Inhibitory effect of epigallocatechin 3-O-gallate on vascular smooth muscle cell hypertrophy induced by angiotensin II.


Source

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Abstract

Recent evidence indicates that epigallocatechin 3-O-gallate (EGCG), the major catechin derived from green tea leaves, lowers the risk of cardiovascular diseases such as atherosclerosis and hypertension. However, a precise mechanism for this biologic function has not yet been clearly delineated. Angiotensin II (Ang II) stimulates vascular smooth muscle cell (VSMC) hypertrophy, which is a critical event in the development of atherosclerosis, hypertension, and angioplasty-induced restenosis. In the present study, we show that EGCG inhibits Ang II-stimulated VSMC hypertrophy, as determined by [3H]leucine incorporation into VSMC. Since mitogen-activated protein kinase (MAPK) families are involved in cell growth, we determined whether EGCG affects them. EGCG pretreatment did not exert any significant changes in Ang II-stimulated activation of extracellular signal-regulated kinase (ERK) and p38 MAPK. EGCG only inhibited Ang II-stimulated activation of c-Jun N-terminal kinase (JNK). Moreover, EGCG suppressed Ang II-induced c-jun mRNA expression. In contrast, EGC, a structural analogue of EGCG, did not inhibit the JNK activity or c-jun mRNA expression. In addition, a specific JNK inhibitor, SP600125, dose-dependently suppressed Ang II-stimulated VSMC hypertrophy. These results suggest that the effect of EGCG on Ang II-induced VSMC hypertrophy is due to specific inhibition of the JNK signaling pathway at both transcriptional and posttranslational levels, which may underlie its beneficial effect on the cardiovascular diseases.

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