{mol/ml}$), almost all the RAW264.7 cells induced by oxLDL simultaneously remained in cell size and macrophages morphology compared with those induced by oxLDL alone. Flow cytometric analysis detected reduced dendritic cell surface markers (CD40, CD86, MHC class II and CD1d). Moreover, atorvastatin-treated RAW264.7 cells induced by oxLDL showed functional changes including increased phagocytic ability in a time-dependent manner and reduced TNF- α as well as IL-12 p70 production. Although I κ B, ERK and p38/MAPK phosphorylation were observed during oxidised LDL-stimulated RAW264.7 cells differentiation, only p38/MAPK was verified to be involved in this differentiation process and related to the inhibitory action of atorvastatin. On the whole, these data suggest the differentiation of macrophages into dendritic-like cells induced by oxLDL can be inhibited by atorvastatin and this process may be inactivated by the p38 MAPK pathway.