

# Selenium bioavailability: current knowledge and future research requirements<sup>1–5</sup>

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## ABSTRACT

Information on selenium bioavailability is required to derive dietary recommendations and to evaluate and improve the quality of food products. The need for robust data is particularly important in light of recent suggestions of potential health benefits associated with different intakes of selenium. The issue is not straightforward, however, because of large variations in the selenium content of foods (determined by a combination of geologic/environmental factors and selenium supplementation of fertilizers and animal feedstuffs) and the chemical forms of the element, which are absorbed and metabolized differently. Although most dietary selenium is absorbed efficiently, the retention of organic forms is higher than that of inorganic forms. There are also complications in the assessment and quantification of selenium species within foodstuffs. Often, extraction is only partial, and the process can alter the form or forms present in the food. Efforts to improve, standardize, and make more widely available techniques for species quantification are required. Similarly, reliable and sensitive functional biomarkers of selenium status are required, together with improvements in current biomarker methods. This requirement is particularly important for the assessment of bioavailability, because some functional biomarkers respond differently to the various selenium species. The effect of genotype adds a potential further dimension to the process of deriving bioavailability estimates and underlines the need for further research to facilitate the process of deriving dietary recommendations in the future. *Am J Clin Nutr* 2010;91(suppl):1484S–91S.

## INTRODUCTION

To derive selenium requirements and establish dietary recommendations for optimal health, estimates of selenium bioavailability are needed. A literature review on the bioavailability of selenium from foods was published in 2006 (1), and it highlights the dependence of bioavailability on food sources associated with different forms of selenium and emphasizes the importance of the assessment of bioavailability with the use of functional assays. Data on chemical speciation and metabolic transformations (in conjunction with information on the relation between selenium intake and status and health outcomes) are required to assess selenium bioavailability and the longer-term health consequences that result from different intakes.

## DIETARY REQUIREMENTS

The 1991 UK Dietary Reference Values (2) used data from older literature and estimated that between 55% and 65% of

dietary selenium is absorbed. The 1993 Population Reference Intakes published by the European Scientific Committee for Food (3) concluded that for selenium “all usual dietary forms are absorbed quite efficiently.” The 2000 report of the US Food and Nutrition Board (4) suggested that most dietary selenium is highly bioavailable: >90% of selenomethionine is absorbed; selenocysteine appears to be absorbed very well; ≈100% of selenate is absorbed, but a significant fraction is lost in the urine; and >50% of selenite is absorbed (depending on luminal interactions) and is better retained than selenate. There is clearly a need to review dietary recommendations in light of more recent data, in particular, information on dietary forms of selenium and the relationships between intake and health outcomes.

## SELENIUM SPECIATION

A recent review (5) provides information on the forms of selenium in food and associated health effects; technical approaches used for speciation have also been reviewed recently (6, 7). The analysis of forms of selenium in food is a challenging task; there are currently no methods that can reliably extract 100% of the selenium from foods without potentially affecting the species, and the techniques are established in only a few laboratories worldwide. Therefore, care has to be taken to extract as much selenium as possible while still retaining the form that is present in the food as consumed; conditions that are devised to maximize the extraction of selenium from a food matrix may cause changes in chemical form. Ideally, the measurements should be made in food that has gone through processing (eg, cooking) followed by simulated gastrointestinal digestion, be-

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cause this is the form present in the lumen of the gut that is of interest. Although it has not been possible to produce comprehensive data that describe forms of selenium in food, there are limited data on the percentage distribution of different species (expressed as percentage of extractible or total selenium); examples are given in **Table 1**.

