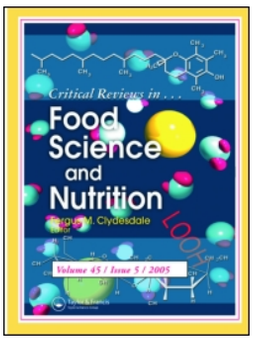

Critical Reviews in Food Science and Nutrition

ISSN: 1040-8398 (Print) 1549-7852 (Online) Journal homepage: http://www.tandfonline.com/loi/bfsn20

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To cite this article: Jorge Moreda-Piñeiro, Antonio Moreda-Piñeiro & Pilar Bermejo-Barrera (2017) In vivo and in vitro testing for selenium and selenium compounds bioavailability assessment in foodstuff, Critical Reviews in Food Science and Nutrition, 57:4, 805-833, DOI: 10.1080/10408398.2014.934437

To link to this article: http://dx.doi.org/10.1080/10408398.2014.934437

Accepted author version posted online: 21 Apr 2015.
Published online: 21 Apr 2015.

CRITICAL REVIEWS IN FOOD SCIENCE AND NUTRITION
2017, VOL. 57, NO. 4, 805–833
http://dx.doi.org/10.1080/10408398.2014.934437

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In vivo and in vitro testing for selenium and selenium compounds bioavailability assessment in foodstuff

Jorge Moreda-Piñeiro[a], Antonio Moreda-Piñeiro[b], and Pilar Bermejo-Barrera[b]

[a]Grupo Química Analítica Aplicada (QANAP), University Institute of Research in Environmental Studies (IUMA), Department of Analytical Chemistry, Faculty of Sciences, University of A Coruña, Campus de A Coruña, A Coruña, Spain; [b]Department of Analytical Chemistry, Nutrition and Bromatology, Faculty of Chemistry, University of Santiago de Compostela, Santiago de Compostela, Spain

ABSTRACT

The assessment of selenium and selenium species bioavailability in foodstuff is of special concern on the context of human nutrition. In vivo (human and animal), and in vitro tests are important approaches for estimating the bioavailability of toxic and essential compounds to humans. An overview on in vivo and in vitro bioavailability assays for releasing selenium and selenium species in foodstuffs is summarized. Se and Se species content in a foodstuff critically influence Se bioavailability and bioactivity to humans and animals. Se bioavailability is affected by foodstuff-matrix major composition and minor components. Foodstuffs processing and/or treatments could enhancement or decrease Se bioavailability. Experimental conditions such as the selection of healthy status of examined people (in in vivo humans approaches), the selection of animal model (in vivo animals approaches), or the selection of GI conditions (in in vitro tests) could determines the results. Thus, international standardized protocol for in vivo and in vitro approaches assessment is mandatory.

KEYWORDS

Foodstuff; bioavailability; bioaccessibility; bioactivity; in vivo and in vitro tests; nutrient content; food processing

Introduction

Human intake of essential/toxic substances occurs mainly via food ingestion. Because of the knowledge of the total content of essential/toxic substances in foodstuffs does not provide enough information for assessing the beneficial/toxic effects after food consumption, the assessment of the amount of the essential/toxic substances that reaches the systemic circulation (after ingestion), and which is available to perform its biological activity or toxic effect (bioaccessibility, bioavailability, or bioactivity), is therefore required. Bioaccessibility indicates the maximum fraction of a nutrient or substance in food that is theoretically soluble from its matrix in the gastro intestinal (GI) tract (bioaccessible fraction), and that is therefore available for subsequent processes of absorption through the intestinal mucosa (i.e., enters the blood stream) (Ruby et al., 1999; Oomen et al., 2002; Stahl et al., 2002). The non-accessible fraction cannot be absorbed through the intestinal membrane and is directly excreted. Bioavailability refers to the fraction of a substance released from food that is absorbed through the intestinal barrier (bioavailable fraction), passes into the bloodstream or an organ, and is available to promote its action in the exposed organism (Ruby et al., 1999). Thus, the term bioavailability includes the term bioaccessibility. The bioavailable fraction is also defined as the quantity of a substance that is absorbed and reaches the systemic circulation, that is distributed to organs and tissues, that is transformed into a biochemically active form (glutathione peroxidase, GPx; thioredoxin reductase, TrxR; and selenoprotein P, SeP; in the case of selenium (Se)), and that is effectively used by the organism (bioactive fraction) (Schümann et al., 1997; Thiry et al., 2012). Bioavailability is defined as a sum of bioaccessibility and bioactivity. The type of food, the composition of the food matrix, and the synergies and antagonisms that may be established among the several components can alter the bioaccessibility, bioavailability, and bioactivity ratios of one substance. The GI conditions, special physiological conditions of the consumer (such as age or health status), and compound species distribution (chemical forms) in the food product are also important factors affecting bioavailability (Ziegler et al., 1978).

The assessment of substances bioavailability in foodstuff can be performed by using several approaches: in vivo (human and animal) and in vitro studies. Both approaches present strengths and drawbacks. Although in vivo human studies are the ultimate test to determine bioavailability of nutrients/substances, compared to in vivo animal or in vitro tests, some experimental approaches are unethically and technically unaffordable (Van Dyck et al., 1996). People are not as standardize as animals or cells and they cannot release as much information as animals which are usually sacrificed at the end of the experiment for a further analysis of biopsied tissues and organs (Thiry et al., 2012). Although in vivo *animals* tests reflect real situation, allowing enough sampling to perform pharmacokinetic studies and the selection of individuals for specific target population of intended use, several drawbacks are associated to in vivo animal approaches. Testing with animals is expensive, difficult to perform, and ethically controversial, it provides limited data in

CONTACT Jorge Moreda-Piñeiro jorge.moreda@udc.es Grupo Química Analítica Aplicada (QANAP), University Institute of Research in Environmental Studies (IUMA), Department of Analytical Chemistry, Faculty of Sciences, University of A Coruña, Campus de A Coruña, s/n. 15071—A, Coruña, Spain.

each experiment (low throughput) and doubts of overall contribution of factors involved on bioaccessibility (Hansen et al., 1996; Fernández-García et al., 2009). The lack of certified reference standards to compare data among studies/laboratories also limits the quality of in vivo test. In addition, the relevance for human situation derived from in vivo animal studies not always could be extrapolated (Fernández-García et al., 2009). As an alternative, bioavailability studies have also been estimated through various in vitro methods. In vitro methods provide an effective approximation to in vivo scenarios and offer the advantages of high-throughput screening tools, simplicity (automation, and miniaturization), rapidity, ease of control, low cost, high precision and accuracy (validation and standardization with reference materials), and good reproducibility. They provide information about efficiency of each digestion/absorption step and transport mechanisms; and the use of laboratory animals is avoided, which connects with the increasing interest in reducing the use of animals for testing. On the contrary, the dynamic environment of intestine is not fully reproduced with biochemical and cell culture models, and, in addition, the effect of intestinal micro-flora and hepatic metabolism is not always considered in in vitro assays (Hansen et al., 1996; Fernández-García et al., 2009).

Due to Se has been largely recognized as a nutritionally essential element for human life, due to the low Se intake has become a problem for many people worldwide (Navarro-Alarcon and Cabrera-Vique, 2008), and due to Se enters the body almost exclusively through food, the knowledge of Se bioavailability in foodstuffs in humans is an interesting research area. In this context, Se bioavailability studies have been focused on foodstuffs containing high Se concentrations, such as Brazilian nuts, yeast and fish, and food biofortificated with several Se species (selenite, Se(IV); selenate, Se(VI); and selenomethionine, SeMet). Foodstuff (i.e., milk, meat, and eggs) are enriched with Se through supplementation of livestock or through Se fertilization of crops (i.e., cereal and vegetables).

Useful information about Se bioavailability in foodstuffs can be found in several reviews focused on in vivo human (Finley, 2006; Fairweather-Tait et al., 2010; Thiry et al., 2012), in vivo animal (Finley, 2006; Thiry et al., 2012), and in vitro (Intawongse and Dean, 2006; Moreda-Piñeiro et al., 2011) approaches. In this article, our aim is to summarize and discuss the current status of Se bioavailability in food samples, focusing on in vivo (human and animals) and in vitro approaches. We also consider the effect of processing/cooking food on Se species bioavailability. The effect of major and minor components of food matrix on Se availability will be also considered.

Selenium bioavailability assessment

As previously mentioned, bioavailability could be assessed by using in vivo (human and animal), and in vitro approaches.

In vivo tests

In in vivo (human and animal) tests, foodstuff or Se-fortified food (isotopically labelled or non-isotopically labelled) is directly dosed (after controlled oral intake) to subjects, or it can be dosed after subjects were Se-depleted with low Se content diet. During the period of repletion, the extent and the rapidity of the Se status recovery are assessed. The increses on the percentage of Se concentration and/or GPx and TrxR activities in several tissues are then measured. In addition, the determination of the Se concentration in several tissues, and further balance studies are then performed, which allows the determination of the absorbed amount of Se or its metabolites (isotopically labelled or non-isotopically labelled).

Balance studies are performed by determining the absorbed amounts of Se (apparent absorption and retention) after measuring the difference between the fed amounts of Se in food and the excreted amounts of Se/metabolite in faeces and urine. Apparent absorption and retention of nutrients/target compounds (expressed as a percentage of the total dose) can be calculated by using the following Equations (1 and 2) (Fox et al., 2004):

$$\text{Apparent}_{\text{Absorption}} = \frac{[Se]_{\text{intake}} - [Se]_{\text{infaeces}}}{[Se]_{\text{intake}}} \times 100 \quad (1)$$

$$\text{Retention} = \frac{[Se]_{\text{intake}} - [Se]_{\text{infaeces}} - [Se]_{\text{inurine}}}{[Se]_{\text{intake}} - [Se]_{\text{infaeces}}} \times 100 \quad (2)$$

where $[Se]_{\text{intake}}$ is the concentration of Se intake (isotopically labelled or non-isotopically labelled) and $[Se]_{\text{infaeces}}$ and $[Se]_{\text{inurine}}$ are the concentrations of Se in faeces and urine, respectively.

