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THE EXPRESSION OF SOCS IS ALTERED IN ATHEROSCLEROSIS

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Aims Atherosclerosis (AS) is an inflammatory disease induced by hypercholesterolemia. Previous studies indicated that suppressors of cytokine signalling (SOCS) protein could alleviate inflammation by inhibiting janus kinases/signal transducers and activators of transduction (JAK/STAT) pathway. The aim of this study is to investigate the expression trends of SOCSs protein in mice's plaque/vessel and human peripheral blood mononuclear cell (PBMC) face to different level and different duration time of circulation cholesterol.

Methods Cholesterol levels in blood, size and composition, expression of SOCS1 and SOCS3 of plaque of apoE null mice and C57BL6/j mice feed with high fat diet or chow during different periods were detected. In the non-coronary heart disease (non-CHD) population, cholesterol levels and mRNA of SOCS1 and SOCS3 in PBMC were also detected.

Results The results showed that different expression patterns of SOCS1 and SOCS3 in the plaque of apoE null mice during increasing feeding duration with chow. The expression of SOCS3 presented an increasing trend (SOCS3/CD68 increased), but SOCS1 reached a peak in the middle of feeding period and returned to the initial level finally (SOCS1/CD68 decreased). In the high fat diet group, the SOCSs expression level increased correspondingly and the same trend occurred. The expression of SOCS1 and SOCS3 in mRNA and protein level of main aorta of mice was consistent with the trend above. The expression of SOCS1 and SOCS3 were barely observed in C57BL6/j mice with low circulation cholesterol level. In the non-coronary heart disease (non-CHD) population, the serum total cholesterol levels were positively correlated with SOCS3 mRNA in the PBMCs ($r=0.433$, $p=0.012$), but not relevant with SOCS1.

Conclusion These results suggest that SOCSs family may play multiple roles in initial and development of AS, SOCS1 play an anti-inflammation role, but the effect of SOCS3 needs to be further researched on and reassessed in AS.