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INHIBITION OF CYCLIC STRAIN-INDUCED ENDOTHELIN-1 SECRETION BY TRILINOLEIN

ABSTRACT

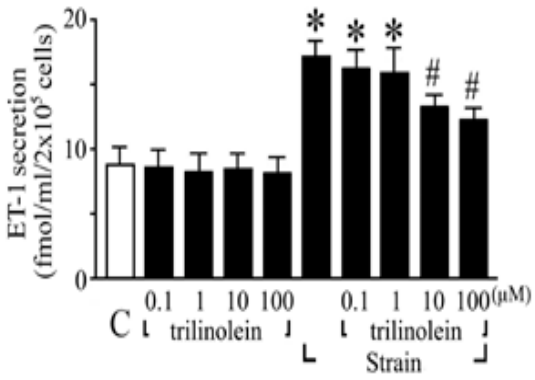
Ttrilinolein, isolated from the traditional Chinese herb *Sanchi* (*Panax notoginseng*), has been shown to have myocardial protective effects via its antioxidant ability. However, the cellular and molecular mechanisms of the protective effect of trilinolein in the vascular system remain to be elucidated. Endothelin-1 (ET-1) is a potent vasopressor synthesized by endothelial cells both in culture and in vivo. The aims of this study were to test the hypothesis that trilinolein may alter strain-induced ET-1 secretion and to identify the putative underlying signaling pathways in endothelial cells. We show that trilinolein inhibits strain-induced ET-1 secretion. Trilinolein also inhibits strain-increased reactive oxygen species (ROS) formation. Furthermore, pretreating cells with trilinolein or antioxidant N-acetyl-cysteine decrease strain-increased ET-1 secretion. Both trilinolein and N-acetyl-cysteine also attenuated the strain-stimulated activator protein-1 reporter activity. In summary, we demonstrate that trilinolein inhibits strain-induced ET-1 secretion, partially via attenuation of ROS formation.

SPECIFIC AIMS

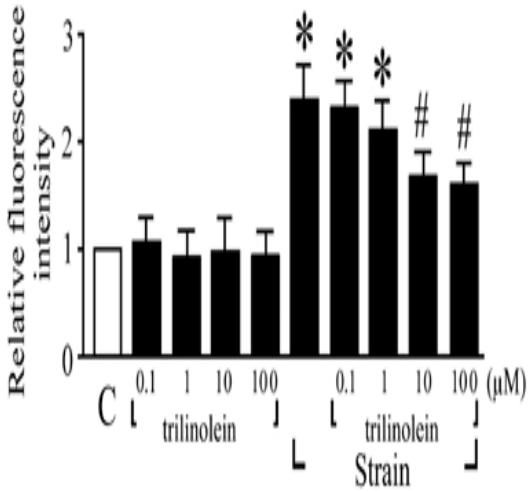
The aims of this study were to test the hypothesis that trilinolein may alter strain-induced ET-1 secretion and to identify the putative underlying signaling pathways in endothelial cells.

RESULTS

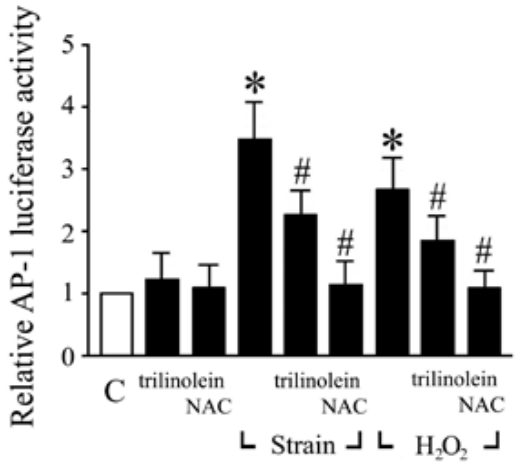
Trilinolein inhibits strain-induced ET-1 gene expression in endothelial cells.



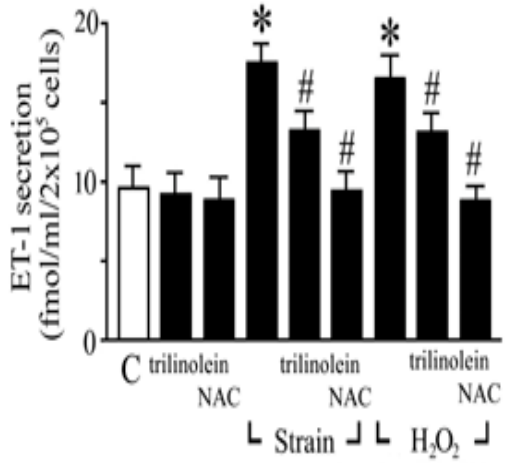
Effect of trilinolein on strain-induced ROS generation in endothelial cells.



Inhibitory effect of trilinolein on strain-increased activator protein-1 reporter activity in endothelial cells.



Trilinolein *via* attenuation of ROS generation in endothelial cells.



CONCLUSIONS

In summary, we demonstrate that trilinolein inhibits strain-induced ET-1 secretion, partially via attenuation of ROS formation.

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