Other methods for assessing in vivo animal tests consist of performing animal feeding in parallel with the selected foodstuff and with a reference selenocompound (selenite and SeMet mainly) during the repletion period. Results obtained when feeding with the tested food are then compared to those obtained when feeding with the single selenenocompound. The comparison is done by using Equation (3) (slope-ratio assay) after achieving the slopes of the regression lines generated by dose–response relationship of a tested food item and dose–response relationship of the reference Se chemical pure.

$$\text{Ratio} = \frac{S_{\text{food}}}{S_{\text{selenocompound}}} \times 100 \quad (3)$$

where S_{food} and $S_{\text{selenocompound}}$ are the slopes of regression lines dose–response relationship for foodstuff and Se chemical pure form (selenocompound), respectively. The resulting ratio can then be used to quantitatively compare the bioavailability of Se in different foods.

Intestinal absorption and apparent digestability estimation can also be used to Se bioavailability assessment by in vivo animal tests. Intestinal absorption of Se could be determined after administration of Se isotopes (i.e., ^{75}Se) by: (1) plotting cumulative faecal excretion of ^{75}Se versus time (Thompson and Stewart, 1974; Griffiths et al., 1976; Thompson et al., 1978; Kasper et al., 1984), and (2) by the in situ ligated intestinal loop procedure (Mutanen and Mykkänen, 1984). The first approach consists of building the straight line joining the last three points on the curve and extrapolating to the zero-time intercept, point which is taken to represent the proportion of tracer not absorbed. The latter approach consists of dosing of ^{75}Se(IV) by injection into the ligated duodenal loop of the anesthetized animal, and allowing the absorption to proceed for several

minutes. The loop is then recovered after animal killing, and the luminal contents are counted for the residual activity. The intestinal absorption is achieved by using Equation (4):

$$\text{Intestinal}_{\text{Absorption}} = \left([^{75}Se_{\text{dose}}] - [^{75}Se_{\text{luminal}}]\right) \times 100 \quad (4)$$

where $[^{75}Se_{\text{dose}}]$ and $[^{75}Se_{\text{luminal}}]$ are the Se concentrations in the administered dose and in the luminal content, respectively.

Apparent digestibility was determined using Equation (5) (Paripatanamont and Lowell, 1997):

$$\text{Apparent}_{\text{Digestability}} = \left(1 - \frac{[X_{\text{diet}}]x[Se_{\text{faeces}}]}{[X_{\text{faeces}}]x[Se_{\text{diet}}]}\right) \times 100 \quad (5)$$

where $[X_{\text{diet}}]$ and $[X_{\text{faeces}}]$, are the concentrations in diet and faeces of a reference substance added to diet; and $[Se_{\text{diet}}]$ and $[Se_{\text{faeces}}]$ are the Se concentrations in diet and faeces, respectively. A reference substance was added to diet as an indicator for apparent digestibility measurement.

In vitro tests

In in vitro methods the physiologic conditions (temperature, shaking or agitation, pH, and enzyme/chemical composition of saliva, and gastric and intestine juices), and the sequence of events that occur in the human GI tract during the human body digestion, are simulated (Miller et al., 1981; Ruby et al., 1999; Fernández-García et al., 2009). Simulate GI digestion occurs in two steps: (1) gastric digestion; i.e., with amylase, pancreatin, and bile salts at neutral pH. After this step a digesta or intestinal preparation is then obtained; and (2) digesta assimilation by the intestinal mucosa. Different experimental options are available for this purpose, i.e. dialysis membranes and cell culture-based models (Caco-2 cell models). Caco-2 cells (derived from human colon carcinoma cells) have similar structural and functional characteristics that colon enterocytes.

The measurements of the maximum soluble concentrations of Se in the simulated GI solution after filtration or centrifugation (bioaccessible fraction), expressed as a percentage, is calculated using Equation (6):

$$\text{Bacc} = \frac{[Se]_{\text{liquid extract}}}{[Se]_{\text{food}}} \times 100 \quad (6)$$

where *Bacc* is the percentage of bioaccessibility, $[Se]_{\text{liquid extract}}$ and $[Se]_{\text{food}}$ are the Se concentrations after in vitro digestion and in food sample before in vitro GI digestion, respectively.

The measurement of the dialyzable fraction of Se which is capable of dialyzing (during intestinal digestion) through a semi-permeable membrane with a specified pore size (dialysate or bioavailable fraction), expressed as a percentage, is calculated using Equations (7) and (8):

$$\text{Bav} = \frac{[Se]_{\text{dialyzate}}}{[Se]_{\text{food}}} \times 100 \quad (7)$$

$$\text{Dial} = \frac{D_{\text{dialyzate}}}{W_{\text{food}}x[Se]_{\text{food}}} \times 100 \quad (8)$$

where *Bav* and *Dial* are the percentages of bioavailability and dialysability, respectively; $[Se]_{\text{dialyzate}}$ is the Se concentration after in vitro digestion plus dialysis process, $D_{\text{dialyzate}}$ is total amount of dialysed compound (expressed in mass), and W_{food} is the sample weight of sample used for intestinal digestion plus dialysis process.

The measurement of the fraction of the Se capable of being retained and/or transported through a solid or a micro-porous support (bioavailable fraction) in which human cells grown are incorporated (intestinal epithelial model) (Karlsson and Artursson, 1991) can be also used to achieve the Se bioavailability by using in vitro tests. In these models, a previous in vitro GI digestion is required. Caco-2 cell model is suitable to study cellular uptake, retention, transport, and metabolism processes of target compounds (Zeng et al., 2008), and offers a more reliable approximation to the in vivo situation in estimating bioavailability at the intestinal level. In this approach a bicameral system is used for transport and retention studies; in which a monolayer of Caco-2 cells is grown on a filter separating two stacked well plates. The upper well (apical side) is filled with the donor solution (containing the digesta), and the bottom (basal side) is filled with the acceptor solution (minimum essential medium Eagle, MEM). For Se bioavailability studies, Dulbecco's modified Eagle's medium (DMEM) is generally used due to it is a Se-free medium (Zeng et al., 2008). Several DMEM formulations (i.e., several glucose or glutamine concentrations) could be used. Uptake (retention and transport percentages) by Caco-2 cells is calculated by Equations (9) and (10):

$$\text{Retention} = \frac{[Se]_{\text{cells}}}{[Se]_{\text{bioaccessible}}} \times 100 \quad (9)$$

$$\text{Transport} = \frac{[Se]_{\text{basal}}}{[Se]_{\text{bioaccessible}}} \times 100 \quad (10)$$

where $[Se]_{\text{cells}}$, $[Se]_{\text{bioaccessible}}$, and $[Se]_{\text{basal}}$ are target compound concentrations in monolayer Caco-2 cells, in the bioaccessible fraction after simulated GI digestion and in the basal side, respectively. The bioavailable fraction is considered to be the sum of the fraction of soluble Se that was transported through a monolayer of Caco-2 cells in a bicameral culture system and the fraction that was retained in cells during the process of absorption (Equation (11)).

$$\text{Bav} = \frac{[Se]_{\text{basal}} + [Se]_{\text{cells}}}{[Se]_{\text{food}}} \times 100 \quad (11)$$

Finally, Se bioavailability can be assessed by using several dynamic models such as the simulator of the human intestinal microbial ecosystem (SHIME) (Molly et al., 1993), which mimics in vivo physical processes. The original SHIME model consists of five reactors that sequentially simulate the stomach (acid conditions and pepsin digestion), the small intestine (digestive processes), and the three regions of the large intestine, i.e., the ascending, transverse, and descending colon (microbial processes). Dynamic models are flexible [they can be adapted to mimic specific conditions related to age (infant, adult, elderly), type of meal (liquid, semi-solid, and solid

meals), and health or disease status], and they are accurate and precise assays. These models are usually computer controlled by software that includes monitoring of all parts in real time.

Selenium bioavailability in foodstuff

As can be seen in Tables 1–5, several in vivo (human and animals) and in vitro approaches are extensively used to assess the Se bioavailability in foodstuffs (Tables 1–4) and Se chemically pure forms (Table 5).

Studies are focussed on several Se rich and Se-fortified foodstuff: (1) raw/processed cereals and Se-fortified cereals, such as wheat, bran, maize, corn gluten, oat, and rice; (2) raw/cooked/processed vegetables and Se-fortified vegetables, such as soybean, tofu, broccoli, mushroom, ramps, garlic, onion, chives, cabbage, radish, leek, and peas; (3) raw/cooked/processed fish and shellfish; (4) raw/cooked/processed algae and edible seaweed; (5) yeast, selenized-yeast, and selenized yeast-based nutritional supplements; (6) raw/processed meat (beef, pork, chicken, sheep, and lamb); and, (7) other foodstuff, such as Brazil nuts, milk (human, cow, goat and sheep), and eggs.

Selenium bioavailability in cereals

Several in vivo human, in vivo animal, and in vitro studies have been carried out to assess the bioavailability of Se in naturally Se-rich, selenized cereals and processed cereal products (Table 1): wheat grain and flour (Levander et al., 1981; Douglass et al., 1981; Alexander et al., 1983; Levander et al., 1983; Thompson et al., 1985; Mutanen, 1986; House and Welch, 1989; Van der Torre et al., 1991; Meltzer et al., 1993; Djujić et al., 2000; Reeves et al., 2005; Reeves et al., 2007; Kirby et al., 2008; Govasmark et al., 2010), rice (Hawkes et al., 2003; Fang et al., 2010; Jaiswal et al., 2012; Wang et al., 2013), maize (Jaiswal et al., 2012), corn gluten (Gabrielsen and Opstvedt, 1980), wheat biscuits (Kirby et al., 2008), and wheat bread and bran (Alexander et al., 1983; Govasmark et al., 2010).

