Does selenium accumulation in meat confer a health benefit to the consumer?

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Abstract

Selenium is an essential nutrient with many potential health benefits for humans, including protection against some cancers, enhancement of neuropsychological function, and maintenance of a healthy immune system. Dietary Se is present in a variety of chemical forms and many of its biological actions depend on the chemical form of Se that is consumed. Meat and meat products, especially beef, are a primary source of dietary Se for humans. However, the Se content of meat may vary depending on the Se content of the soil, the feedstuffs grown on that soil, and whether the animal has been supplemented with Se. As a result, the actual contribution of Se from meat to an individual's diet may be quite variable. The bioavailability of Se has traditionally been assessed by feeding different forms of Se to Se-deficient animals and measuring the increases in selenoprotein activities (e.g., glutathione peroxidase) and tissue accumulation of Se. When Se bioavailability from meat is assessed using this method, meat is considered a highly bioavailable source. However, when bioavailability is assessed in such a manner it does not address many of the other important health benefits of Se such as cancer protection and improvement of neuropsychological function. Consequently, specific studies must be conducted to determine whether the Se in meat does more than merely increase Se status and whether Se in meat can improve health.

Key Words: Selenium, Meat, Cancer, Selenomethionine, Seleno Amino Acids

Introduction

Selenium is a nutrient of considerable interest to the livestock industry. Initial interest developed when domestic animals grazing the high plains of Wyoming and the grasslands of the Dakotas were reported to have problems characterized by the loss of hair from mane and tails, emaciation, sloughing of hooves, and lameness. This condition was termed alkali disease and was ultimately ascribed to chronically high intakes of Se (Levander, 1986).

Interest in the essentiality of Se began with Schwarz and Foltz (1958), who reported that Se was the active ingredient in "Factor 3," and it protected against liver necrosis in rats and pigs and exudative diathesis in chickens. Selenium was subsequently found to prevent white muscle disease in calves and lambs (Oldfield et al., 1978).

During the 1970s and 1980s the basic biochemistry of Se was characterized. Selenium was found to be a component of the active site of glutathione peroxidase (**GSH-Px**) (Rotruck et al., 1973), where it was present in the form selenocysteine. Selenocysteine was subsequently found to become incorporated into GSH-Px by a unique mechanism that used UGA as the encoding codon (Leinfelder et al., 1988). Epidemiological data collected during this period also first hinted at the anticarcinogenic properties of Se (Clark, 1985; Stampfer et al., 1987), and animal studies clearly established a connection between Se intake and decreased risk of some cancers (Ip et al., 1991; Vadhanavikit et al., 1993; Ganther and Lawrence, 1997). In the 1990s a number of studies demonstrated the health benefits of Se, and their results elicited editorials in major medical journals in the United States (Taylor and Albanes, 1998) and Europe (Rayman, 1997), questioning whether immmediate action should be taken to increase Se intakes.

Selenium and Human Health

The possible anticarcinogenic properties of Se resulted in several large nutritional intervention studies in humans. For example, in the Linxian area of the People's Republic of China, which had one of the world's highest rates of gastric and esophageal cancer, various combinations of nutritional supplements were given for 6 yr to 29,584 subjects (Blot et al., 1993). Only Se, ß-carotene, and vitamin E (administered together) significantly decreased the relative risk and mortality of cancer. Clark et al. (1996) supplemented subjects having a previous history of skin cancer with a placebo or 200 µg Se/d provided as high-Se yeast for 10 yr. Supplemental Se depressed the relative risk of lung cancer to less than 60% of controls and reduced risk of colorectal and prostate cancers to around 40% of controls. More recently, an epidemiologic study concluded that Se concentrations were lower in the prediagnostic toenail clippings of men with prostate cancer compared to healthy men (Yoshizawa et al., 1998).

In addition to cancer protection, Se in selenoproteins is part of the body's antioxidant system (Chen et al., 1993; Olin et al., 1994; Burk et al., 1995; Yeh et al., 1997), selenoproteins control thyroid hormone metabolism through the action of several deiodinase proteins (Berry and Larsen, 1995), and a selenoprotein is necessary for proper sperm function (Niemi et al., 1981; Scott et al., 1998). More recently, Se has been found to be necessary for maintaining a healthy immune system (McKenzie et al., 1998). When a coxsackie virus is injected into a Se-deficient rat and a Se-sufficient rat, the virus from the Se-deficient animal mutates in a manner that results in a more virulent form of the virus (Beck and Levander, 1998). This suggests that Se-deficient immune systems may be compromised to the extent that they allow pathogens to mutate into even more virulent strains.