The selenium content and species of both plant and animal foodstuffs depend on environmental conditions, in particular, the quantity and species of selenium to which the animal/plant is exposed (6, 24). Selenomethionine is predominant in cereals, and selenium concentrations vary from 0.01 to 0.55  $\mu\text{g/g}$  fresh weight (5), whereas in other plant foods the content is generally lower, with the exception of Brazil nuts and vegetables, which are selenium-accumulating plants, namely those in the allium and brassica families. The selenium content of Brazil nuts varies depending on soil content and other environmental factors, and nuts from trees in the central part of Brazil contain  $\leq 10$  times more selenium than those from West Brazil (6). The reason for the high content of selenium in Brazil nuts is that the proteins are high in sulfur-containing amino acids, and selenomethionine can nonspecifically replace methionine. The major species in non-selenium-accumulating plant foods are selenate and selenomethionine, plus smaller amounts of selenocysteine. In contrast, the predominant form of selenium in selenium-accumulating plants is  $\gamma$ -glutamyl methylselenocysteine (13, 14). There are limited data on the forms of selenium in animal foodstuffs, but it appears that the major forms are selenomethionine and selenocysteine, which are incorporated nonspecifically into muscle protein (19). In addition, selenate and selenite have been detected in fish (18, 20) and there appear to be large differences between fish species in relation to selenoproteins (25). In foods of animal origin, supplementation with organic compared with inorganic selenium results in meat of higher selenium concentration. For example, when a comparison is made between the effect of selenium yeast and sodium selenite supplements, skeletal muscle from lambs contained 0.12 and 0.08  $\mu\text{g}$  selenium/g fresh weight, respectively (26), and beef contained 0.41 and 0.30 mg/kg dry weight, respectively (27).

### ABSORPTION, RETENTION, AND METABOLISM

Data on selenium metabolism from different foods and selenium supplements indicate differences in the absorption and use of selenium between inorganic and organic forms in humans (28, 29) and rats (30). The absorptive pathways have not yet been fully characterized, but selenium as selenate or selenite appears to be very well absorbed but less well retained in the body than organic forms of selenium, such as selenomethionine and selenocysteine (31–33). The proposed metabolic pathways for different forms of selenium are shown in **Figure 1** (5). Most forms of selenium are efficiently absorbed, but subsequent metabolism depends on the form in which they are present in plasma. Selenomethionine, selenocysteine, selenate, and selenite enter the selenide pool and from here the selenium is either used for selenoprotein synthesis or excreted in the urine as a selenosugar. Selenomethionine can, however, also be incorporated directly (and nonspecifically) into proteins through the replacement of methionine. A separate pathway is followed by the organic compound,  $\gamma$ -glutamyl methylselenocysteine, found in brassica and allium vegetables, whereby it is first converted to Se-methylselenocysteine and

then transformed by  $\beta$ -lyase into methylselenol, which is primarily excreted in breath and urine but may also enter the selenide pool.

Several approaches have been used to measure the bioavailability of selenium in various foods, as summarized in **Table 2**. These include the measurement of changes in plasma selenium concentration, measurement of glutathione peroxidase (GPx) enzyme activity, and absorption/retention studies. For the last, intrinsic techniques with the use of stable isotopes of selenium have been developed to label the endogenous forms of selenium in foods (40). In general, selenium is absorbed efficiently, but it is not possible to assign specific figures for retention and use (bioavailability) to individual forms of selenium because of the complexity of many foods (Table 1). However, a study by Bügel et al (39), on the assumption that selenomethionine is the major form in meat, showed that most of the selenium was absorbed and just over half retained in the body (ie, not excreted in the urine). Selenium in Brazil nuts appeared to be better used than selenomethionine, in terms of the response of plasma selenium concentration and red blood cell GPx activities: the plasma selenium increase was similar despite the fact that the daily intake from Brazil nuts was half that from selenomethionine (35). Changes in selenium status that reflect changes in intake occur over a period of several weeks or months, although the feeding trial of Hawkes et al (38) showed a significant difference between a beef, rice, and powdered milk diet with low selenium content and one with high selenium content after only 14 d. In a study by Kirby et al (11), the plasma selenium response in a feeding trial appeared to be related to the form of selenium in wheat flour biscuits: intake of selenomethionine in biofortified wheat-biscuits resulted in a greater increase in plasma selenium after 6 mo than the oxidized selenomethionine (selenomethionine selenoxide) in fortified biscuits (Table 2).