In vivo human tests show that Se levels in plasma or whole blood increased significantly after supplementation with Se-fortified wheat (Thompson et al., 1985; Mutanen, 1986; Van der Torre et al., 1991; Djujić et al., 2000; Kirby et al., 2008). The increase depends on the presence of different Se species in foodstuff (Kirby et al., 2008). Plasma Se concentrations with Se-fortified wheat were found to increase from 122 μg L^{-1} to 194 μg L^{-1} after 6 months of supplementation; in contrast, supplementation with process-fortified Se biscuits showed a little increase in the total Se plasma concentrations mean (122 μg L^{-1} at 0 months to 140 μg L^{-1} at 4 months). The difference in total Se plasma concentrations may be due to the presence of different Se species in the fortified wheat (SeMet, mainly) and process-fortified biscuits (SeOMet, mainly) (Kirby et al., 2008). In vivo animal tests showed that Se bioavailability ratios in wheat and Se-fortified wheat were similar to those obtained when using SeMet. This fact confirms that SeMet represents almost the totality of the Se species in wheat grain. In vivo tests also showed that similar Se bioavailability was found for whole wheat flour, whole wheat bread, or bran (Alexander et al., 1983). The restoration of Se dependent enzymes activity (liver GPx and TrxR) and tissue Se concentrations in Se-deficient rats, induced by Se-fortified wheat and wheat bran, seems to be satisfactory (60–85% as high as selenite or SeMet) (Douglass et al., 1981; House and Welch, 1989; Reeves et al., 2005; Reeves et al., 2007). On the contrary, Gabrielsen and Opstvedt (Gabrielsen and Opstvedt, 1980) reported very low Se bioavailability in corn gluten (26%) in Se-depleted chicks. Se yields (within the 50–75% range in wheat and bran) were assessed when using a simulated gastric digestion (Govasmark et al., 2010). The bioaccessible ratio, however, did not increase after the simulated duodenal digestion. Se bioaccessible levels within the 35–65% range were also achieved for rice (Fang et al., 2010; Jaiswal et al., 2012; Wang et al., 2013) and maize flour (Jaiswal et al., 2012). Low molecular weight Se-containing proteins, SeMet, and SeCys were the major Se-containing moieties found in the GI extract (Fang et al., 2010; Wang et al., 2013).

Finally, Se in naturally produced Se-fortified oats (harvested in selenized soils) was found to be highly bioavailable (Yan and Johnson, 2011). The Se bioavailability was 92% higher in oats than in SeMet solutions.

Selenium bioavailability in vegetables

Several vegetables and selenized vegetables such as soybean and soybean products (Gabrielsen and Opstvedt, 1980; Marks and Mason, 1993; Yan et al., 2009; Jang et al., 2010; Yan et al., 2010), broccoli (Finley, 1998; Finley, 1999; Finley et al., 2004; Zeng et al., 2008), mushroom (Mutanen, 1986; Chansler et al., 1986; da Silva et al., 2010; Turlo et al., 2010, Bhatia et al., 2013), ramps (Whanger et al., 2000), radish (Pedrero et al., 2006), green onion (Kàpolna and Fodor, 2007), chives (Kàpolna and Fodor, 2007), cabbage (Seo et al., 2008), garlic (Ip and Lisk, 1993; Dumont et al., 2006a; Seo et al., 2008), leek (Lavu et al., 2012), and peas (Yan and Johnson, 2011) has been used as Se sources in several in vivo and in vitro approaches (Table 1).

Bioavailability of Se in soybean (grown with selenite or selenate) was found to be very high (Marks and Mason, 1993; Yan et al., 2009; Jang et al., 2010; Yan et al., 2010) after in vivo rat and pig tests. High bioavailability was also obtained for protein isolate from soybean (101%) and soybean products such as tofu (94%) (Yan et al., 2010). On the contrary, Gabrielsen and Opstvedt (Gabrielsen and Opstvedt, 1980) reported very low Se bioavailability in soybean (18%) by using chicks as animal tests.

Se in Se-fortified broccoli was much less effective than selenite, selenate and SeMet in restoring kidney and plasma Se concentrations, and GPx activity in rats (Finley, 1998; Finley et al., 2004). The use of different metabolic pathway by Se in Se-fortified broccoli could explain this fact (Finley et al., 2004). On the contrary, similar Se bioavailability was found in broccoli and selenate by using in vivo human approaches (Finley, 1999). Similar Se biavailability ratios in Se-fortified broccoli extracts and in seleno-methylselenocysteine (SeMSeCys) and SeMet aqueous standards were found by using Caco-2 cell models after simulated GI digestion (Zeng et al., 2008).

Some in vivo human (Mutanen, 1986) and in vivo rat (Chansler et al., 1986, da Silva et al., 2010) studies regarding mushrooms have proven a very low capacity in stimulating Se levels and GPx activity, which suggested that mushrooms may contain slightly available Se species (non-protein Se

Table 1. Application of in vivo and in vitro tests for Se bioavailability in cereals and vegetables.

Measured parameter	Sample matrix	Model of study	Duration of supplementation (Dose of supplementation)	Se concentration increase (%) after supplementation	GPx increase (%) after supplementation	Relative bioavailability (%)	Ref.
Se concentration	Wheat and wheat plus tuna fish	Human (Whole body)	25 days (200 μg Se d^{-1})			62[a]	Levander et al., 1981
Se concentration	Wheat	Rat (Young RBC and liver)	4 weeks 0.2 μg Se g^{-1} food)			111[a]	Douglass et al., 1981
Se concentration and GPx activity	Selenized-wheat	Human (Plasma and platelet)	11 weeks (200 μgSe d^{-1})	139 for plasma	162 for platelet		Levander et al., 1983
Se concentration	Wheat flour, whole wheat bread and bran	Rat (Whole blood, liver, kidney and muscle)	4 weeks (0.05–0.15 μg Se g^{-1} food)			80, 100, 100 200[b] for whole blood, liver, kidney and muscle, resp.	Alexander et al., 1983
GPx activity	Wheat flour, whole wheat bread and bran	Rat (Whole blood, liver and kidney)	4 weeks (0.05–0.15 μg Se g^{-1} food)			108, 70, 133[b] for whole blood, liver and kidney, resp.	Alexander et al., 1983
Se concentration and GPx activity	Selenized-wheat	Human (Plasma and platelet)	13 weeks (200 μg Se(IV) d^{-1})	132–214, 116–167 for plasma and platelet, resp.	25–119, 56–172 for plasma and platelet, resp.		Thompson et al., 1985
Se concentration	Wheat	Human (Plasma)	4 weeks(150 μg Se d^{-1})	55			Mutanen, 1986
Se concentration	Wheat	Rat (Whole body)	1 day (3.5 μg Se g^{-1} food (as ^{75}Se(VI)))			35–52[a]	House and Welch, 1989
Se concentration and GPx activity	Wheat	Human (Whole body and plasma)	9 weeks (55, 135, and 215 pg Se d^{-1})	69 for plasma		76[a]	Van der Torre et al., 1991
Se concentration and GPx activity	Wheat	Human (Plasma and platelet)	6 weeks (135 $\pm$ 25 μg Se d^{-1})	17 for plasma	NI for plasma and platelet, resp.		Meltzer et al., 1993
Se concentration and GPx activity	Wheat	Human (Plasma)	6 weeks (26–44 μg Se d^{-1})–[c]	53	83.7		Djujić et al., 2000
Se concentration	Selenized-wheat and bran	Rat (Plasma)	(10–150 μg Se kg^{-1} food)			85 and 60[b] for wheat and bran, resp.	Reeves et al., 2007
Se concentration	Wheat	Human (Plasma)	6 months (258 μg Se d^{-1})	59			Kirby et al., 2008
SeMet concentration	Wheat, bran	**In vitro**				75.4 and 51.4[d] for wheat and bran, resp.	Govasmark et al., 2010
Se concentration	Buckwheat bran	Rat (Plasma, liver, kidney and muscle)	37 days (–[c])			69.4, 72.0, 84.2 and 93[b] for plasma, liver, kidney and muscle, resp.	Reeves et al., 2005
GPx activity	Buckwheat bran	Rat (Liver and whole blood)	37 days (–[c])			81.2 and 76.5[b] for liver and whole blood, resp.	Reeves et al., 2005
Se concentration	Wheat biscuit	Human (Plasma)	6 months (315 μg Se d^{-1})	14.7			Kirby et al., 2008
Se concentration and GPx activity	Rice plus beef	Human (Whole body and plasma)	99 days (14 μg Se d^{-1} (low) 297 μg Se d^{-1} (high))	85 (low) and 91(high) for plasma	NI (low) and 14 (high) for plasma	19–45[a] (low) 75–88[a] (high)	Hawkes et al., 2003
LMW Se-containing protein, SeMet concentration	Rice	In vitro				35.8 and 52.3[d] for LMW Se-containing protein and SeMet, resp.	Fang et al., 2010
Se concentration	Rice and maize flour	In vitro				65 and 51[d] for rice and maize flour, resp.	Jaiswal et al., 2012
SeMet and SeCys concentration	Cooked rice	In vitro				–[c]	Wang et al., 2013

(*Continued on next page*)

Table 1. (Continued).