Selenium may also benefit neuropsychological function. Healthy young men consumed diets that supplied either 33 or 226 μg Se/d for 105 d (Finley and Penland, 1998). The high-Se diet was supplied by Se-rich foods, of which a substantial portion was beef. People consuming the high-Se diet reported significantly improved mood; specifically, they reported being more clearheaded and less confused, more elated and less depressed, more composed and less anxious, and more confident and less unsure. Similar findings have been reported by at least two other research groups (Benton and Cook, 1990; Hawkes and Hornbostel, 1996).

Chemical Forms of Selenium and Selenium Metabolism

Results from human trials provide compelling reasons to increase one's Se intake. However, various foods may supply different chemical forms of Se, and different chemical forms will be processed by separate metabolic pathways. Thus, the health benefits of supplemental Se may depend on the chemical form of Se present in the food or supplement. The primary inorganic forms of Se are salts such as sodium selenite and sodium selenate; these salts are often used in human and animal supplements. Most foods, however, contain organic forms of Se, the most common being amino acids in which Se substitutes for the sulfur of methionine or cysteine, forming selenomethionine (**SeMet**) or selenocysteine (**SeCys**) (Levander, 1983). A third class of organic Se is found in certain plants, including brassica species. These plants contain many methylated forms of Se, including Se-methyl selenocysteine (**SeMSC**) (Cai et al., 1995).

A generalized scheme of Se metabolism is shown in Figure 1. Particular physiologic functions are mediated by specific forms of Se. Antioxidative and thyroid hormone-regulatory functions are performed by selenoproteins that require Se as SeCys at their active sites (Burk and Hill, 1993). Apparently the cancer-preventive properties of Se are not associated with selenoprotein expression, although the most recently discovered selenoprotein, thioredoxin reductase, is involved in cell growth regulation (Gallegos et al., 1997). Rather, Se may be associated with methylated forms of Se produced in the excretory pathway (Ip et al., 1991). The accumulation of Se in tissues such as muscle is a consequence of a third metabolic fate; SeMet may randomly substitute for methionine in general body proteins. The SeMet form seems to be able to effectively substitute for methionine (Whanger, 1986; Butler et al., 1989).

A pivotal point of Se metabolism apparently is the reduced form, selenide. Selenide is available for the formation of SeCys and subsequent insertion into selenoproteins. It can also enter the excretory pathway and become sequentially methylated (Foster et al., 1986; Ganther, 1986). Inorganic salts of Se can easily be converted to selenide through a nonenzymatic process, whereas SeMet must undergo enzymatic transformation to SeCys before a specific lyase can cleave Se to form selenide (Foster et al., 1986). Methylated forms of Se are excreted in the breath and urine, but depending on Se status, this pathway may run in reverse and be sequentially demethylated until the selenide is formed (Ganther, 1986).

Consequently, the point at which a particular chemical form of Se enters the metabolic pathway partially determines its ultimate fate. Inorganic salts are readily incorporated into specific selenoproteins and(or) enter the excretory pathway (Sunde and Hoekstra, 1980; Beilstein and Whanger, 1988). Selenocysteine may be cleaved with the Se subsequently becoming available for selenoprotein synthesis or entering the methylation/excretory pathway (Sunde and Hoekstra, 1980). Selenium-methyl selenocysteine can be cleaved to directly form monomethyl Se (Foster et al., 1986). Selenomethionine, the form found in most foods, can be metabolized to SeCys and then cleaved to form selenide, or the SeMet can directly substitute for methionine and incorporate into general proteins (Deagen et al., 1987; Waschulewski and Sunde, 1988b; Butler et al., 1989). The direction of SeMet metabolism may be determined in part by the sulfur amino acid status of the organism (Waschulewski and Sunde, 1988a; Butler et al., 1989).