### FUNCTIONAL MEASURES

There are 25 known selenoprotein genes in humans (41, 42), which encode selenoproteins with a variety of functions, as summarized in **Table 3**. Several of the selenoproteins, which include selenoproteins P and W and the GPx 1, 3, and 4, have been used widely as biomarkers of selenium status. Functional biomarkers are only useful if they can be measured in readily accessible tissues, such as blood. At present, the most promising biomarker appears to be selenoprotein P, which appears to reach a plateau after 2–4 wk of supplementation (88, 89) and is well correlated with plasma selenium across a wide range of selenium status (90), up to a plasma selenium concentration of  $\approx 125$  ng/mL (33). Selenoprotein P typically accounts for approximately half of the selenium in plasma (46). It is generally more sensitive than other selenoproteins, such as GPx, in both deficiency (90) and after supplementation (89–91), and, in addition, the response of selenoprotein P to different forms of selenium appears to be similar (92).

Biomarkers of selenium status have recently been the subject of a systematic review (93), in which the response of each biomarker to either depletion or supplementation (only studies that intervened with selenomethionine or selenium-enriched yeast were included) was assessed and evaluated for different population groups. However, for most biomarkers there was

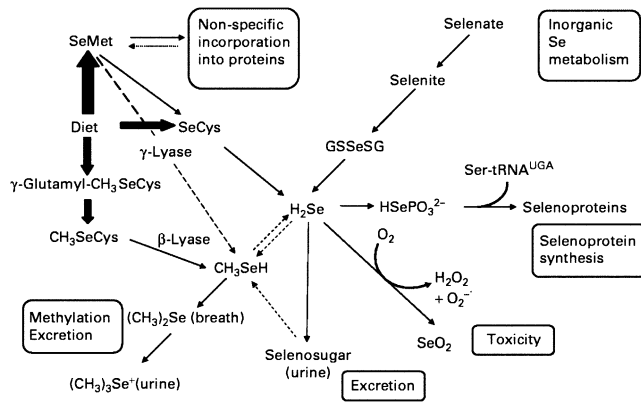


**TABLE 1**

Examples of forms of selenium (percentage of total or extractable selenium) in foods

Food (reference)	Typical selenium content <sup>1</sup>	Forms of selenium
	<i>µg/g fresh weight</i>	
Selenium-enriched yeast (5, 8)	1200–2200	60–84% Selenomethionine, usual percentage in high-quality commercial preparation of selenium-enriched yeast but values for selenomethionine content can vary: 23–83% Selenomethionine 3–21% Selenocysteine 1–20% $\gamma$ -Glutamyl-Se-methylselenocysteine 4% Selenate 13–51% Other forms
Brazil nuts ( <i>Bertholletia excelsa</i> ) (9)	2.54 (0.85–6.86)	≈25% Selenomethionine
Wheat (8, 10)	0.1–30 0.08–44	12–19% Selenate/ite 56–83% Selenomethionine 4–12% Selenocysteine 1–4% Se-methylselenocysteine ≈55% Selenomethionine
Wheat (biofortified) (11)	8.3	76–85% Selenomethionine
Wheat-flour (biofortified) biscuits (11)	4.4	76–85% Selenomethionine
Wheat flour (unfortified) soaked in aqueous solution of selenomethionine and baked into biscuits (11)	8.5	55% Selenomethionine selenoxide 5% Selenomethionine
Broccoli (selenium enriched) (12)	62.3 <sup>2</sup>	45% Se-methylselenocysteine 20% Selenate 20% Selenate 12% Selenomethionine
Onions ( <i>Allium cepa</i> ) (13)	<0.5	100% Selenate (extractable selenium)
Onions (selenium enriched) (13)	140	63% $\gamma$ -Glutamyl-Se-methylselenocysteine 10% Selenate 5% Selenomethionine
Garlic ( <i>Allium sativum</i> ) (13)	<0.5	53% Selenomethionine 31% $\gamma$ -Glutamyl-Se-methylselenocysteine 12% Se-methylselenocysteine 4% Selenate
Garlic (selenium enriched) (14)	296	73% $\gamma$ -Glutamyl-Se-methylselenocysteine (total eluted selenium) 13% Selenomethionine 4% $\gamma$ -Glutamyl-selenomethionine 3% Se-methylselenocysteine 2% Selenate
Lentils ( <i>Lens culinaris</i> L.) (15)	0.24–0.36	90% Organic selenium 10% Selenate
Carrots (16)	<0.05	Undetectable
Carrots (selenium enriched) (16)	0.4–2.2	Selenium-enriched with the use of selenate (% extractable): ≈54% Selenomethionine 32% Selenate ≈14% $\gamma$ -Glutamyl-selenomethionine Selenium-enriched with the use of selenite: ≈71% Selenomethionine 17% Selenite ≈12% $\gamma$ -Glutamyl-selenomethionine
Potatoes (17)	0.12	50% Selenomethionine (extractable) 50% Selenate (extractable)
Shellfish (18)	0.36–1.33	7.6–44.8% Selenate
Cod (19, 20)	1.5	70% Selenomethionine 12% Selenite
Tuna (canned in water) (21)	5.6	29% Selenomethionine (extractable)
Shark (21)	2.0	56% Selenomethionine (extractable)
Swordfish (22)	Not quantified	Selenomethionine, selenenyl sulfide, selenite
Chicken (23)	0.5	56–66% Selenomethionine (extractable) 20–31% Selenocysteine (extractable)
Lamb (23)	0.4	56–60% Selenomethionine (extractable) 50% Selenocysteine (extractable)