Measured parameter	Sample matrix	Model of study	Duration of supplementation (Dose of supplementation)	Se concentration increase (%) after supplementation	GPx increase (%) after supplementation	Relative bioavailability (%)	Ref.
GPx activity	Corn gluten	Chick (Serum)	9 days (–[c])			26[b]	Gabrielsen and Opstvedt, 1980
Se concentration	Selenized-oat	Rat (Plasma, liver, muscle, and kidney)	50 days (15–33.5 μg Se kg^{-1})			94.4, 108.4, 104.6 and 89.5[b] for plasma, liver, muscle and kidney, resp.	Yan and Johnson, 2011
GPx activity	Selenized-oat	Rat (Whole blood and liver)	50 days (15–33.5 μg Se kg^{-1})			86.7 and 84.6[b] for whole blood and liver, resp.	Yan and Johnson, 2011
TrxR Activity	Selenized-oat	Rat (Liver)	50 days (15–33,5 μg Se kg^{-1})			83.5[b]	Yan and Johnson, 2011
GPx activity	Soybean	Chick (Serum)	9 days (–[c])			18[b]	Gabrielsen and Opstvedt, 1980
GPx activity	Selenized-soybean	Rat (Liver and plasma)	7 week (100 ng Se g^{-1} food)		~400 and ~390 for liver and plasma, resp.		Marks and Mason, 1993
GPx activity	Selenized-soybean	Rat (Liver and plasma)	7 week (50 ng Se g^{-1} food)		~31 and ~260 for liver and plasma, resp.		Marks and Mason, 1993
Se concentration	Soybean	Rat (Plasma, liver, muscle and kidney)	50 days (20–40 μg Se g^{-1} food)			85.6, 113, 120.6 and 99.5[b] for plasma, liver, muscle and kidney	Yan et al., 2009
GPx activity	Soybean	Rat (Whole blood and liver)	50 days (20–40 μg Se g^{-1} food)			98.3 and 85.7[b] for whole blood and liver, resp.	Yan et al., 2009
TrxR activity	Soybean and tofu	Rat (Liver)	50 days (20–40 μg Se g^{-1} food)			64.2 and 70.2[b] for soybean and tofu, resp.	Yan et al., 2009
Se concentration	Soybean protein	Pig (Serum, loin, liver, kidney, pancreas and spleen)	6 weeks (11000 μg Se g^{-1} food)	23, 6, 60, NI, NI and NI for serum, loin, liver, kidney, pancreas and spleen, resp.			Jang et al., 2010
Se concentration	Soybean protein	Rat (Plasma, liver, muscle and kidney)	50 days (14, 24, and 30 μg Se g^{-1} food)			98.6, 117,2, 101.6 and 99.5[b] for plasma, liver, muscle and kidney resp.	Yan et al., 2010
GPx activity	Soybean protein	Rat (Whole blood and liver)	50 days (14, 24, and 30 μg Se g^{-1} food)			119.4 and 100.8[b] for whole blood and liver, resp.	Yan et al., 2010
TrxR activity	Soybean protein and tofu	Rat (Liver)	50 days (20–40 μg Se g^{-1} food)			64.2 and 70.2[b] for soybean and tofu, resp.	Yan et al., 2010
Se concentration	Tofu	Rat (Plasma, liver, muscle and kidney)	50 days (20–40 μg Se g^{-1} food)			100.6, 110,6, 119,9 and 99.6.5[b] for plasma, liver, muscle and kidney	Yan et al., 2009
GPx activity	Tofu	Rat (Whole blood and liver)	50 days (20–40 μg Se g^{-1} food)			96.5 and 103.8[b] for whole blood and liver, resp.	Yan et al., 2009

Se concentration	Tofu	Rat (Plasma, liver, muscle and kidney)	50 days (13, 23, and 31 μg Se g^{-1} food)			99.8, 115,1, 78.4 and 114.0[b] for plasma, liver, muscle and kidney resp.	Yan et al., 2010
GPx activity	Tofu	Rat (Whole blood and liver)	50 days (13, 23, and 31 μg Se g^{-1} food)			74.6 and 74.2[b] for whole blood and liver, resp.	Yan et al., 2010
Se concentration	Broccoli	Rat (Whole body)	9 weeks (0.1 μg Se g^{-1} food)			85[a]	Finley, 1998
Se concentration	Broccoli	Human (Whole body)	1 day (210 μg Se d^{-1} (as ^{82}Se (VI) and ^{74}Se(VI) in broccoli)			70.4 and 72.3[a] for broccoli and Se(VI), resp.	Finley, 1999
Se concentration	^{75}Se-labeled broccoli	Rat (Whole body)	6 weeks (0.1–1.5 μg Se g^{-1} food)			79.0 and 80.4[a] for 0.1 and 1.5 μg Se g^{-1}, resp.	Finley et al., 2004
Se(IV), SeMeSeCys and SeMet concentration	Broccoli	In vitro (Caco-2 cell model)				–[c]	Zeng et al., 2008
Se concentration and GPx activity	Mushrooms	Human (Plasma and platelets)	4 weeks (150 μg Se d^{-1})	12 and 26 for plasma and platelets, resp.	19 for plasma		Mutanen, 1986
Se concentration	Mushroom	Rat (Plasma and liver)	4 weeks (0.1 μg Se g^{-1} food)			28 and 62[b] for plasma and liver, resp.	Chansler et al., 1986
GPx activity	Mushroom	Rat (Plasma and liver)	4 weeks (0.1 μg Se g^{-1} food)			14 and 5[b] for plasma and liver, resp.	Chansler et al., 1986
Se concentration	Mushroom	Rat (Plasma)	21 days (–[c])	153–172 and 7–10 for selenized and non-selenized mushroom, respectively			da Silva et al., 2010
Se concentration	Shiitake mushroom	In vitro				60 to 80[d] depending on sample preparation (dried mycelium or mycelial lyophilisate)	Turlo et al., 2010
Se concentration	Oyster mushrooms	In vitro				75[d]	Bhatia et al., 2013
GPx activity	Selenized-ramps	Rat (whole blood and liver)	–[c] (600–748 μg Se g^{-1} food)			128 and 114[b] for whole blood and liver, resp.	Whanger et al., 2000
Se concentration	Selenized garlic and cabbage	Boiler chicken (whole body)	4 weeks (0.48–0.51 μg Se g^{-1} food)			70.7 and 61.2[a] for garlic and cabbage, resp.	Seo et al., 2008
GPx activity	Selenized garlic and cabbage	Boiler chicken (liver and plasma)	4 weeks (0.48–0.51 μg Se g^{-1} food)		30 (liver) and 183 (plasma) for garlic and 17 (liver) and 127 (plasma) for cabbage		Seo et al., 2008

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Table 1. (Continued).

Measured parameter	Sample matrix	Model of study	Duration of supplementation (Dose of supplementation)	Se concentration increase (%) after supplementation	GPx increase (%) after supplementation	Relative bioavailability (%)	Ref.
GPx and I 5′deiodinase activity	Selenized-garlic	Rat (Liver)	4–16 days (0.5 μg Se g^{-1} food)		511, 12330 and 20100 for 4, 8 and 16 days, resp.		Ip and Lisk, 1993
SeMet, SeMeSeCys and γ-glu-Se-MeSeCys concentration	Selenized-garlic	In vitro				–[c]	Dumont et al., 2006a
$SeCys_2$, SeMetSeCys, SeMet and Se(IV) concentration	Radish	In vitro				–[c]	Pedrero et al., 2006
Se(IV), Se(VI) and SeMet concentration	Selenized-green onion and chives	In vitro				80–90, 12–30, and 20–22[d] for Se(VI)-, Se(IV)- and SeMet-fortified green onion and chives, resp.	Kápolna and Fodor, 2007
Se concentration	Selenized-cooked leek	In vitro (SHIME model)				78–81[d] for Se(VI)-, and Se(IV)-enriched soil, resp.	Lavu et al., 2012
Se concentration	Selenized-peas	Rat (Plasma, liver, muscle, and kidney)	50 days (13.2–29 μg Se Kg^{-1})			95.4, 103.5, 70.7 and 93.9[b] for plasma, liver, muscle and kidney, resp.	Yan and Johnson, 2011
GPx activity	Selenized-peas	Rat (whole blood and liver)	50 days (13.2–29 μg Se kg^{-1})			86.3 and 84.2[b] for whole blood and liver, resp.	Yan and Johnson, 2011
TrxR Activity	Selenized-peas	Rat (Liver)	50 days (13.2–29 μg Se kg^{-1})			83.0[b]	Yan and Johnson, 2011

[a]Apparent absorption (Equation 1); [b]slope-ratio method (Equation 3); [c]Not given; [d]bioaccessibility (Equation 6).

GPx, glutathione peroxidise; LMW, low molecular weight; NI, no increase; RBC, red blood cell; SeCys, selenocysteine; $SeCys_2$, selenocystine, SeMet, selenomethionine; SeMetSeCys, seleno-methylselenocysteine; γ-glutamyl-Se-MeSeCys, γ-glutamyl-selenomethylselenocysteine; Se(IV), selenite; Se(VI), selenate; SHIME, simulator of human intestinal microbial ecosystem; TrxR, thioredoxin reductase.

Table 2. Application of in vivo and in vitro tests for Se bioavailability in seafood and seaweeds.

