

Product Data Sheet

cells[1][2][3].

Treatment with BMS-309403 significantly decreased MCP-1 production from THP-1 macrophages in a dose- and time-dependent manner[1].

BMS-309403 sodium (15 mg/kg; chronic treatment; daily for 6 weeks) improves endothelial function, phosphorylated and total eNOS and reduced plasma triglyceride levels but did not affect endothelium-independent relaxations[3]. Animal Model: C57BL/6J mice (ApoE^{-/-} mice)[3]

[1]. Sulsky R, et al. Potent and selective biphenyl azole inhibitors of adipocyte fatty acid binding protein (aFABP). *Bioorg Med Chem Lett*. 2007;17(12):3511-3515. [2]. Lin W, et al. BMS309403 stimulates glucose uptake in myotubes through activation of AMP-activated protein kinase. *PLoS One*. 2012;7(8):e44570. [3]. Lee MY, et al. Chronic administration of BMS309403 improves endothelial function in apolipoprotein E-deficient mice and in cultured human endothelial cells. *Br J Pharmacol*. 2011;162(7):1564-1576.

Caution: Product has not been fully validated for medical applications. For research use only.

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