

ANGIOTENSIN II INDUCES CYCLOOXYGENASE-2 EXPRESSION IN MOUSE AORTIC ENDOTHELIAL CELLS

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The renin angiotensin system (RAS) is involved in inflammation, endothelial proliferation, and angiogenesis.¹ In particular, the vasoactive peptide angiotensin II (ANGII) is thought to mediate many pro-inflammatory actions via signalling through the angiotensin type 1a receptor (AT1aR).² Thus, ANGII may be a factor in tumor development.

Cyclooxygenase-2 (COX-2), a cytokine-inducible enzyme that is upregulated by many pro-inflammatory stimuli, facilitates angiogenesis.³ Two downstream effectors of COX-2 that stimulate angiogenesis are vascular endothelial growth factor (VEGF) and basic fibroblast growth factor (bFGF).⁴ Although AT1aR signalling is known to increase VEGF², it remains unclear whether COX-2 plays a role in ANGII-mediated angiogenesis.

Here we hypothesized that ANGII induces COX-2 in mouse aortic endothelial cells (MAEC). MAEC were treated with or without 1 μ M ANGII for 1, 4, or 24 hours. Samples were collected for each time point (n=3) and protein was isolated for western blotting. Blotting for COX-2 indicated a 2.5- and 2.75-fold increase in protein at 1 and 4 hours respectively. COX-2 expression at 24 hours, however, was no different than control. The induction of COX-2 in endothelial cells in response to ANGII may stimulate angiogenesis.

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