Measured parameter	Sample matrix	Model of study	Duration of supplementation (Dose of supplementation)	Se concentration increase (%) after supplementation	GPx increase (%) after supplementation	Relative bioavailability (%)	Ref.
GPx activity	Fish (capelin and mackerel)	Chick (Serum)	9 days (–[a])			48 and 34[b] for capelin and mackerel, resp.	Gabrielsen and Opstvedt, 1980
Se concentration	Tuna	Rat (Young RBC and liver)	4 weeks (0.2 μg Se g^{-1} food)			79[c]	Douglass et al., 1981
Se concentration	Raw, precooked and canned tuna	Rat (Whole blood, liver, kidney, and muscle)	4 weeks (0.05–0.15 μg Se g^{-1} food)			60, 90, 100[b] for whole blood, liver and muscle, resp.	Alexander et al., 1983
GPx activity	Raw, precooked and canned tuna	Rat (Whole blood, liver, and kidney)	4 weeks (0.05–0.15 μg Se g^{-1} food)			44, 38, 75 and 34[b] for whole blood, liver and kidney, resp.	Alexander et al., 1983
Se concentration	Crab, oyster and shrimp	Rat (Whole body)	4 weeks (0.1 μg Se g^{-1} food)			38, 22 and 57[c] for crab, oyster, shrimp, resp.	Mutanen et al., 1986a
Se concentration	Crab, oyster and shrimp	Rat (Whole body)	4 weeks (0.2 μg Se g^{-1} food)			78, 53 and 90[c] for crab, oyster, shrimp, resp.	Mutanen et al., 1986a
Se concentration and GPx activity	Fish	Human (Plasma)	6 weeks (115 $\pm$ 31 μg Se d^{-1})	NI	NI		Meltzer et al., 1993
Se(IV), Se(VI), SeCys and SeMet concentration	Cod	In vitro				–[a]	Crews et al., 1996
Se concentration	Plaice, cod, herring, maatjesherring, coocked shrimp	In vitro				25.8, 16.7, 21.3, 31.3 and 17.2[d] for plaice, cod, herring, maatjesherrin and shrimp, resp.	Shen et al., 1997
Se concentration and GPx activity	Tuna and flounder	Rat (Liver)	9 weeks (0.8 $\pm$ 0.4 μg Se g^{-1} food)	~70 and ~80 for tuna and flounder, resp.	101 and 106 for tuna and flounder, resp.		Wen et al., 1997
Se concentration	Tuna and flounder	Rat (Muscle)	9 weeks (0.8 $\pm$ 0.4 μg Se g^{-1} food)	~65 and ~110 for tuna and flounder, resp.			Wen et al., 1997
GPx activity	Fish based food	Rat (Plasma and liver)	8 weeks (0.1 μg Se g^{-1} food)	203 and 500 for plasma and liver, resp.			Ros et al., 1998
Se concentration and GPx activity	Shrimp	Human (Whole body and plasma)	6 weeks (98 μg d^{-1})	7.6 for plasma	3.6 for plasma	83[c]	Bügel et al., 2001
Se concentration	Raw and cured selenized-salmon	Rat (Whole body)	(–[a]) (0.1 μg Se g^{-1} food)			81.5 and 78.7[c] for raw and cured salmon, resp.	Ørnsrud and Lorentzen, 2002
Se concentration	Raw and cured selenized-salmon	Rat (Plasma, liver, muscle, and femur)	30 days (0–0.2 μg Se g^{-1} food)			125, 115, 225 and 113[b] (raw salmon) for plasma, liver, muscle and femur, resp; and 113, 130, 274 and 135[b] for (cured salmon) for plasma, liver, muscle and femur, resp.	Ørnsrud and Lorentzen, 2002
GPx activity	Raw and cured selenized-salmon	Rat (Serum)	30 days (0–0.2 μg Se g^{-1} food)			1.39 and142[b] for raw and cured salmon, resp.	Ørnsrud and Lorentzen, 2002
Se concentration	Cooked and salted fish	Human (Whole body)	3 days (–[a])			87.8 and 90.4[c] for cooked fish and salted fish, resp.	Fox et al., 2004

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Table 2. (Continued).

Measured parameter	Sample matrix	Model of study	Duration of supplementation (Dose of supplementation)	Se concentration increase (%) after supplementation	GPx increase (%) after supplementation	Relative bioavailability (%)	Ref.
GPx activity	Processed anchovy	Rat (Liver)	7 weeks (0.02 and 0.24 μg Se g^{-1} food)		~100 and ~540 for 0,02 and 0.24 μg Se g^{-1} food, resp.		Haratake et al., 2007
Total Se and SeMet concentrations	Swordfish, sardine, tuna	In vitro				50–83 and 14–19[e] for total Se and SeMet, resp.	Cabañero et al., 2004 Cabañero et al., 2007
Se concentration	Fish and shellfish	In vitro (Caco-2 cell model)				12–100[f]	Calatayud et al., 2012
Se concentration	Seafood	In vitro				4.0–13[d]	Moreda-Piñeiro et al., 2013a; 2013b
Se concentration	Seafood	In vitro				98, 76, 52 and 50[e] for total butter clam, salmon eggs, chinook salmon and Sockeye salmon, resp.	Laird and Chan, 2013
Se concentration	Selenized spirulina	Rat (Liver and Kidney)	56 days (75 μg Se kg^{-1} food)			38 and 84[b] for liver and kidney, resp.	Cases et al., 2001
GPx activity	Selenized spirulina	Rat (Liver, kidney, erythrocyte and plasma)	56 days (75 μg Se kg^{-1} food)	43, 66, 94 and 103 for liver, kidney, erythrocyte and plasma, resp.			Cases et al., 2001
Se concentration	Selemized spirulina pellet fraction	Rat (Liver and kidney)	56 days (75 μg Se kg^{-1} food)			98 and 48[b] for liver and kidney, resp.	Cases et al., 2002
GPx activity	Selemized spirulina pellet fraction	Rat (Liver, kidney, erythrocyte and plasma)	56 days (75 μg Se kg^{-1} food)			20, 36, 65 and 63[b] for liver, kidney, erythrocyte and plasma, resp.	Cases et al., 2002
Se concentration	Selemized spirulina retentate fraction	Rat (Liver and kidney)	56 days (75 μg Se kg^{-1} food)			89 and 112[b] for liver and kidney, resp.	Cases et al., 2002
GPx activity	Selemized spirulina retentate fraction	Rat (Liver, kidney, erythrocyte and plasma)	56 days (75 μg Se kg^{-1} food)			106, 103, 131 and 133[b] for liver, kidney, erythrocyte and plasma, resp.	Cases et al., 2002
Se concentration	Selemized spirulina	Rat (Whole body)	56 days (75 μg Se kg^{-1} food)			82, 86 and 87[c] for spirulina, spirulina pellet and spirulina retentate, resp.	Cases et al., 2002
Se concentration	Edible seaweed	In vitro				48–100[d]	Moreda-Piñeiro et al., 2013a; 2013b
Se concentration	Cooked seaweed	*In-vitro*				15.0, 61.3, 15.7 and 14.6[d] for cooked Kombu, Wakame, Sea lettuce and Nori, resp.	García-Sartal et al., 2013
Se concentration	Laver	In vitro				65[e]	Laird and Chan, 2013

[a]Not given; [b]Slope-ratio method (Equation 3); [c]Apparent absorption (Equation 1); [d]Bioavailability (Equation 7); [e]Bioaccessibility (Equation 6); [f]Bioavailability (Equation 11).
GPx, glutathione peroxidise; NI, no increase; RBC, red blood cell; SeCys, selenocysteine; SeMet, selenomethionine; Se(IV), selenite; Se(VI), selenate.

Table 3. Application of in vivo and in vitro tests for Se bioavailability in yeast and yeast-based nutritional supplements.

Measured parameter	Sample matrix	Model of study	Duration of supplementation (Dose of supplementation)	Se concentration increase (%) after supplementation	GPx (TrxR) increase (%) after supplementation	Relative bioavailability (%)	Ref.
Se concentration and GPx activity	Selenized-yeast	Human (Plasma and platelet)	11 weeks (200 μSe d^{-1})	148 for plasma	170 for platelet		Levander et al., 1983
Se concentration and GPx activity	Se yeast	Rat (RBC)	18 days (0.1–0.5 μg Se g^{-1})			520 and 120[a] for Se conc. and GPx activity, resp.	Smith and Picciano, 1987
Se concentration and GPx activity	selenized-yeast	Human (Plasma and platelet)	8.5 weeks (100 μSe d^{-1})	30 for plasma	NI and 50 for plasma and platelet, resp.		Nève et al., 1988
Se concentration and GPx activity	selenized-yeast	Human (Plasma and platelet)	8.5 weeks (200 μSe d^{-1})	40 for plasma	NI and 100 for plasma and platelet, resp.		Nève et al., 1988
Se concentration	Selenized yeast	Human (Plasma)	14 days (5 μg Se d^{-1})	29.3			Bogye et al., 1988
Se concentration and GPx activity	Selenized yeast	Human (Plasma)	12 weeks (200 μg Se-yeast d^{-1})	55	NI		Alfihan et al., 1991
Se concentration and GPx activity	Selenized yeast	Human (Plasma)	52 weeks (200 μg Se-yeast d^{-1})	650	210		Xia et al., 1992
Se concentration and GPx activity	Selenized yeast	Human (Plasma and platelet)	32 weeks (200 μg Se d^{-1} (as brewer's yeast or as Se-enriched yeast)	258 and 312 for plasma and platelet, resp.	37 and 64 for plasma and platelet, resp.		Thompson et al., 1993
Se concentration	Se-yeast	Channel catfish (Liver and muscle)	9 weeks (0–0.4 μg Se g^{-1} food)			184 and 453[a] for liver and muscle, resp.	Wang and Lovell, 1997
GPx activity	Se-yeast	Channel catfish (Plasma and liver)	9 weeks (0–0.4 μg Se g^{-1} food)			116 and 149[a] for plasma and liver, resp.	Wang and Lovell, 1997
Se concentration	Selenized tablets	Human (Plasma)	6 months (10–40 μg Se d^{-1})	16.4			Finley et al., 1999
Se concentration	Se-yeast	Rat (Liver serum and erythrocyte)	4 weeks (1.420 μg Se g^{-1} food)			165, 135 and 137[a] for liver serum and erythrocyte, resp.	Yoshida et al., 1999
Se concentration	Se-yeast	Rat (Liver serum and erythrocyte)	4 weeks (1.214 μg Se g^{-1} food)			148, 155 and 143[a] for liver serum and erythrocyte, resp.	Yoshida et al., 1999
GPx activity	Se-yeast	Rat (Liver serum and erythrocyte)	4 weeks (1.420 μg Se g^{-1} food)			111, 197 and 120[a] for liver serum and erythrocyte, resp.	Yoshida et al., 1999
GPx activity	Se-yeast	Rat (Liver serum and erythrocyte)	4 weeks (1.214 μg Se g^{-1} food)			105, 114 and 1250[a] for liver serum and erythrocyte, resp.	Yoshida et al., 1999
Se concentration and GPx activity	Selenized yeast	Human (Plasma)	16 weeks (200 μg Se d^{-1})	710	300		Alfthan et al., 2000
Se concentration	Brewers yeast	Human (Whole body)	3 days (–[b])			53.5[c]	Fox et al., 2004
Se concentration	Yeast-based intervention agents: SelenoPrecise	Human (Plasma)	6 months (100 or 200 or 300μg Se d^{-1})	61, 113 and153 for 100 or 200 or 300μg Se d^{-1}, resp.			Larsen et al., 2004
Se concentration	Yeast-based intervention agents: Cypress System plus SelenoPrecise	Human (Platelet)	24 months (100 or 200 or 300 μg Se d^{-1})	89, 232, and 359 for 100 or 200 or 300 μg Se d^{-1}, resp.			Larsen et al., 2004
Se concentration	Baker's yeast and yeast tablets	In vitro				55–80[d]	Dumont et al., 2004

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Table 3. (Continued).

