Selenium Metabolism and Bioavailability

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ABSTRACT

Selenium (Se) is at once an essential and toxic nutrient that occurs in both inorganic and organic forms. The biological functions of Se are mediated through at least 13 selenoproteins that contain Se as selenocysteine (Se-cyst). The endogenous synthesis of this amino acid from inorganic Se (selenide Se⁻²) and serine is encoded by a stop codon UGA in mRNA and involves a unique tRNA. Selenium can also substitute for sulfur in methionine to form an analog, selenomethionine (Se-meth), which is the main form of Se found in food. Animals cannot synthesize Se-meth or distinguish it from methionine and as a result it is nonspecifically incorporated into a wide range of Se-containing proteins. The metabolic fate of Se varies according to the form ingested and the overall Se status of an individual. This paper reviews the bioavailability, including absorption, transport, metabolism, storage, and excretion, of the different forms of exogenous and endogenous Se.

Index Entries: Selenium; seleno-cysteine; selenoproteins; metabolism; bioavailability; transport; review.

INTRODUCTION

Selenium (Se) presents a nutritional conundrum through its dual status as an essential, yet highly toxic, nutrient. From early this century, Se has been known to cause toxicity in animals producing conditions such as "blind staggers" and "alkali disease" (1). In 1957, Schwarz and Foltz...
demonstrated trace amounts of Se protected against liver necrosis in vitamin E sufficient rats and hence established nutritional essentiality (2). A wide range of selenium-responsive deficiency conditions have been identified in young animals that graze on plant material growing in localized areas with low soil Se. Symptoms include exudative diathesis, pancreatic and hepatic fibrosis, and myopathy of skeletal and cardiac muscle known as white muscle disease (1).

The Se responsiveness of Keshan Disease, an endemic fatal cardiomyopathy, found in areas of China with particularly low soil Se, demonstrated human essentiality as recently as 1979 (3). Human Se deficiency has also been reported in patients on long-term parenteral nutrition (4,5) and preterm infants experience significant Se depletion (6,7). Generally however, overt Se deficiency in humans is relatively rare and many populations with very low Se intakes and blood levels show no apparent ill-effects. Nevertheless, in recent years, Se status has been implicated in a wide range of disorders, including heart disease and cancer (8).

Selenium is in the same group of the periodic table as sulfur and may substitute for sulfur in the sulfur-amino acids to form the Se analogs selenomethionine (Se-meth) and selenocysteine (Se-cyst) (8). These amino acids, particularly Se-meth, are the predominant form of Se in food (9). Inorganic Se (selenite and selenate with valencies of +4 and +6 respectively) is generally only included in the diet through supplements. Most of the biological functions of Se are mediated through selenoproteins that contain Se-cyst, which has been termed the twenty-first amino acid (10). Differences between the chemistry of Se and sulfur result in the sulfhydryl groups of cysteine being mostly protonated at physiological pH, whereas the analogous groups of Se-cyst are largely dissociated, which facilitates the catalytic role of Se in the selenoproteins (11). At least 13 different selenoproteins have now been identified (12,13), but only glutathione peroxidase (GSHPx), selenoprotein P, and Type I iodothyronine 5'-deiodinase (IDI) have been well characterized in animals. Although biological significance has yet to be determined for the majority of these proteins, they are likely to facilitate more complex antioxidant functions and wider, nonantioxidant roles for Se than are currently known (12,14), and are the focus of much research activity. The purpose of this paper is to review the metabolism of Se with particular reference to the impact of the form ingested and the selenoproteins.

SYNTHESIS AND REGULATION OF SELENOPROTEINS

Animals are able to endogenously synthesize Se-cyst from inorganic Se. They cannot synthesize Se-meth and apparently do not distinguish between methionine and its Se analog (13,15). Proteins containing endogenously synthesized Se-cyst are referred to as selenoproteins and