

The impact of dibenzocyclooctadiene lignans on *C. pneumoniae* induced macrophage foam cell formation

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Background
Chlamydia pneumoniae is an obligate intracellular bacterium which is associated with atherosclerosis and other inflammatory diseases. The infection promotes lipid oxidation and accumulation into macrophages which leads to foam cell formation, a hallmark of atherosclerosis. The impact of *Schisandra chinensis* lignans on *C. pneumoniae* induced redox balance alteration were studied.

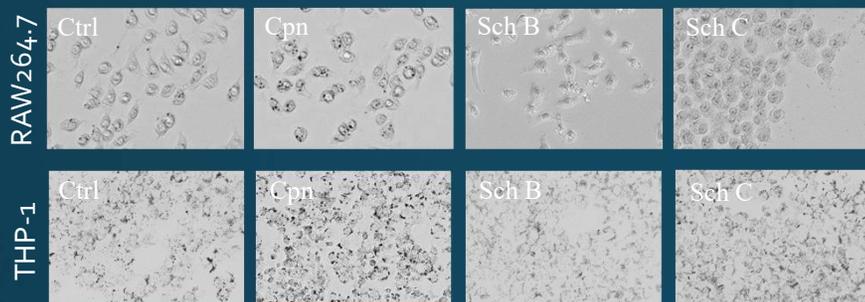


Fig.1. Impact of Schisandrin B and C on *C. pneumoniae* induced Foam cell formation (lipid vacuole accumulation inside the cells) in murine (RAW264.7) and human (THP-1) macrophages was studied with Oil Red O staining. Both compounds inhibited the foam cell formation efficiently.



Fig.2. RT-PCR analysis revealed that Schisandrin B and C induced ABCA1 (cholesterol efflux pump) expression in murine raw264.7 cells, which indicates more efficient cholesterol efflux¹. Schisandrin B and C also upregulated genes involved with glutathione (GSH) synthesis (GCL, GGT-1). In human THP-1 macrophages, schisandrin B downregulated genes involved with GSH synthesis while schisandrin C upregulated gene affecting GSH degradation (ChaC1), indicating that the lignans affect GSH metabolism with different mechanisms.

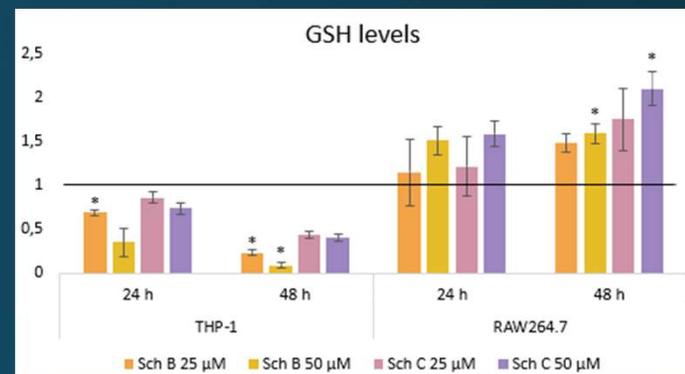


Fig.3. Impact of Schisandrin B and C on cellular GSH levels were studied with a fluorescent microplate assay. In human THP-1 cells lignans decreased the levels, but in murine RAW264.7 cells levels were increased.

Conclusion
Schisandrin B and C decrease the *C. pneumoniae* induced foam cell formation and thus could inhibit the onset of early atherosclerosis. The species-specific impact on GSH levels warrant further investigation.

¹Kortesoja, M., Taavitsainen, E., & Hanski, L. The influence of dibenzocyclooctadiene lignans on macrophage glutathione and lipid metabolism associated with *Chlamydia pneumoniae*-induced foam cell formation. *Advances in Redox Research*. 2021;1:100001.