<sup>1</sup> Values are means and/or ranges.<sup>2</sup>  $\mu\text{g/g}$  dry weight.



**FIGURE 1.** Metabolic pathway of dietary selenium in humans. Se, selenium; SeMet, selenomethionine; SeCys, selenocysteine; GSSeSG, selenodiglutathione; γ-glutamyl-CH<sub>3</sub>SeCys, γ-glutamyl-Se-methylselenocysteine; H<sub>2</sub>Se, hydrogen selenide; HSePO<sub>3</sub><sup>2-</sup>, selenophosphate; CH<sub>3</sub>SeCys, Se-methylselenocysteine; CH<sub>3</sub>SeH, methylselenol; (CH<sub>3</sub>)<sub>2</sub>Se, dimethyl selenide; SeO<sub>2</sub>, selenium dioxide; (CH<sub>3</sub>)<sub>3</sub>Se<sup>+</sup>, trimethyl selenonium ion. Reproduced with permission from reference 5.

a paucity of data for meaningful subgroup or dose-response analysis. In the included studies plasma selenium was the most commonly measured biomarker, and it responded positively to intervention, as did whole-blood and erythrocyte selenium, plasma selenoprotein P, and platelet, plasma, erythrocyte and whole-blood GPx activity, albeit with significant heterogeneity in each case. The review concluded that further large-scale interventions are required to assess the usefulness of selenium-

responsive biomarkers, and these could conceivably include aspects of speciation. Plasma selenium concentration reflects dietary exposure to most forms of selenium, but in the absence of a well-described homeostatic regulation there is no absolute plateau, although the concentration will reach a steady state at any constant level of intake after ≈10–12 wk (33, 91, 92, 94–97). In addition to dose, the plasma response to dietary selenium is species dependent, so consumption of 2 different forms may result in different plasma selenium concentrations (33, 92, 95, 96, 98, 99).

**EFFECT OF GENOTYPE**

The response by individuals to 6 wk of selenium supplementation with 100 μg sodium selenite/d has been shown to be influenced by genetic polymorphisms in the selenoprotein P gene (*SEPP*) (100) and *GPX4* gene (101). Biomarkers that are commonly used to assess selenium bioavailability (plasma selenium, selenoprotein P, and GPx3) were associated with 2 common single nucleotide polymorphisms in *SEPP* in both baseline and postsupplementation samples (100). The *GPX4* polymorphism was shown to influence lymphocyte GPx4 concentration and other selenoproteins in vivo (101). A single nucleotide polymorphism in GPx1 (Pro198Leu) was associated with selenium deficiency and impaired GPx1 activity (102) and also may be associated with a different response of GPx1 activity to selenium (103). This observation raises the issue of whether common polymorphisms in selenoprotein genes, such as *SEPP*, *GPX1*, *GPX4*, and selenoprotein S (*SELS*) (92), will

