Biochem Biophys Res Commun. 2013 Jul 12;436(4):601-6. doi: 10.1016/j.bbrc.2013.06.003. Epub 2013 Jun 10.

Far-infrared radiation acutely increases nitric oxide production by increasing Ca(2+) mobilization and Ca(2+)/calmodulin-dependent protein kinase II-mediated phosphorylation of endothelial nitric oxide synthase at serine 1179.

Park JH, Lee S, Cho DH, Park YM, Kang DH, Jo I.

Source
Department of Molecular Medicine and Ewha Medical Research Institute, Ewha Womans University Medical School, Seoul 158-710, South Korea.

Abstract
Repeated thermal therapy manifested by far-infrared (FIR) radiation improves vascular function in both patients and mouse model with coronary heart disease, but its underlying mechanism is not fully understood. Using FIR as a thermal therapy agent, we investigate the molecular mechanism of its effect on endothelial nitric oxide synthase (eNOS) activity and NO production. FIR increased the phosphorylation of eNOS at serine 1179 (eNOS-Ser(1179)) in a time-dependent manner (up to 40min of FIR radiation) in bovine aortic endothelial cells (BAEC) without alterations in eNOS expression. This increase was accompanied by increases in NO production and intracellular Ca(2+) levels. Treatment with KN-93, a selective inhibitor of Ca(2+)/calmodulin-dependent protein kinase II (CaMKII) and H-89, a protein kinase A inhibitor, inhibited FIR radiation-stimulated eNOS-Ser(1179) phosphorylation. FIR radiation itself also increased the temperature of culture medium. As transient receptors potential vanilloid (TRPV) ion channels are known to be temperature-sensitive calcium channels, we explore whether TRPV channels mediate these observed effects. Reverse transcription-PCR assay revealed two TRPV isoforms in BAEC, TRPV2 and TRPV4. Although ruthenium red, a pan-TRPV inhibitor, completely reversed the observed effect of FIR radiation, a partial attenuation (~20%) was found in cells treated with Tranilast, TRPV2 inhibitor. However, ectopic expression of siRNA of TRPV2 showed no significant alteration in FIR radiation-stimulated eNOS-Ser(1179) phosphorylation. This study suggests that FIR radiation increases NO production via increasing CaMKII-mediated eNOS-Ser(1179) phosphorylation but TRPV channels may not be involved in this pathway. Our results may provide the molecular mechanism by which FIR radiation improves endothelial function.
Copyright © 2013 Elsevier Inc. All rights reserved.

KEYWORDS:
AMP-activated protein kinase, AMPK, BAEC, Ca(2+)/calmodulin-dependent protein kinase II, CaMKII, EC, Endothelial nitric oxide synthase, FIR, Far-infrared, HUVEC, NO, Nitric oxide, PKA, Phosphorylation, TRP, TRP ankyrin, TRP melastatin, TRP vanilloid, TRPA, TRPM, TRPV, Transient receptor potential vanilloid ion channel, bovine aortic EC, eNOS, eNOS at serine 1179, eNOS-Ser(1179), endothelial cell(s), endothelial nitric oxide synthase, far-infrared, human umbilical vein EC, nitric oxide, protein kinase A, siRNA, small interference RNA, transient receptors potential

PMID:23756809
[PubMed - in process]

http://www.ncbi.nlm.nih.gov/pubmed/23756809