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Vasorelaxant Effect Produced by Aqueous Extract of *Cladophora sp.* in Aorta from Normotensive Rats

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The present study aims to examine the vasorelaxant activity of the aqueous extract of green alga, *Cladophora sp.* collected from Malaysian coastal regions. Aorta from male Wistar-Kyoto normotensive rats (250-350g) was excised, cleaned, cut into 2-3 mm transverse rings and mounted onto 10 ml organ baths containing oxygenated Krebs-Henseleit solution at 37°C. In some experiments, the endothelial layer of the aortic rings was removed. Cumulative concentration-response (CCR) curves of aqueous extract of *Cladophora sp.* (1 µg/ml - 3 mg/ml) constructed on phenylephrine (PE) pre-contracted rings with functional endothelium showed concentration-dependent relaxation at extract concentrations ranging from 1 µg/ml to 0.1 mg/ml. A maximum relaxation of 48.4 ± 4.3% (n=8, P<0.001) was obtained at 0.1 mg/ml of extract, after which the responses were reduced in a concentration-dependent manner to 16.2 ± 4.8 % (n=8, P<0.001) at the highest extract concentration tested. The responses produced over the whole CCR curves examined were abolished in endothelium-denuded aortic rings. In another study, the CCR curves of aqueous extract were constructed on endothelium-intact aortic rings in the presence or absence of antagonists: N^G-nitro-L-arginine-methyl ester (L-NAME, 30 µM), 1H-[1,2,4]oxadiazolo[4,3-alpha]quinoxalin-1-one (ODQ, 10 µM), methylene blue (100 µM), indomethacin (10 µM), glibenclamide (10 µM) and atropine (1 µM). The effects of the extract were unaltered by the cyclooxygenase inhibitor indomethacin, ATP-sensitive potassium (K^+) channel blocker glibenclamide, and muscarinic receptor antagonist atropine. However, the non-selective nitric oxide (NO) synthase inhibitor L-NAME, and soluble guanylate cyclase inhibitors ODQ and methylene blue, were able to block the effects of the extract completely. In conclusion, vasorelaxation effect of the aqueous extract of *Cladophora sp.* is mediated through endothelium-dependent NO-cGMP pathway, and does not involve the release of prostanoids nor ATP-sensitive K^+ channels opening. The vasodilatory effect of the extract is also not related to muscarinic receptor activation.