**TABLE 2**  
Bioavailability of selenium from various foods<sup>1</sup>

Food (reference)	Technique used	Results
Selenium (Se)-yeast, 300 μg/d for 10 wk, then single dose of <sup>77</sup> Se-yeast (34)	Absorption from stable isotopically labeled material (327 μg selenium)	89%
	Retention (absorption minus urinary excretion)	74%
Brazil nuts, 53 μg/d for 3 mo (35)	Plasma selenium increase	64.2%
	Plasma GPx increase	8.2%
	Whole-blood GPx increase	13.2%
Selenomethionine, 100 μg/d for 3 mo (35)	Plasma selenium increase	61%
	Plasma GPx increase	3.4%
	Whole-blood GPx increase	5.3%
Biofortified wheat-flour biscuits, mean intake 172 μg/d for 6 mo (11)	Plasma selenium increase after 6-mo feeding trial	72-μg/L increase
Fortified wheat-flour biscuits, mean intake 208 μg/d for 6 mo (11)	Plasma selenium increase after 6-mo feeding trial	16-μg/L increase
Basal diet, 52 μg selenium + cow milk, 15 μg selenium (36)	Fractional absorption in ileostomists	65.5%
		73.3%
Shrimp, 88 μg/d for 6 wk (37)	Plasma selenium increase	6.3-μg/L increase
	Apparent absorption	83%
Beef, rice, and powdered milk, 14 μg/d (low) compared with 297 μg/d (high) for 14 d (38)	Plasma selenium change	-40 μg/L (low); 97 μg/L (high)
	Muscle selenium	-0.37 μg/g protein (low); 0.57 μg/g protein (high)
	Platelet GPx	-120 nkat/g protein (low); 100 nkat/g protein (high)
	Red blood cell selenium	-42 μg/L (low); 106 μg/L (high)
	Red blood cell GPx	-15 nkat/g protein (low); 13 nkat/g protein (high)
Pork, 106 μg/d for 3 wk; 7 d metabolic balance in final week (39)	Apparent absorption	94%
	Retention	58%

<sup>1</sup> GPx, glutathione peroxidase; nkat, nanokatal.