Selenium in Foods

On average, the majority of the Se consumed in the North American diet comes from wheat and meat products. Beef is the single greatest contributor to dietary Se intake and accounts for approximately 17% of the total consumed (Schubert et al., 1987; Holden et al., 1991). Other foods contribute varying amounts, depending on where they are raised (see below). Brazil nuts are particularly rich in Se (Ip and Lisk, 1994a). Most fruits and vegetables supply relatively little Se, but that may also vary depending on where they are grown (Finley et al., 1996).

Selenium also may be consumed as a dietary supplement. Some supplemental Se is supplied as inorganic salts, but the most common supplement uses high-Se yeast that contains SeMet and other uncharacterized forms of Se (Alfthan et al., 1991; Butler and Whanger, 1992; Clark et al., 1996; Reilly, 1998).

The Se concentration in a food is primarily determined by its geographical origin. Foods from the shelves of a single grocery store were analyzed for Se content by hydride generation atomic absorption spectrometry (HGAAS) (Table 1; Finley et al., 1996). Although nutritional databases often provide values for Se in selected foods, data in Table 1 show that an average value is meaningless. For example, corn masa mix produced by one company averaged 0.047 μg Se/g, but the mix from another company had a 10-fold higher concentration of Se (0.50 μg/g). These differences probably reflect the geographic origin of the corn; corn from the Dakotas could be quite high in Se, whereas corn from the Ohio River basin would be quite low (Snook et al., 1987). Wheat products are considered to be a primary source of Se, yet the Se content of all-purpose white flour varied 300%, again probably reflecting a difference in the origin of the wheat.

Beef is considered to be a major source of dietary Se (Holden et al., 1991), but this may be confounded by variability in the Se content of edible beef products. Although there are limited reports of the Se content of beef, a study reported by Finley et al. (1996) demonstrated that the concentration of Se in ground beef varied depending on its geographical origin. Ground beef from animals raised in eastern North Dakota averaged 0.336 μg Se/g beef, but ground beef from Central Virginia contained 0.083 μg Se/g beef, and beef from the mountainous region of southwestern Missouri averaged only 0.060 μg Se/g beef (Table 1). Differences in the Se concentrations of beef probably result from a combination of different production practices (e.g., availability of trace mineralized salt) and geographical origin. Variation caused by geographical origin is a reflection of the soil Se concentration, and that is determined primarily by the Se content of the underlying rock strata. Seleniferous shale underlies much of the Dakotas, and where this is exposed the soil may have quite high concentrations of Se.

To investigate the connection between geographical area, geology, and the Se content of beef, cull cows were purchased from five locations throughout North Dakota. The locations were selected primarily because of the potential presence or absence of underlying seleniferous rock strata. Tissue samples were collected when animals were slaughtered and later analyzed for Se (Table 2; unpublished observations). The eastern location was selected as a control because it did not have underlying Se-containing rock, and because of scattered reports of possible Se deficiency. Tissue samples from the control area had Se values similar to the national average; however, other areas of the state had averages as much as threefold higher, and individual animals were higher yet. Based on a national average red meat consumption of approximately 57 kg/yr (Gerrior and Zizza, 1994), daily intakes of Se from beef from these areas would range from 40 μg/d (about half of the U.S. RDA) for the low-Se area to 100 μg/d (well above the U.S. RDA) for the high-Se area.

It is not known how much Se can accumulate in beef without inducing selenosis in the animal. Table 3 shows the Se concentrations of various agricultural products and Se accumulator plants collected on a ranch with a history of selenosis. Of particular note are the high concentrations of Se in alfalfa hay; feeding of alfalfa was probably the major reason for the selenosis problem. The highest Se concentrations were confined to a relatively small area of the ranch, and by removing 10.1 hectares of hayland from production the ranch was able to almost completely eliminate problems with Se toxicity.

Bioavailability of Selenium

Selenium bioavailability has classically been determined by measuring the repletion of tissue Se concentrations and GSH-Px activities in Se-deficient rats (Levander, 1983; Shi and Spallholz, 1994a,b; Wen et al., 1997). Although this method is generally accepted to provide good estimates of Se bioavailability, recent information on health benefits of Se shows the need for alternate measures. The main impetus for improving Se intakes in humans today is its cancer-protective ability (Clark et al., 1996); however, cancer protection is neither associated with selenoprotein production nor concentrations of Se in tissues (Ip et al., 1991). Consequently, foods with highly bioavailable Se, as determined by saturation of tissue Se concentrations and(or) GSH-Px activities, may not provide optimal cancer protection, whereas cancer protection may be greater in foods with lower (classically determined) bioavailability.