Measured parameter	Sample matrix	Model of study	Duration of supplementation (Dose of supplementation)	Se concentration increase (%) after supplementation	GPx (TrxR) increase (%) after supplementation	Relative bioavailability (%)	Ref.
Se and SeMet concentrations	Selenized yeast	In vitro				89 (total Se) and 41[d] (SeMet) for yeast; and 26 –37[d] for yeast-based nutritional supplements	Hinojosa-Reyes et al., 2006a; 2006b
Se concentration	Se-yeast	Pig (Drip loss, loin and hair)	–[b] (0.3 mg Se Kg^{-1})	23, 79, 40 and 76 for liver, lojn serum and hair, resp.		306, 192 and 197[a] for drip loss, lojn and hair, resp.	Mateo et al., 2007
Se concentration	Selenized yeast	Human (Whole body)	1 days (327 μg Se d^{-1})			89[c]	Bügel et al., 2008
Se concentration	Se-yeast	Boiler chicken (Muscle, kidney, liver and pancreas)	21 days (~0.2 μg Se g^{-1} food)	57.8, 163.9, 100 and 133.3 for muscle, kidney, liver and pancreas, resp.			Wang and Xu, 2008
GPx activity	Se-yeast	Boiler chicken (Plasma and liver)	21 days (~0.2 μg Se g^{-1} food)	72.4 and 39.7 for plasma and liver, resp.			Wang and Xu, 2008
Se concentration	Selenized yeast	Human (Plasma)	3 years (200μg Se d^{-1})	92			Bost and Blouin, 2009
GPx and TrxR activity	Se-yeast	Trout (Liver)	10 weeks (~2–8 μg Se g^{-1} food)		NI–6.5 (14.3–85.7)		Rider et al., 2009
Se concentration	Se-yeast	Pig (Serum, loin, liver, kidney, pancreas and spleen)	6 weeks (1.0 μg Se g^{-1} food)	NI, 11, NI, 1.0, NI and NI for serum, loin, liver, kidney, pancreas and spleen, resp.			Jang et al., 2010
Se concentration	Se-yeast	Pig (Serum, loin, liver, kidney, pancreas and spleen)	6 weeks (1000 μg Se g^{-1} food)	NI, 61, 54, 3.0, NI, and 11 for serum, loin, liver, kidney, pancreas and spleen, resp.			Jang et al., 2010
Se concentration	Se-yeast	Trout (Whole body)	10 weeks (–[b])			~65[e]	Rider et al., 2010
Se concentration	Se-yeast	Trout (Plasma, liver, muscle, kidney and whole blood)	10 weeks (~2 μg Se g^{-1} food)	35.7,79, 76, 189 and 29 for plasma, liver, muscle, kidney and whole blood, resp.			Rider et al., 2010
GPx and TrxR activity	Se-yeast	Trout (Liver)	10 weeks (~2 μg Se g^{-1} food)		49.4 (15.7)		Rider et al., 2010
Se concentration	Selenized tablets	Human (Plasma)	1.5 hours (300 mg SeCys)	219			Mahmoud, 2012
Se concentration	Se-yeast	Ewe (Whole blood)	1 year (4.9–24.5 mg Se wk^{-1} food)	~40,~280 and ~230 for 4.9, 14.7 and 24.5 mg Se wk^{-1}, resp.			Hall et al., 2012
Se concentration	Se-yeast	Broiler chicken (Whole body)	24 days (0.3 μg Se g^{-1} food)			49[c]	Briens et al., 2013

Se, SeMet, Se(IV) and SeMeSeCys concentrations	Se-enriched yeast and Se-based food supplement	*In-vitro* (Caco-2 cell model)					57, 100 and 80[d] for Se-enriched yeast, Se(VI)-based food supplement, and Se (IV)-based food supplement, resp. 7, 14 and 1[f] for Se-enriched yeast, Se(VI)-based food supplement, and Se (IV)-based food supplement, resp. 70, 100 and 100[d] for SeMet, Se(IV) and SeMeSeCys, resp. 24, 0 and 0[f] for SeMet, Se (IV) and MeSeCy, resp.	Thiry et al., 2013a
Se concentration	Se-yeast	Yellowtail kingfish (Muscle)	6 weeks (2μg Se g^{-1} food)	195		34.9		Le and Fotedar, 2014

[a]slope-ratio method (Equation 3); [b]not given[c]apparent absorption (Equation 1); [d]bioaccessibility (Equation 6); [e]apparent digestibility (Equation 5); [f]bioavailability (Equation 11).
GPx, glutathione peroxidise; NI, no increase; RBC, red blood cell; SeMet, selenomethionine; SeMetSeCys, seleno-methylselenocysteine; Se(IV), selenite; Se(VI), selenate; TrxR, thioredoxin reductase.

Table 4. Application of in vivo and in vitro tests for Se bioavailability in meat, Brazil nut, milk, eggs and selected diets.

Measured parameter	Sample matrix	Model of study	Duration of supplementation (Dose of supplementation)	Se concentration increase (%) after supplementation	GPx increase (%) after supplementation	Relative bioavailability (%)	Ref.
Se concentration	Chicken meat	Human (Whole body)	5 /10 days (85 μg ^{74}Se(VI))			70.9 and 72.0[a] for 5 and 10 days respectively	Christensen et al., 1983
Se concentration and GPx activity	Meat	Human (Plasma)	9 weeks (55, 135 and 215 pg Se d^{-1})	69		76[a]	Van der Torre et al., 1991
Se concentration	Sheep muscle, liver and hemoglobin	Rat (Whole blood, muscle and liver)	4 weeks (0.05–0.15 μg Se g^{-1} food)			87–119, 67–142 and 78–118[b] for whole blood, muscle and liver, resp.	Butler et al., 1991
GPx activity	Sheep muscle, liver and hemoglobin	Rat (Whole blood, muscle and liver)	4 weeks (0.05–0.15 μg Se g^{-1} food)			76–135, 80–93 and 80–108[b] for whole blood, muscle and liver, resp.	Butler et al., 1991
GPx activity	Raw and cooked beef liver and beef striploin	Rat (Liver)	8 weeks (~0.115 μg Se g^{-1} food)		108 and 103–106 for beef liver and beef, resp.		Shi and Spallholz, 1994a
Se concentration	Raw and cooked beef	Rat (Whole blood, muscle and liver)	8 weeks (108 μg Se g^{-1} food)			87–119, 59–142 and 78–118[b] for whole blood, muscle and liver, resp.	Shi and Spallholz, 1994b
Se concentration and GPx activity	Raw and cooked beef	Rat (Liver)	8 weeks (108 μg Se g^{-1} food)	~125 and ~135 for raw and cooked beef, resp.	~110 and ~115 for raw and cooked beef, resp.		Shi and Spallholz, 1994b
Se concentration and GPx activity	Veal, chicken, beef, pork and lamb	Rat (Liver)	9 weeks (0.8±0.4 μg Se g^{-1} food)	~80, ~80, ~57, ~60 and ~80 for veal, chicken, beef, pork and lamb, resp.	77, 77, 80, 86 and 58 for veal, chicken, beef, pork and lamb, resp.		Wen et al., 1997
Se concentration	Veal, chicken, beef, pork and lamb	Rat (Muscle)	9 weeks (0.8±0.4 μg Se g^{-1} food)	~85, ~62, ~65, ~75 and ~65 for veal, chicken, beef, pork and lamb, resp.			Wen et al., 1997
Se concentration and GPx activity	Pork meat	Human (Whole body and plasma)	3 weeks (106μg Se d^{-1})		NI for plasma	94[a]	Bügel et al., 2004
Se concentration	^{75}Se-labeled beef and pork	Rat (Whole body)	6 weeks (1.5 μg Se g^{-1} food)			82.0 and 75.8[a] for 0.1 and 1.5 μg Se g^{-1}, resp.	Finley et al., 2004
Se concentration	Meat chicken	*In-vitro*				67.0[c]	Govasmark et al., 2010
Se concentration	Fortified chicken meat	*In-vitro*				62–65[c]	Brandt-Kjelsen et al., 2012
Se concentration	Unaged and aged beef	*In-vitro*				75–91 and 58–90[c] for unaged and aged meat, resp.	Ramos et al., 2012
Se concentration	Wild game meat and organs	*In-vitro*				45, 37, 29, 52, 62 and 35[c] for rabbit meat, deer meat, moose meat, deer liver, moose kidney and moose liver, resp.	Laird and Chan, 2013
Se concentration	Brazil nut	Rat (Plasma and liver)	4 weeks (0.1 μg Se g^{-1} food)			143 and 162[b] for plasma and liver, resp.	Chansler et al., 1986
GPx activity	Brazil nut	Rat (Plasma and liver)	4 weeks (0.1 μg Se g^{-1} food)			122 and 124[b] for plasma and liver, resp.	Chansler et al., 1986
SeMet and $SeCys_2$ concentrations	Brazil nuts	*In-vitro*				–[d]	Dumont et al., 2006b

