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Rapid Letter

Preischemic Selenium Status as a Major Determinant of Myocardial Infarct Size *In Vivo* in Rats

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ABSTRACT

Prospective epidemiological studies have shown that the incidence of numerous cardiovascular pathologies is correlated with body selenium status. However, it remains unclear whether selenium status also influences the outcome of myocardial infarction. The aim of the present study was to test whether dietary selenium intake affects myocardial necrosis induced by transient regional ischemia *in vivo* in rats. For this purpose, male Wistar rats received either a high-selenium (High-Se: 1.5 mg of Se/kg) or a low-selenium (Low-Se: 0.05 mg of Se/kg) diet for 10 weeks. Animals were subjected to 30 min of myocardial ischemia induced by coronary artery ligation followed by 60 min of reperfusion. Pre- and postischemic blood samples were collected for glutathione (GSH and GSSG) determination and for glutathione peroxidase (GSH-Px) assessment. Our results show that high-selenium intake reduces myocardial infarct size (High-Se: 25.16 ± 1.19% versus Low-Se: 36.51 ± 4.14%, $p < 0.05$), preserves postischemic GSH/GSSG ratio (High-Se: 1.37 ± 0.37 versus Low-Se: 0.47 ± 0.10, $p < 0.05$), increases plasma GSH-Px activity, and improves postischemic mean arterial pressure. In conclusion, preischemic body selenium status is a major determinant of the outcome of myocardial ischemia *in vivo* in rats probably because it influences the cellular redox status. *Antioxid. Redox Signal.* 6, 792–796.

INTRODUCTION

Selenium is a constituent of the enzyme glutathione peroxidase (Se-GSH-Px) (14). As Se-GSH-Px can reduce oxidative stress and limit platelet aggregation (16), high selenium status might theoretically be protective against cardiovascular disease (11). On this basis, for several decades, numerous experimental and epidemiological studies have attempted to determine whether body selenium status influences both the incidence and the severity/prognosis of cardiovascular disease.

The association between low selenium status and the incidence of ischemic heart disease was established more than 30 years ago for populations in areas such as Finland, where selenium intake was exceptionally low (15). However, several other studies, especially when carried out in North America, have not shown a clear relationship between cardiovascular risk and low selenium status (17, 21). The possible explanation for this discrepancy is that the influence of selenium on the incidence of ischemic heart diseases might only be relevant in populations with low selenium status, lower than the concentrations that are generally observed in the U.S.A., as well as in a large part of Europe (13).

As a matter of fact, selenium deficiency has been identified as a major factor in the etiology of certain nonischemic chronic heart failure syndromes, especially in very low-selenium soils such as eastern China (Keshan disease) and western Africa (7, 11). More recently, we have demonstrated

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