**TABLE 3**Summary of human selenoproteins and their functions<sup>1</sup>

Group/name (reference)	Abbreviation(s)	Location	Main functions
<b>Glutathione peroxidases</b>			
Glutathione peroxidase-1 (43)	GPx1, cGPx, GSH-Px	Widely expressed throughout the body, intracellular enzyme	Cystolic enzyme, antioxidant activity
Glutathione peroxidase-2 (44)	GPx2, GI-GPx	Mainly in gastrointestinal tissue, also in liver	Protection of GI tract from oxidative damage
Glutathione peroxidase-3 (45)	GPx3, eGPx	Plasma [accounts for 10–30% selenium in plasma (46)] and extracellular fluid, expressed in liver, kidney, heart, lung, thyroid, GI tract, and breast (47)	Plasma antioxidant, can decrease lipid hydroperoxides (48)
Glutathione peroxidase-4	GPx4, PHGPx	Widely expressed, high expression in the testes (49, 50); cytosolic and membrane-bound forms (51, 52)	Antioxidant activity, protects membranes from peroxidative degradation (51); can decrease phospholipid, cholesterol and cholesterol ester hydroperoxides to less toxic derivatives (52); protection against oxidatively damaged DNA (53); regulation of 15-lipoxygenase pathway (54) and 5-lipoxygenase (55); important for male fertility and sperm maturation/function/motility (56–59)
Glutathione peroxidase-6 (41)	GPx6	Embryo and olfactory epithelium	Unknown
<b>Thioredoxin reductases</b>			
Thioredoxin reductase-1 (60, 61)	TrxR-1, TR1, Txnrd1	Intracellular (cytosolic/nuclear), widely distributed	Regulation of intracellular redox state, cell signaling; decreases thioredoxin
Thioredoxin reductase-2 (60, 61)	TrxR-2, TR2	Mitochondrial, widely distributed	Regulation of intracellular redox state; decreases thioredoxin
Thioredoxin reductase-3 (60, 61)	TrxR-3, TR3	Testis-specific	Regulation of intracellular redox state
<b>Iodothyronine deiodinases</b>			
Iodothyronine 5' deiodinase-1, type 1	DIO-1, DI1, 5'IDI	Kidney, liver, thyroid, and brown adipose tissue (62–64)	Thyroid hormone metabolism, converts inactive thyroxine to active 3,3'-5'triiodothyronine; activation of thyroid hormones (65)
Iodothyronine 5' deiodinase-2, type 2 (63, 66, 67)	DIO-2, DI2	Thyroid, CNS, pituitary, brown adipose tissue, skeletal muscle	Activation of thyroid hormones
Iodothyronine 5 deiodinase-3, type 3 (68)	DIO-3, DI3	Placenta, CNS, fetus	Inactivation of thyroid hormones
<b>Selenoproteins and other</b>			
Selenoprotein-P (69, 70)	SeIP, Sepp1	Plasma [accounts for 30–50% of selenium in plasma (46, 71)] and also ubiquitously expressed in most tissues; high expression in brain, liver, and testes	Selenium homeostasis (72) and transport of selenium to tissues; antioxidant activity and decrease of lipid hydroperoxides (73)
Selenoprotein-W (74, 75)	SeIW	Most tissues, abundant in brain, colon, heart, skeletal muscle, and prostate	Involved in skeletal and cardiac muscle metabolism/function, antioxidant function
Selenoprotein-N	SeIN	Most tissues, ubiquitous expression, transmembrane glycoprotein associated with endoplasmic reticulum (76, 77)	Unknown, may be important in muscle and development (76)
Selenoprotein-S	SeIS	Membrane protein, located in the endoplasmic reticulum, widely expressed	Inflammatory response, regulation of inflammatory cytokines (interleukin 1 $\beta$ and 6 and tumor necrosis factor alpha) (78), removal of misfolded proteins from the endoplasmic reticulum (79)
Selenoprotein-K (80)	SeIK	Membrane protein, localized to endoplasmic reticulum	Possible antioxidant activity
Selenoprotein-R (81)	SeIR / MsrB1	Cytosol and nucleus; widely expressed	Antioxidant, protein repair and methionine metabolism (82)
Selenoprotein-H (83)	SeIH	Widely expressed in tissues, localized to the nucleus	DNA binding protein, regulation of glutathione synthesis genes, and phase II detoxification
Selenoprotein-I (41)	SeII	Unknown	Unknown

(Continued)

TABLE 3 (Continued)

Group/name (reference)	Abbreviation(s)	Location	Main functions
Selenoprotein-M (84)	SeIM	Localized in the endoplasmic reticulum	Protein folding in the endoplasmic reticulum, antioxidant activity
Selenoprotein-O (41)	SeIO	Unknown	Unknown
Selenoprotein-T (41)	SeIT	Unknown	Unknown
Selenoprotein-V (41)	SeIV	Testes	Unknown
15 kD selenoprotein (85)	SeI15	Localized in the endoplasmic reticulum	Thioredoxin-like, role in unfolded protein response (86)
Selenophosphate synthetase-2	SPS-2	Unknown	Selenoprotein biosynthesis (87)

<sup>1</sup> The list of selenoproteins that contain selenocysteine was generated from information on the selenoprotein database SelenoDB (42). Glutathione peroxidases 5, 7, and 8 (GPx5, GPx7, GPx8); selenoproteins R2, R3, and W2 (SeIR2, SeIR3, SeIW2); selenium-binding protein 2 (SBP2); selenophosphate synthetase1 (SPS1); and eukaryotic elongation factor (eEFSec) are not listed in the table of selenoproteins because they are homologs that contain cysteine or other amino acids that do not contain selenocysteine (SelenoDB). CNS, central nervous system; GI, gastrointestinal.

have a significant effect on the metabolism of dietary selenium and will generate different figures for bioavailability. However, it is most likely that the effect of genotype on the biomarkers used to predict bioavailability is subtle and only becomes relevant when longer-term health outcomes are considered (104–106).