Se concentration and GPx activity	Brazil nut	Human (Plasma)	12 weeks (100 μg Se (content in two Brazil nut))	64	8.3	Thompson et al., 2008
Se concentration	Brazil nut	*In-vitro*			74[c]	Gomes da Silva et al., 2013
Se concentration	Selenized-milk	Rat (Plasma)	4 weeks (86–121 μg Se g^{-1} food)		67–100[b]	Mutanen et al., 1986b
GPx activity	Selenized-milk	Rat (Plasma and liver)	4 weeks (86–121 μg Se g^{-1} food)		66–87 and 80–95[c] for plasma and liver, resp.	Mutanen et al., 1986b
Se concentration	Human, cow, goat and sheep milk	*In-vitro*			11.1 (human), 6.8 (cow), 6.2 (goat) and < 2[e] (sheep) for whole milk; and 15 (human), 10 (cow), 12 (goat) and 2.4[e] (sheep) for skim milk	Shen et al., 1996
Se concentration	Milk-based infant formula	Human (Whole body)	1 day (10μg Se d^{-1} (as ^{76}Se(VI) or ^{74}Se(IV)))		97.1 and 73.4[a] for ^{76}Se(VI) and ^{74}Se(IV), resp.	Van Dael et al., 2002
Se concentration	Cow and goat milk	Rat (Whole body)	30 days (–[d])		87.3–87.4 and 91.5–92.9[a] for cow and goat milk, resp.	Alférez et al., 2003; Barrionuevo et al., 2003
Se concentration	Milk	Human (Whole body)	8 weeks (–[d])		61–77[a]	Chen et al., 2004
Se concentration	Cow milk	*In-vitro*			$\sim$11 and < 10[e] after supplementation with forage with organic and inorganic Se, resp.	Muñiz-Naveiro et al., 2006
Se concentration	Skimmed and whole milk	*In-vitro*			–[d]	Zohoori et al., 2009
Se concentration	Fermented milk	*In-vitro*			76[d]	Alzate et al., 2010
Se concentration	Selenized eggs	Human (Whole body)	36 days (–[d])		54.1–55.4, 76.7–83.0 and 79.0–85.2[a] for egg white (high dose), egg yolk (high dose and egg yolk (low dose), resp.	Sirichakwal et al., 1985
SeMet and SeCys concentrations	Chicken eggs	*In-vitro*			–[d]	Lipiec et al., 2010

[a]apparent absorption (Equation 1); [b]slope-ratio method (Equation 3); [c]bioaccessibility (Equation 6); [d]not given; [e]bioavailability (Equation 7).
GPx, glutathione peroxidise; SeMet, selenomethionine; SeCys, selenocysteine, $SeCys_2$, selenocystine.

Table 5. Application of in vivo and in vitro tests for Se bioavailability in Se chemically pure forms.

Measured parameter	Se chemically pure species	Model of study	Duration of supplementation (Dose of supplementation)	Se concentration increase (%) after supplementation	GPx (TrxR) increase (%) after supplementation	Relative bioavailability (%)	Ref.
Se concentration	Se(IV)	Human (Whole body)	1 day (10 μg ^{75}Se(IV))			55.5[a]	Thompsonand Stewart, 1974
Se concentration	SeMet	Human (Whole body)	1 day (2 μg ^{75}SeMet)			95.5–97.3[a]	Griffiths et al., 1976
Se concentration	SeMet	Rat (Whole body)	4 weeks (4 μg (as ^{75}SeMet))			91.3[b]	Thompson et al., 1975a
Se concentration	SeCys and SeMet	Rat (Whole body)	7 days (5 μg (as ^{75}SeCys and ^{75}SeMet))			81 and 86[b] for ^{75}SeCys and ^{75}SeMet, resp.	Thompson et al., 1975b
Se concentration	Se(IV) and SeMet	Human (Whole body, plasma and platelet)	5 days (1.0 mg as Se(IV) and 1.1 mg as SeMet)	45.2 and 25.6 for plasma and platelet, resp.		69–85 and 22[a] for Se(IV) and SeMet, resp.	Thompson et al., 1978
Se concentration	Se(IV)	Human (Platelet)	20 days (0.5 mg as Se(IV))	78.6			Thompson et al., 1978
GPx activity	SeMet	Chick (Serum)	9 days (–[c])			78[d] for SeMet	Gabrielsen and Opstvedt, 1980
Se concentration and GPx activity	Se(IV)	Human (Plasma and platelet)	17 weeks (100μg Se d^{-1} (as Se(IV)))	54 and 26.2 for plasma and platelet, resp.	24 for plasma		Thompson et al., 1982
Se concentration and GPx activity	SeMet	Human (Plasma and platelet)	17 weeks (**100μg Se d^{-1} (as SeMet))**	196 and 197 for plasma and platelet, resp.	29 for plasma		Thompson et al., 1982
Se concentration and GPx activity	Se(IV)	Human (Plasma and platelet)	4 weeks (**500μg Se d^{-1} (as Se(IV)))**	100 and 50 for plasma and platelet, resp.	19 for plasma		Thompson et al., 1982
Se concentration and GPx activity	Se(IV)	Human	11 weeks (200μg Se(IV) d^{-1})	61 for plasma	170 for platelet		Levander et al., 1983
Se concentration	Se(VI)	Human (Whole body)	5 /10 days (69 μg ^{76}Se (VI))			34.7 and 37.6[a] for 5 and 10 days respectively	Christensen et al., 1983
Se concentration	Se(VI)	Human (Whole body)	10 days (100μg Se d^{-1} (as ^{74}Se(VI))			52.9–54.2[a]	Kasper et al., 1984
Se concentration	Se(IV)	Chick (Whole body)	3 weeks (0.5 μg Se g^{-1} (as ^{75}Se(IV))			56.6–76.7[b]	Mutanen and Mykkänen, 1984
Se concentration and GPx activity	Se(IV)	Human (Plasma)	4 weeks (150μg Se(IV) d^{-1})	32			Mutanen, 1986
Se concentration	Se(IV) and SeMet	Rat (RBC)	18 days (0.1–0.5μg Se g^{-1} (as Se(IV) and SeMet))			100 and 800[d] for Se(IV) and SeMet, resp.	Smith and Picciano, 1987
GPx activity	Se(IV) and SeMet	Rat (RBC)	18 days (0.1–0.5μg Se g^{-1} (as Se(IV) and SeMet))			100 and 250[d] for Se(IV) and SeMet, resp.	Smith and Picciano, 1987
Se concentration	Se(IV) and Se(VI)	Human (Whole body)	1 day (10 mg Se d^{-1} (as Se(VI) and Se(IV))			94 and 62[a]for Se(VI) and Se(IV), resp.	Thompson and Robinson, 1986
Se concentration and GPx activity	Se(IV)	Human (Plasma and platelet)	28 days (200μg Se d^{-1} (as Se(IV)))	40 and 70 for plasma and platelet, resp.	24 and 105 for plasma and platelet, resp.		Thompson et al., 1988
Se concentration and GPx activity	SeMet	Human (Plasma and platelet)	60 days (100μg Se d^{-1} (as SeMet))	40 for plasma	NI and 100 for plasma and platelet, resp.		Nève et al., 1988
Se concentration	SeMet	Rat (Plasma, liver and muscle)	28 days (0.5 μg Se g^{-1} (as SeMet))	160, 150 and 120 for plasma, liver and muscle, resp.			Waschulewski and Sunde, 1988
Se concentration	SeMet	Rat (Liver, whole blood and muscle)	9 weeks (0.2–4.0 μg Se g^{-1} (as SeMet))	124–335, 124–413 and 280–2641, for liver, whole blood and muscle, resp.			Whanger and Butler, 1988

GPx activity	SeMet	Rat (BBC, muscle, liver and kidney)	9 weeks (0.2–4.0 μg Se g^{-1} (as SeMet))		96–104, 111–120, 81–103 and 92–118 for RBC, nuscle, liver and kidney, resp.		Whanger and Butler, 1988
Se concentration	Se(VI)	Human (Whole body)	30 day (18 $\pm$ 1 and 119 $\pm$ 1μg Se d^{-1} (as ^{74}Se (VI))			58 and 89[a] for 18 $\pm$ 1 and 119 $\pm$ 1μg Se d^{-1}, resp.	Martin et al., 1989
Se concentration	SeMet	Human (Platelet)	12 months (300 μg Se d^{-1} (as SeMet))	241			Clausen et al., 1989
Se concentration and GPx activity	Se(VI)	Human (Plasma and platelet)	14 days (100 μg Se d^{-1} (as Se(VI)))	223 and 58 for people from low and high Se areas, resp (plasma) and 118 and 13 for people from low and high Se areas, resp. (platelet)	193 and 28 for people from low and high Se areas, resp (plasma)		Xia et al., 1989
Se concentration and GPx activity	Se(VI)	Human (Plasma and platelet)	14 days (200 μg Se d^{-1} (as Se(VI)))	200 and 62 for people from low and high Se areas, resp (plasma) and 100 and 30 for people from low and high Se areas, resp (platelet)	90 and 16 for people from low and high Se areas, resp (plasma)		Xia et al., 1989
Se concentration and GPx activity	Se(IV) and Se(VI)	Human (Plasma)	12 weeks (200 μg Se(IV) d^{-1} and 200 μg Se(VI) d^{-1})	15 and NI for Se(IV) and Se(VI), resp.	30 and 30 for Se(IV) and Se(VI), resp.		Alfihan et al., 1991
Se concentration and GPx activity	Se(VI)	Human (Plasma)	52 week (200 μg Se d^{-1} (as Se(VI)))	275	200		Xia et al., 1992
Se concentration and GPx activity	Se(VI)	Human (Plasma and platelet)	32 weeks (200 μg Se d^{-1} (as Se(VI))	107 and 54 for plasma and platelet, resp.	37 and 99 for plasma and platelet, resp.		Thompson et al., 1993
GPx activity	Se(IV)	Rat (Liver)	4–16 days (0.5 μg Se g^{-1} (as Se(IV)))		580, 16670 and 22330 for 4, 8 and 16 days, resp.		Ip and Lisk, 1993
GPx activity	SeMet	Rat (Liver)	8 weeks (0.115 μg Se g^{-1} (as SeMet))		122		Shi and Spallholz, 1994a
Se concentration	SeMet	Channel catfish (Liver and muscle)	9 weeks (0–0.4 μg Se g^{-1} (as SeMet))			197 and 478[d] for liver and muscle, resp.	Wang and Lovell, 1997
GPx activity	SeMet	Channel catfish (Plasma and liver)	9 weeks (0–0.4 μg Se g^{-1} (as SeMet))			116 and 147[d] for plasma and liver, resp.	Wang and Lovell, 1997
Se concentration	Se(IV)	Rat (Whole blood, plasma, muscle, kidney and liver)	7 days (0.1 μg Se g^{-1} (as Se(IV)))	28, 30, 60, 200 and 261 for whole blood, plasma, muscle, kidney and liver, resp.			Serra et al., 1997
GPx activity	Se(IV)	Rat (Plasma and liver)	8 weeks (0.1 μg Se g^{-1} (as Se(IV)))			500 and 500[e] for plasma and liver, resp.	Ros et al., 1998

(*Continued on next page*)

Table 5. (Continued).

