Acute hypoxia enhances proteins' S-nitrosylation in endothelial cells..

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摘要

Abstract

Hypoxia-induced responses are frequently encountered during cardiovascular injuries. Hypoxia triggers intracellular reactive oxygen species/nitric oxide (NO) imbalance. Recent studies indicate that NO-mediated S-nitrosylation (S-NO) of cysteine residue is a key posttranslational modification of proteins. We demonstrated that acute hypoxia to endothelial cells (ECs) transiently increased the NO levels via endothelial NO synthase (eNOS) activation. A modified biotin-switch method coupled with Western blot on 2-dimensional electrophoresis (2-DE) demonstrated that at least 11 major proteins have significant increase in S-NO after acute hypoxia. Mass analysis by CapLC/Q-TOF identified those as Ras-GTPase-activating protein, protein disulfide-isomerase, human elongation factor-1-delta, tyrosine 3/tryptophan 5-monooxygenase activating protein, and several cytoskeleton proteins. The S-nitrosylated cysteine residue on tropomyosin (Cys 170) and beta-actin (Cys 285) was further verified with the trypsic peptides analyzed by MASCOT search program. Further understanding of the functional relevance of these S-nitrosylated proteins may provide a molecular basis for treating ischemia-induced vascular disorders.

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