The bioavailabilities of Se salts and SeMet have been extensively studied. Selenium salts provide for a more immediate increase in GSH-Px activity in Se-deficient animals, whereas SeMet, because of its ability to incorporate into non-selenoproteins, is more effective in raising tissue Se status (Beilstein and Whanger, 1988). The incorporation of Se into non-selenoproteins was demonstrated by an increased retention of Se in muscle when SeMet, as compared to salts of Se, was the dietary source (Whanger and Butler, 1988). Chromatography studies have also demonstrated that Se from SeMet or salts is distributed among different proteins (Beilstein and Whanger, 1986a,b; Whanger, 1986; Butler et al., 1990). Human studies have demonstrated some of the same differences between SeMet and selenite distribution (Beilstein and Whanger, 1986a,b; Whanger, 1986; Butler et al., 1990).

Although the chemical form of Se certainly influences its metabolic pathway, the amount of dietary Se, and the Se status of the animal, also have major effects. Consequently, the ultimate fate of a Se-compound depends on an interaction between its chemical form and Se status of the organism (Butler et al., 1990). Thomson et al. (1978) demonstrated that Se from SeMet was retained better than Se from selenite in New Zealand women but that Se retention from selenite was improved when it was consumed in very small doses. The greater retention of SeMet, as compared to salts of Se, has been confirmed in studies conducted among a Se-deficient population of the People's Republic of China (Xia et al., 1992).

Differences in human retention of Se from SeMet and salts of Se are a consequence of altered distribution among selenoproteins. Deagen et al. (1991) showed chromatographically that plasma Se was primarily distributed between selenoprotein P, extracellular GSH-Px, and albumin. In erythrocytes, Se was in hemoglobin and GSH-Px (Butler et al., 1991). More Se was retained when SeMet was the dietary source, but that was a consequence of increased deposition into hemoglobin and albumin (i.e., not specific selenoproteins). Chinese men with very low Se intakes were supplemented with 200 μg Se/d supplied as SeMet or selenate (Xia et al., 1992). Selenium from SeMet, compared to selenate, accumulated faster in their erythrocytes and plasma, but less Se from SeMet was associated with GSH-Px.

Many human Se supplementation studies have used high-Se yeast as a supplemental form of Se. Although the chemical forms of Se in yeast are not fully characterized, most Se-researchers assume a large proportion of the Se is SeMet (Alfthan et al., 1991). Similar to studies using SeMet, more Se from yeast than from inorganic Se accumulated in the erythrocytes of Finnish men (Alfthan et al., 1991), although platelet GSH-Px activity increased more with inorganic Se. High-Se yeast has other biological activities, because it was the form of supplemental Se used in the cancer trial of Clark et al. (1996). High-Se yeast given as a supplement to healthy adults also increased blood Se concentrations and concentrations of Se in plasma selenoproteins (Tarp et al., 1986).

Bioavailability of Selenium in Meat

The bioavailability of Se in meat from domestic animals and fish has been assessed in human and animal models; the Se from fish is apparently utilized differently from Se from red meat. Douglass et al. (1981) repleted Se-deficient rats with Se from wheat, beef kidney, selenite, or tuna and demonstrated that Se from tuna was only 54 to 58% as effective as selenite in restoring liver GSH-Px activity. Meltzer et al. (1993) measured the response of human platelet GSH-Px to supplemental Se supplied as fish or wheat and concluded that Se from fish had poor bioavailability, although it must be noted that the test subjects had relatively high Se status. Wen et al. (1997) used Se-deficient rats to compare the bioavailability of Se from a number of foods, including tuna and flounder, and found Se from both types of fish effectively restored liver GSH-Px, but tuna-Se was ineffective in restoring muscle Se concentrations.

