
Flow-mediated vasodilation is mediated by endothelium-derived nitric oxide

Letter to Editor: The question as to whether endothelium-derived nitric oxide is responsible for flow-mediated vasodilation is an important one (1). There are few techniques available to measure the bioactivity of nitric oxide in humans. One such technique that has gained considerable popularity among clinician scientists employs high-resolution vascular ultrasonography to measure the change in the brachial artery diameter after a flow stimulus, i.e., reactive hyperemia. Flow-mediated vasodilation of the brachial artery is abnormal in patients with risk factors for atherosclerosis, such as hypercholesterolemia, cigarette smoking, and diabetes mellitus, and has been shown to predict the risk of future cardiovascular events (3). My laboratory addressed the hypothesis that endothelium-derived nitric oxide is responsible for flow-mediated vasodilation a decade ago and reported its findings (2). Specifically, we measured the vasodilator responses of the brachial artery to flow (after 1 min of reactive hyperemia after an ischemic stimulus) and to intra-arterial infusions of acetylcholine and nitroprusside before and after administration of the nitric oxide synthase antagonist \( \text{L}-\text{NMMMA} \). \( \text{L}-\text{NMMA} \) inhibited flow-mediated vasodilation and the vasodilator response to acetylcholine but did not affect the response to nitroprusside. These observations enabled us to conclude that flow-mediated vasodilation of the brachial artery is an endothelium-dependent process in humans, mediated by nitric oxide. It is important to point out that Dr. Green cites our article in his point and rebuttal, whereas Drs. Tschakovsky and Pyke failed to acknowledge this contribution in their point and rebuttal. Issues of this import are enlightened by debate but are solved by carefully performed experiments. Our data support the notion that flow-mediated vasodilation of the brachial artery is mediated by endothelium-derived nitric oxide.

REFERENCES


Mark A. Creager
Harvard Medical School
Boston, Massachusetts
e-mail: mcreager@partners.org