Beware of the pickle: health effects of nitrate intake

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TO THE EDITOR: In a recent paper by Bailey et al. (2), dietary nitrate supplementation was shown to reduce the oxygen cost during submaximal exercise and to increase the time to exhaustion during high-intensity exercise. These data confirm and expand on the surprising and original findings by Larsen et al. (4) of nitrate-induced improvement of exercise efficiency in humans. This newly discovered ergogenic potential of dietary nitrate will certainly inspire athletes to boost their performance and nutriceutical companies to boost their sales. But what are the safety concerns of nitrate supplementation?

What health risks could possibly be expected from the nitrate content of a—on first thought—normal dose of vegetables, such as beetroot or spinach? The daily ingested dose of nitrate of 4.16 mg/kg body wt as beetroot juice (2) or 6.2 mg/kg body wt as sodium nitrate (4) exceeds the acceptable daily intake (ADI) of 3.65 mg/kg body wt in both studies. The concern about nitrate is possible carcinogenicity. Nitrate and nitrite are probably not carcinogenic, but nitrite formed from dietary nitrate might react with dietary amines from proteins to form carcinogenic nitrosamines in the acidic gastric environment and is generally associated with intake of pickled or preserved food. The recommendation not to combine spinach with fish in some countries is also based on these findings. The ADI guidelines have a large safety margin, so the health risk of the used nitrate intake in the above-mentioned studies is probably very limited. However, the problem may enlarge when nitrate is combined with other nutritional supplements, for there is a growing and troubling tendency among supplement-selling companies to create “ergogenic cocktails” without sufficient scientific support for their efficacy or safety.

It can be anticipated that the combined intake of creatine and nitrate will be promoted by companies and adopted by athletes. Creatine in itself has been allegedly linked to cancer risk because creatine can react with nitrite to form the highly carcinogenic N-nitrososarcosine (NSAR) in vitro (1). Urinary NSAR levels correlate positively with esophageal cancer mortality rates (6). However, in vivo nitrosation of creatine is probably negligible, as urinary NSAR levels do not increase in people daily ingesting 20 g of creatine monohydrate (3). However, this does not exclude that the toxicological hazard becomes realistic when not only the nitrosation-prone substrate (creatine or protein) but also the nitrosation agent (nitrite) is increased by elevated dietary nitrate consumption above ADI, leading to elevated gastric and circulating nitrite levels (2).

Moreover, when considering vegetables as a nitrate source, vitamin C or other antioxidants are also present in the vegetables, which could partially limit the formation of nitrosamines in the gastrointestinal tract (5). Dietary nitrate supplementation as sodium nitrate, in absence of this “natural” antioxidant protection, could prove to be more harmful for human health, especially in conditions of enhanced protein or creatine intake.

The recent findings on nitrate (2, 4) are valuable in promoting our understanding of oxygen consumption regulation in contracting muscles. Yet, progress in our field can often have relevance to performance enhancement. The safety of new ergogenic supplements falls under our responsibility and should be subject to further investigation.

REFERENCES