In contrast to fish, most studies have found Se in red meat to be highly bioavailable as determined by restored GSH-Px activity and tissue Se concentrations in Se-deficient rats. Shi and Spallholz (1994a) demonstrated that Se in raw or cooked beef was 127 to 139% as bioavailable as selenite. Various cuts of beef were found to have similar bioavailabilites (Shi and Spallholz, 1994b), intermediate between selenite and SeMet (most bioavailable). Butler et al. (1991) repleted Se-deficient rats with Se from the tissues of sheep that had consumed high-Se grain and concluded that Se from muscle was more bioavailable than Se from liver or hemoglobin.

The bioavailabilities of Se from meat and wheat were studied in Dutch men who consumed 55, 135, or 215 µg Se/d for nine consecutive weeks (Van Der Torre et al., 1991). Bioavailabilites were similar for both foods, except erythrocyte Se concentration was increased more by meat-Se than by wheat-Se. As discussed previously, young men in North America consumed 33 or 226 µg Se/d in diets composed of different ingredients (Finley and Penland, 1998), with a primary difference being that the high-Se diet included beef with normal concentrations of Se (approximately 0.35 µg Se/g beef), whereas the low-Se diet included low-Se meat (approximately 0.06 µg Se/g beef). The diet high in Se resulted in increased plasma Se concentrations, increased Se incorporation into proteins, and improved mood states. A second North American study that used high-Se meat to help formulate a diet providing 356 µg Se/d also found improved mood scores in adults consuming high Se (Hawkes and Hornbostel, 1996).

Health Benefits of Selenium in Beef

Because the biological actions of Se are dependent on its chemical form, the biological actions of Se in meat also are dependent on the chemical form of Se in meat. The chemical form(s) of Se in beef has(ve) not been directly determined, but numerous studies in nonruminants have demonstrated that the deposition of Se into muscle depends on the chemical form given to the animal. Whanger and Butler (1988) provided rats with 0.2 mg Se/kg diet as selenite or SeMet; the muscle accumulation of Se from SeMet was 2.6-fold greater than Se from selenite. Accumulation also depended on the amount of Se in the diet ; when diets contained 4.0 mg Se/kg, Se from SeMet was accumulated 27-fold greater than Se from selenite. Beilstein and Whanger (1988) demonstrated that muscle contained more Se as SeMet when SeMet was the dietary source (compared to selenite), but most Se was in the form of SeCys when selenite was the dietary form. The effect of chemical form on the deposition of Se in muscle is unrelated to differences in absorption or transport (Vendeland et al., 1992) and is instead related to the differences in metabolism of the various chemical forms.

Extrapolating results found in rats to cattle may be problematic because of the influence of ruminal microorganisms that have the ability to reduce selenite to insoluble Se (Whanger et al. 1968). Also, when ruminal microorganisms encountered selenite as the dietary Se source, they apparently converted the Se to SeCys, but, when they encountered SeMet in the rumen, the primary form of Se in the microbe was also SeMet (van Ryssen et al., 1989). Thus, the form of Se in edible tissues of beef is probably dependent on the dietary form of Se. When cattle consume Se as selenite (the primary form in animal supplements), beef tissue likely contains Se primarily as SeCys. However, when animals consume Se as SeMet (such as when they consume grains or forage high in Se), a higher percentage of the Se in beef tissue may be in the form of SeMet. Based on rat studies, the percentage of Se present as SeMet in meat may be expected to increase as the total Se content of the beef increases.

The primary chemical form of Se in wheat is SeMet (Olson et al., 1970); therefore, studies of the biological actions of Se from wheat may provide an indication of the biological benefits of Se present in beef raised on high-Se forage or grain. High-Se wheat has been used successfully to raise the Se status of several human populations having inadequate Se intakes. Mean blood Se concentrations of South Island, New Zealand residents increased from 57 to 87 ng/mL in the Hamilton region (Watkinson, 1981) and from 61 to 81 ng Se/mL in the Otago region (Thomson and Robinson, 1996) with the importation of Australian wheat. Australian wheat had a mean Se content of 150 ng/g, as compared to New Zealand wheat, which had a mean concentration of 11 ng Se/g. Longnecker et al. (1993) provided high-Se whole-wheat bread to healthy males for 1 yr and demonstrated a dose response of serum and whole blood Se to the amount of Se consumed as wheat. Because residents have very low Se intakes, Finland instituted a national policy of adding Se to all fertilizer in the 1980s (Varo et al., 1988); this resulted in an increase in the Se content of grains. This also increased the average daily Se intake from 20 to 30 to 80 to 90 µg/d. The mean concentration of Se in the blood of 108 healthy adults (Mäkelä et al., 1993) increased from 83 to 126 ng/mL blood, but GSH-Px activity remained unchanged.

