

external link (opens in a new window)

Search Sources Analytics Alerts My list Settings Live Chat Help Tutorials

Quick Search

Search

Back to results | < Previous 103 of 125 Next >



View at publisher | Download Export Print E-mail Create bibliography Add to My List

European Journal of Pharmacology

Volume 535, Issue 1-3, 27 March 2006, Pages 228-233

The beneficial in vitro effects of lovastatin and chelerythrine on relaxatory response to acetylcholine in the perfused mesenteric bed isolated from diabetic rats

Fatehi-Hassanabad, Z.^{ab}, Imen-Shahidi, M.^a, Fatehi, M.^a, Farrokhfall, K.^a, Parsaei, H.^a^a Department of Physiology and Pharmacology, Faculty of Medicine, Mashhad University of Medical Sciences, Mashhad, Iran^b Department of Biomedical Sciences, Atlantic Veterinary College, University of Prince Edward Island, 550 University Avenue, Charlottetown, PEI. C1A 4P3, Canada

Abstract

View references (24)

Diabetes mellitus is associated with an increased risk of cardiovascular disease. Endothelial dysfunction (i.e. decreased endothelium-dependent vasorelaxation) plays a key role in the pathogenesis of diabetic vascular disease. The present study was undertaken to determine whether diabetes induced by streptozotocin alters mesenteric responses to vasodilators and, if so, to study the acute in vitro effects of lovastatin and chelerythrine. Endothelial function was assessed in constantly perfused preparation removed from rats, 12 weeks after treatment with either saline or streptozotocin (45 mg/kg, intraperitoneally). In pre-contracted mesenteric beds (with 100 μ M phenylephrine) removed from diabetic rats, the concentration response curve to acetylcholine, but not to sodium nitroprusside, was significantly reduced. Perfusion with lovastatin (10 μ M for 20 min) or chelerythrine (1 μ M for 20 min) significantly improved the acetylcholine-mediated relaxation in preparations removed from diabetic but not control rats. Pre-incubation of tissue with N^G-nitro-L-arginine methyl ester hydrochloride (10 μ M for 20 min) inhibited the beneficial effect of lovastatin but not chelerythrine. Pre-treatment of tissue with indomethacin (10 μ M for 20 min) did not modify the effects of lovastatin or chelerythrine on acetylcholine responses. The present results demonstrate that endothelial dysfunction induced by diabetes (in a resistant vasculature, such as rat mesenteric bed) may be improved by an acute exposure to either lovastatin or chelerythrine. Furthermore, our results suggest that the beneficial effect of lovastatin is mediated via the nitric oxide pathway. © 2006 Elsevier B.V. All rights reserved.

Reaxys Database Information

View Compounds |

Author keywords

Chelerythrine; Diabetes; Endothelium; Lovastatin; Rat mesenteric bed; Streptozotocin

Indexed keywords

EMTREE drug terms: acetylcholine; chelerythrine; glucose; indometacin; mevinolin; n(g) nitroarginine methyl ester; nitroprusside sodium; phenylephrine; streptozocin

EMTREE medical terms: animal experiment; animal model; animal tissue; arterial pressure; article; body weight; controlled study; drug mechanism; endothelial dysfunction; glucose blood level; in vitro study; male; mesentery blood vessel; nonhuman; priority journal; rat; vasoconstriction; vasodilatation

MeSH: Acetylcholine; Alkaloids; Animals; Benzophenanthridines; Blood Glucose; Blood Pressure; Body Weight; Diabetes Mellitus, Experimental; Dose-Response Relationship, Drug; Enzyme Inhibitors; Hydroxymethylglutaryl-CoA Reductase Inhibitors; Lovastatin; Male; Mesenteric Arteries; Nitroprusside; Perfusion; Phenanthridines; Phenylephrine; Protein Kinase C; Rats; Rats, Wistar; Vasoconstriction; Vasoconstrictor Agents; Vasodilation; Vasodilator Agents

Medline is the source for the MeSH terms of this document.

Chemicals and CAS Registry Numbers: acetylcholine, 51-84-3, 60-31-1, 66-23-9; chelerythrine, 34316-15-9; glucose, 50-99-7, 84778-64-3; indometacin, 53-86-1, 74252-25-8, 7681-54-1;

Cited by since 1996

This article has been cited **3 times** in Scopus:
(Showing the 2 most recent)Badavi, M., Abedi, H.A., Dianat, M.
Exercise training and grape seed extract co-administration improve endothelial dysfunction of mesenteric vascular bed in STZ-induced diabetic rats
(2011) *International Journal of Pharmacology*Ghaffari, N., Ball, C., Kennedy, J.A.
Acute modulation of vasoconstrictor responses by Pravastatin in small vessels
(2011) *Circulation Journal*

View details of all 3 citations

Inform me when this document is cited in Scopus:

Set alert | Set feed

Related documents

Showing the 2 most relevant related documents by all shared references:

Pannirselvam, M., Anderson, T.J., Triggie, C.R.
Endothelial cell dysfunction in type I and II diabetes: The cellular basis for dysfunction
(2003) *Drug Development Research*Matsumoto, T., Kobayashi, T., Wakabayashi, K.
Cilostazol improves endothelium-derived hyperpolarizing factor-type relaxation in mesenteric arteries from diabetic rats
(2005) *American Journal of Physiology - Heart and Circulatory Physiology*

View all related documents based on all shared references or select the shared references to use

Find more related documents in Scopus based on:

Authors | Keywords

More By These Authors

The authors of this article have a total of **47 records** in Scopus:
(Showing 5 most recent)Lang, V.Y., Fatehi, M., Light, P.E.
Pharmacogenomic analysis of ATP-sensitive potassium channels coexpressing the common type 2 diabetes risk variants E23K and S1369A.
(2012) *Pharmacogenetics and genomics*Fatehi, M., Raja, M., Carter, C., Soliman, D., Holt, A., Light, P.E.
The ATP-sensitive K⁺ channel ABCC8 S1369A type 2 diabetes risk variant increases MgATPase activity

Add apps | Help