## RESEARCH REQUIREMENTS

The bioavailability of different selenium species requires further investigation with the use of stable isotope labels, and the mechanism of absorption of the different forms of selenium needs to be elucidated. Further data on selenium species in food are required, but with the well-known extraction constraints it will not be possible to generate comprehensive information for all foods; therefore, dietary intervention studies may be required to study foods that make a major contribution to selenium intake. The native forms of selenium need to be labeled intrinsically with stable isotopes of selenium to measure uptake and retention of food selenium in acute studies, and longer-term studies need to be undertaken to measure changes in functional biomarkers; the most promising at present is selenoprotein P, but other novel biomarkers should be sought. Interactions between selenium and other micronutrients, such as vitamin E, should be taken into consideration [possibly with the use of a network biology approach (107)], particularly in relation to health outcomes that are associated with antioxidant nutrients, such as inflammation. Finally, the effect of common selenoprotein gene polymorphisms on metabolism (and hence requirements) remains to be clarified.

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## Erratum

Fairweather-Tait SJ, Collings R, Hurst R. Selenium bioavailability: current knowledge and future research. *Am J Clin Nutr* 2010;91(suppl):1484S–91S.

In the print version of this article, the term “selenium-methylselenocysteine” appears in error. The correct term, as provided by the authors, is “Se-methylselenocysteine.” This error occurs in Table 1 on page 1486S, in the legend to Figure 1 on page 1487S, and in text on page 1485S. The correct term appears in the online version.

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## Erratum

Morris MS, Jacques PF, Rosenberg IH, Selhub J. Circulating unmetabolized folic acid and 5-methyltetrahydrofolate in relation to anemia, macrocytosis, and cognitive test performance in American seniors. *Am J Clin Nutr* 2010;91:1733–44.

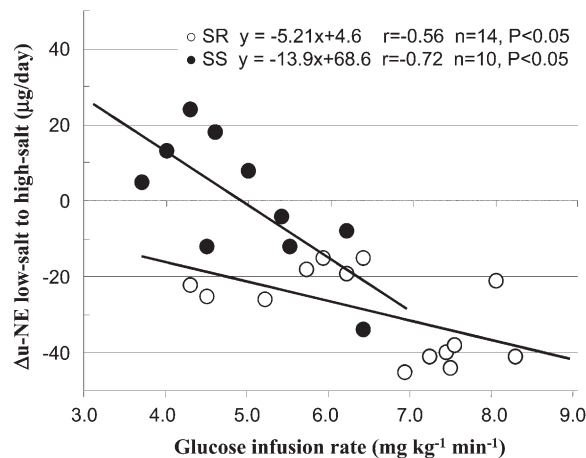
On page 1735, the last sentence of the first paragraph of the section entitled “Classification of subjects according to vitamin B-12 status and folate fractions” contains a detection limit. The published detection limit for the assay is 0.18 nmol/L, not 0.027 nmol/L. The incorrect limit was also given in the footnotes to Table 1 and Table 4. There were 7 eligible subjects with measured serum folic acid concentrations between 0 and 0.18 nmol/L who were included in the group with detectable unmetabolized folic acid. In addition, in Table 4, the multivariate model was not specified for the row labeled “Mean cell volume.” The results in that row were controlled for sex, age, race-ethnicity, current smoking, current alcohol intake, BMI, self-reported cancer history, and serum concentrations of ferritin, cystatin C, and C-reactive protein.

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## Erratum

Yatabe MS, Yatabe J, Yoneda M, et al. Salt sensitivity is associated with insulin resistance, sympathetic overactivity, and decreased suppression of circulating renin activity in lean patients with essential hypertension. *Am J Clin Nutr* 2010;92:77–82.

In the print version of this article, 2 lines appear for each group represented in Figure 7; however, only one line should appear for each group. The correct version of Figure 7 appears below. The online version of this figure is correct.



**FIGURE 7.** Relation between glucose infusion rate and change in urinary norepinephrine ( $\Delta u$ -NE) from the low- to high-salt diet in salt-resistant (SR) subjects ( $n = 14$ , open circles) and salt-sensitive (SS) subjects ( $n = 10$ , filled circles) with essential hypertension. Statistical significance was estimated with Pearson’s correlation coefficient.

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