Selenium in the beef from animals that have consumed inorganic salts of Se will probably contain Se primarily as SeCys. Because SeCys must be initially metabolized to the selenide form, the metabolism of this form of Se in beef should be more like that of selenite; the biological actions of selenite have already been discussed.

Selenium from beef has not been directly investigated in cancer studies, but SeMet and selenite have. Early studies found SeMet to be less effective than selenite in preventing DNA adducts (Milner et al., 1985) or tumor development in laboratory animals (Stampfer et al., 1987). Our laboratory has studied the efficacy of Se in preventing aberrant colon crypt foci (**ACF**), a preneoplastic marker of colon cancer, in rats (Feng et al., 1999). Selenite and selenate significantly reduced ACF in rats treated with a chemical carcinogen (3,2'-dimethyl-4-aminobiphenyl, DMABP), but Se-Met did not have a significant effect. Selenite and selenate also significantly decreased the occurrence of DNA adducts in DMABP-treated rats, whereas SeMet did not have a significant effect (Davis et al., 1999). Ip and Lisk (1994b) studied the inhibition of dimethylbenz[a]anthracene (**DMBA**)-induced mammary tumors in rats. They demonstrated a numerical, but nonsignificant, reduction in tumor incidence but a significant reduction in number of tumors with 3.0 µg Se/g diet as SeMet, compared to control animals provided 0.1 µg Se/g diet as selenite. The Se from SeMet accumulated in rat tissues to a greater extent than other inorganic or food forms of Se (Ip and Lisk, 1994a). The methionine status of animals affects the biopotency of SeMet against carcinogenesis (Ip, 1988), presumably because low methionine status causes more of the SeMet to substitute for methionine and, thus, lowers its effective concentration for use against carcinogenesis.

When the mass of information on the chemical forms of Se in cancer prevention is considered, SeMet apparently is less effective than selenite in inhibiting carcinogenesis. Thus, the ability of meat to inhibit carcinogenesis may depend on the origin of the meat. Animals produced in the Se-deficient areas of the west and east coasts of the United States have encountered Se primarily as a supplemental salt and consequently the form of Se may primarily be SeCys. However, animals grazing the plains of the Dakota's with high soil Se concentrations may contain Se primarily as SeMet. Regardless of the geographical origin, the actual efficacy of Se from beef against cancer may not be simple to predict. This is demonstrated by the conflicting results of a study that fed Brazil nuts (Ip and Lisk, 1994b). Brazil nuts have high concentrations of Se, which suggests they may be a good potential source of Se. Although the forms of Se in Brazil nuts are not fully characterized, SeMet is assumed to be a primary form because Se from Brazil nuts accumulates in tissues similar to Se from SeMet (Ip and Lisk, 1994b). However, Se from Brazil nuts has been demonstrated to be as effective, if not more effective, than Se from selenite in preventing DMBA-induced mammary tumors (Ip and Lisk, 1994a).

In reality, most Se in meat is probably a combination of SeMet and SeCys; the high-Se yeast used in human cancer trials is also probably a mixture of many forms of Se, although the chemical analysis of this compound has not been completely determined. The results of many of the above studies seem confusing and contradictory, and consequently, the answer to whether Se in meat is effective against carcinogenesis depends on results from controlled studies designed specifically to answer that question.

Potential of Selenium Toxicity

As discussed previously, Se is toxic, and it can probably be assumed that a substantial number of people worldwide view Se toxicity as a more important issue than Se deficiency. In addition to cases of selenosis and alkali disease from the Western range, this view has been perpetuated in recent years by the reports of high mortality among waterfowl in California reservoirs that has been attributed to Se toxicity (Saiki and Lowe, 1987). Human toxicity has been documented, especially in high-Se areas of the People's Republic of China (Levander, 1986). Consequently, any attempt to supplement dietary Se intakes in humans should be considered with the possibility of excess accumulation. The ideal form of supplemental Se would be one that efficiently produces the desired biological activity but does not accumulate in the body and(or) specific tissues. Studies indicate that Se from SeMet accumulates more than other forms of Se; however, Se in meat is probably a mixture of SeMet and SeCys. Consequently, specific studies measuring the accumulation of Se forms are needed. In general, however, it is better to obtain nutrients from the diet than from supplements, because of a lower possibility of overconsumption and a lower chance for adverse dietary interactions. If meat is to be marketed or advertised as a potentially rich source of Se, then it will be important to answer questions regarding Se accumulation in the body.

