Dietary nitrite supplementation protects against myocardial ischemia-reperfusion injury

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Nitrite has emerged as an endogenous signaling molecule with potential therapeutic implications for cardiovascular disease. Steadystate levels of nitrite are derived in part from dietary sources; therefore, we investigated the effects of dietary nitrite and nitrate supplementation and deficiency on NO homeostasis and on the severity of myocardial ischemia-reperfusion (MI/R) injury. Mice fed a standard diet with supplementation of nitrite (50 mg/liter) in their drinking water for 7 days exhibited significantly higher plasma levels of nitrite, exhibited significantly higher myocardial levels of nitrite, nitroso, and nitrosyl–heme, and displayed a 48% reduction in infarct size (Inf) after MI/R. Supplemental nitrate (1 g/liter) in the drinking water for 7 days also increased blood and tissue NO products and significantly reduced Inf. A time course of ischemia-reperfusion revealed that nitrite was consumed during the ischemic phase, with an increase in nitroso/nitrosyl products in the heart. Mice fed a diet deficient in nitrite and nitrate for 7 days exhibited significantly diminished plasma and heart levels of nitrite and NO metabolites and a 59% increase in Inf after MI/R. Supplementation of nitrite in the drinking water for 7 days reversed the effects of nitrite deficiency. These data demonstrate the significant influence of dietary nitrite and nitrate intake on the maintenance of steady-state tissue nitrite/nitroso levels
and illustrate the consequences of nitrite deficiency on the pathophysiology of MI/R injury. Therefore, nitrite and nitrate may serve as essential nutrients for optimal cardiovascular health and may provide a treatment modality for cardiovascular disease.

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