Implications

Meat, especially red meat and beef, has received much negative publicity in recent years, so it is important to publicize its nutritional advantages and its role in a healthy diet. One of the nutritional advantages of red meat is its trace element content. Red meat, especially beef, is a rich source of Se. However, the biological actions of Se depend on the amount and chemical form of the Se consumed. The amount of Se in beef depends on the geographical area of the country in which the feed was raised and whether the animal received supplemented Se. Also, the chemical form of Se in beef may depend on the form of Se consumed by the animal. Thus, even though meat is potentially an excellent source of supplemental Se, future research must answer specific questions regarding the forms of Se in beef, its biological actions, and its potential for toxicity.

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Notes

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Table 1. Selenium content of common foods purchased in a grocery store in North Dakota

Item	Concentration, μg/g	Standard serving, g	Mass in serving, μg
Bread and grain products [a]			
Bread, whole wheat (Brand 1)	0.33 ± 0.06	28	9.1 ± 1.8
Bread, whole wheat (Brand 2)	0.23 ± 0.003	28	6.4 ± 0.1
Bread, whole wheat (Brand 3)	0.29 ± 0.05	28	8.0 ± 1.3
Flour, white, all-purpose (Brand 1)	0.30 ± 0.01	16	4.7 ± 0.2
Flour, white, all-purpose (Brand 2)	0.16 ± 0.009	16	2.5 ± 0.2
Flour, white, all-purpose (Brand 3)	0.40 ± 0.01	16	6.4 ± 0.2
Tortilla, corn (Brand 1)	0.03 ± 0.002	25	0.9 ± 0.0
Tortilla, corn (Brand 2)	0.35 ± 0.03	25	8.7 ± 0.8
Corn masa mix, instant	0.05 ± 0.006	16	0.8 ± 0.1
Corn masa mix	0.05 ± 0.008	16	8.0 ± 0.1
Corn meal, white, degermed	0.07 ± 0.002	16	1.2 ± 0.0
Corn meal, yellow, degermed	0.08 ± 0.006	16	1.2 ± 0.1
Rice, white, instant, cooked (Brand 1)	0.09 ± 0.02	137	11.8 ± 2.9
Rice, white, instant, cooked (Brand 2)	0.34 ± 0.07	137	45.9 ± 9.3
Rice, white, long-grain, cooked	0.09 ± 0.007	137	12.9 ± 1.0
Meats			
Beef, ground, raw, eastern North Dakota	0.37 ± 0.10	85	31.5 ± 8.11
Beef, ground, raw, southwestern Missouri	0.06 ± 0.01	85	5.1 ±1.0
Beef, round, raw, central Virginia	0.08 ± 0.004	85	7.1 ± 0.3
Beef, round, raw, local supermarket	0.33 ± 0.02	85	28.3 ± 1.5
Venison, uncooked, western North Dakota	0.20 ± 0.004	85	16.9 ± 0.3
Venison, uncooked, central South Dakota	0.45 ± 0.01	85	38.1 ± 1.2
Ham, 95% fat free, deli	0.35 ± 0.02	85	30.1 ± 1.4
Lamb, raw, eastern North Dakota	0.30 ± 0.01	85	25.3 ± 1.0
Liver, beef, raw, local supermarket	1.01 ± 0.03	85	86.3 ± 2.9
Fruits			
Apple, red delicious, raw		138	—
Blueberries, frozen, unsweetened	*	128	—
Pineapple, canned, juice pack	*	98	—
Strawberries, frozen, unsweetened	*	128	—
Vegetables			
Asparagus, spears, canned	0.03 ± 0.01	124	3.2 ± 1.4
Broccoli, chopped, frozen, uncooked	*	103	—
Carrots, canned	*	76	—
Green beans, canned, low sodium	*	68	—
Lettuce, iceberg	*	55	—
Mushrooms, canned	0.13 ± 0.009	78	10.4 ± 0.7

[a] Each value is the mean of three separate analyses. Samples randomly taken from one unit of purchase (i.e. bag or box for dry goods, 454 to 908 g of meat, 454 g of fruits or vegetables). Values expressed on an as-fed basis.

*Value below detection limit (2.0 ng/g).

Table 2. Selenium concentrations in tissues of cattle from different areas of North Dakota

	Region of the United States				
Item	Central	Northwest	Southeast	South-central	Southwest
n	8	21	14	20	29
Muscle[a]	0.40 ± 011^{yz}	0.67 ± 0.22^{x}	0.28 ± 0.07^{z}	0.48 ± 0.10^{y}	0.43 ± 0.08^{y}
Liver	0.61 ± 0.11^{xyz}	0.72 ± 0.21^{xy}	0.47 ± 0.07^{z}	0.78 ± 0.14^{x}	0.60 ± 0.17^{yz}

[a] Values are (µg Se/g fresh meat) means ± SD; means with different superscripts are significantly different ($P < 0.05$).

Table 3. Selenium content of plant and animal material from an area in South Dakota with a previous history of selenosis

Item	Se concentration, µg Se/g
Wheat[a]	0.9 ± 0.1
Alfalfa hay	1.59 ± 0.02
Ground beef, raw	0.64 ± 0.5
Princess plume	28.43 ± 1.70
Astragalus plant	16.58 ± 1.22
Astragalus flower	31.78 ± 0.50
Astragalus, non-seleniferous area	6.30 ± 0.62

aValues are expressed on a dry matter basis except wheat and beef which are expressed as fed.

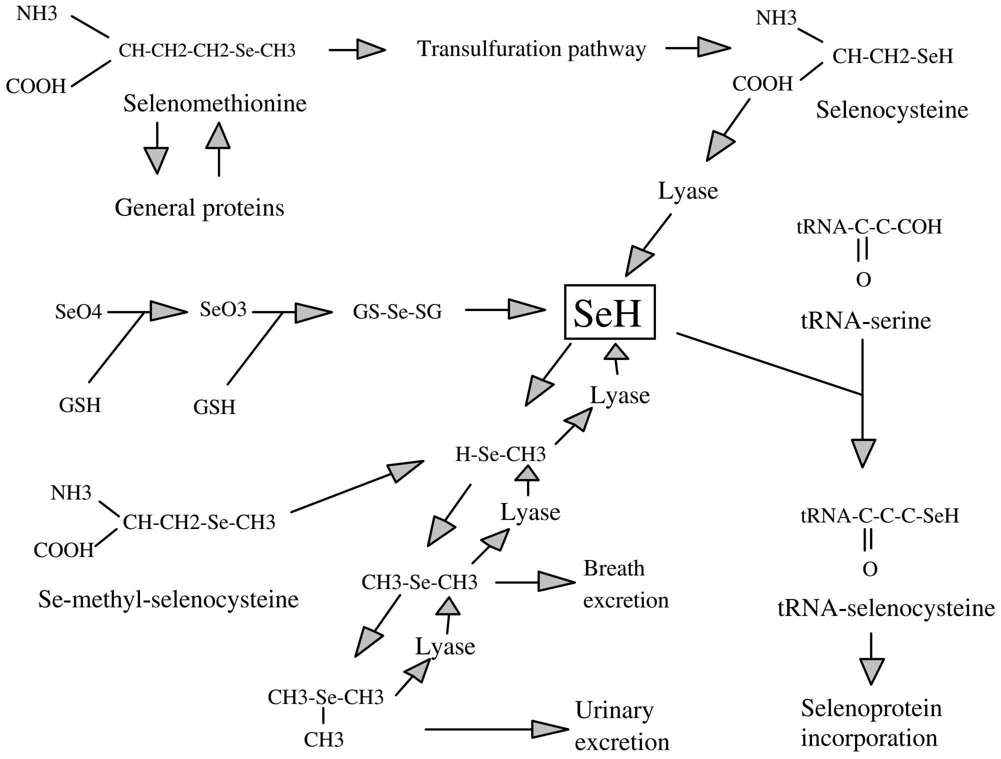

Figure 1. Generalized pathways of selenium metabolism.