Measured parameter	Se chemically pure species	Model of study	Duration of supplementation (Dose of supplementation)	Se concentration increase (%) after supplementation	GPx (TrxR) increase (%) after supplementation	Relative bioavailability (%)	Ref.
Se concentration and GPx activity	Se(IV)	Human (Plasma)	16 weeks (200 μg Se(VI) d^{-1})	530	300		Alfthan et al., 2000
Se concentration	SeMet	Rat (Liver and Kidney)	56 days (75 μg Se Kg^{-1} (as SeMet))			59 and 125[d] for liver and kidney, resp.	Cases et al., 2001
GPx activity	SeMet	Rat (Liver, kidney, erythrocyte and plasma)	56 days (75 μg Se Kg^{-1} (as SeMet))			66, 80, 96 and 100[d] for liver, kidney, erythrocyte and plasma, resp.	Cases et al., 2001
GPx activity	Se(IV)	Crucian carp (Plasma and liver)	30 days ~0.5 μg Se g^{-1} (as Se(IV))		~100 and ~55 for plasma and liver, resp.		Wang et al., 2007
GPx activity	SeMet	Crucian carp (Plasma and liver)	30 days (~0.5 μg Se g^{-1} (as SeMet)		~130 and ~69 for plasma and liver, resp.		Wang et al., 2007
Se concentration	$SeSO_3$	Rat (Liver, kidney and whole blood)	2 and 7 days (0.1 mg Se Kg^{-1} (as $SeSO_3$))			64[d](liver), 86[d] (kidney) and 65[d] (whole blood) after 2 days; and 85[d] (liver) and 93[d] (kidney) after 7 days	Peng et al., 2007
GPx Activity	$SeSO_3$	Rat (Liver and kidney)	2 and 7 days (0.1 mg Se Kg^{-1} (as $SeSO_3$))			65[d] (liver) for after 2 days and 88[d] (kidney) after 7 days	Peng et al., 2007
TrxR Activity	$SeSO_3$	Rat (Liver and kidney)	7 days (0.1 mg Se Kg^{-1} (as $SeSO_3$))			75 and 78[d] for liver and kidney, resp.	Peng et al., 2007
Se concentration	Se(IV), SeMeSeCys and SeMet	In vitro (Caco-2 cell model)				–[c]	Zeng et al., 2008
Se concentration	SeMet	Strip bass (Whole body)	12 weeks (0.9–2.55 mg Se Kg^{-1} (as SeMet))			330[d]	Jaramillo et al., 2009
Se concentration and GPx activity	Se(IV) and SeMet	Trout (Serum)	12 weeks (0.15 and 0.30 mg Se Kg^{-1} (as Se(IV) and SeMet))	19–22 (0.1 mg Se Kg^{-1}) and 35–50 (0.30 mg Se Kg^{-1}) for Se(IV); and 25–34 (0.1 mg Se Kg^{-1}) and 25–37 (0.1 mg Se Kg^{-1}) for SeMet	38–45 (0.1 mg Se Kg^{-1}) and 58–72 (0.30 mg Se Kg^{-1}) for Se(IV); and 131–139 (0.1 mg Se Kg^{-1}) and 152–174 (0.1 mg Se Kg^{-1}) for SeMet		Küçükbay et al., 2009
Se concentration	Se(IV) and SeMet	Trout (Muscle)	12 weeks (0.15 and 0.30 mg Se Kg^{-1} (as Se(IV) and SeMet))	40–42 (0.1 mg Se Kg^{-1}) and 74–82 (0.30 mg Se Kg^{-1}) for Se(IV); and 120–123 (0.1 mg Se Kg^{-1}) and 152–154 (0.1 mg Se Kg^{-1}) for SeMet			Küçükbay et al., 2009
Se concentration and GPx activity	SeMet and nano-Se	Crucian carp (Muscle)	30 days (0.55 mg Se Kg^{-1} (as SeMet and nano-Se))	122 and 169 for SeMet and nano-Se, resp.	60 (SeMet) and 62 (nano-Se) for liver and 166 (SeMet) and 173 (nano-Se) for plasma		Zhou et al., 2009
Se concentration	Se(IV)	Trout (Whole body)	10 weeks (–[c])			~58[f]	Rider et al., 2010
Se concentration	Se-yeast	Trout (Plasma, liver, muscle, kidney and whole blood)	10 weeks (~2 μg Se g^{-1} food)	21.4, 83.8, 9.5, 10 and 15.5 for plasma, liver, muscle, kidney and whole blood, respectively			Rider et al., 2010

GPx and TrxR activity	Se(IV)	Trout (Liver)	10 weeks (~2 μg Se g^{-1} food)		7.8 (13.4)		Rider et al., 2010
GPx activity	Se(VI)	Rat (Whole body, liver and erythrocyte)	40 days (0.180 mg Se Kg^{-1} (as Se(VI)))		1.9 and 19.2 for liver and erythrocyte, respectively	85.4[a]	Díaz-Castro et al., 2011
Se concentration	Se(IV) and Se(VI)	Ewe (Whole blood)	1 year (4.9–24.5 mg Se wk^{-1} (as Se(IV) and Se (VI)))	NI,~45 and ~45 for 4.9, 14.7 and 24.5 mg Se wk^{-1}, resp.			Hall et al., 2012
Se concentration	Se(VI), SeMet and nano-Se	*In-vitro* (Caco-2 cell model)				8.36, 4.75 and 0.99[g]for nano-Se, SeMet and Se(VI), resp.	Wang et al., 2012
Se concentration	Se(VI) and HMSeBA	Broiler chicken (Whole body)	24 days (0.3 mg Se Kg^{-1} (as Se(VI) and HMSeBA)			24 and 46[a] for Se(VI) and HMSeBA, resp.	Briens et al., 2013
Se concentration	Se(VI), Se(IV), SeMet and SeMeSeCys	In vitro (Caco-2 cell model)				–[c]	Thiry et al., 2013b
Se concentration	Se(VI), Se(IV), SeMet and SeMeSeCys	In vitro (Caco-2 cell model)				–[c]	Pick et al., 2013
Se concentration	Se(IV), SeMet and SeCys	Yellowtail kingfish (Muscle)	6 weeks (2μg Se g^{-1} food)	14.3, 191 and 52.4 for Se(IV), SeMet and SeCys, resp.	127.8, 36.1 and 34.9 for Se(IV), SeMet and SeCys, resp.		Le and Fotedar, 2014

[a]Apparent absorption (Equation 1); [b]intestinal absorption (Equation 4); [c]not given; [d]slope-ratio method (Equation 3); [e]GPx activity increase after supplementation (compared with a diet control); [f]apparent digestibility (Equation 5); [g]bioavailability (Equation 11).

GPx, glutathione peroxidise; HMSeBA, 2-hydroxy-4-methylselenobutanoic acid; nano-Se, seleno-nanoparticle; NI, no increase; RBC, red blood cell; SeCys, selenocysteine; SeMet, selenomethionine; SeMetSeCys, seleno-methylselenocysteine; Se(IV), selenite; Se(VI), selenate; $SeSO_3$, selenosulfate;TrxR, thioredoxin reductase.

compounds). However, the Se concentration in the plasma of rats fed with Se-fortified mushrooms was higher than in rats fed with a normal diet containing selenate (da Silva et al., 2010). These results showed that Se-fortified mushrooms could increase the Se bioavailability of this food (da Silva et al., 2010). Similarly, high bioaccessibility (60–80%) was found in Se-fortified mushroom by in vitro tests (Turlo et al., 2010; Bhatia et al., 2013). Approximately 25% of the solubilized Se was dissolved in the pancreatic juice (Turlo et al., 2010). Se speciation in the gastrointestinal extract was dominated by SeMet, which accounted for 73% of the extracted Se. A small SeMet percentage (2%) was present as SeOMet, and as other oxidized Se-species (Bhatia et al., 2013). Only a percentage of 2% of the detected species was present as inorganic Se (Se(IV)). A number of other low molecular weigh selenocompounds generated during the gastrointestinal digestion, accounting for 25% of the detected Se-based chromatographic signals, remained unidentified (Bhatia et al., 2013).

Se in Se-fortified ramps, garlic, and cabbage (Whanger et al., 2000; Seo et al., 2008) has been found to be equally bioavailable than selenite and selenate solutions when bioavailability was based on repletion of tissue Se concentrations and GPx activity in rats (Ip and Lisk, 1993; Whanger et al., 2000) and boiler chickens (Seo et al., 2008). Thus, Se in Se-fortified garlic, cabbage and ramp is highly bioavailable and can potentially be beneficial in enhancing Se status and GPX activity. Bioavailability of Se in Se-fortified garlic was also achieved by in vitro approaches (Dumont et al., 2006a). Garlic samples were treated with saliva and simulated gastric and intestinal fluids, and findings showed the same Se species pattern [SeMet, SeMetSeCys and γ-glutamyl-selenomethylselenocysteine (γ-glutamyl-Se-Me-SeCys)]. In addition, unknown Se compounds were assessed in saliva and in both simulated fluids. No data regarding bioaccessibility percentages for each Se species were given.

In vitro approaches were also applied to Se bioaccessibility assessment in Se-fortified radish (Pedrero et al., 2006), green onion (Kápolna and Fodor, 2007), chieves (Kápolna and Fodor, 2007) and leek (Lavu et al., 2012). Low Se transformation into organic forms was observed in radish plants grown in a Se(VI)-enriched culture media. On the contrary, studies developed with plants exposed to selenite showed that percentages higher than 95% in the bioaccesibility fraction comprised selenocystine ($SeCys_2$), SeMet, and SeMetSeCys (Pedrero et al., 2006). The concentrations of $SeCys_2$, SeMet, and SeMetSeCys in raw samples remained almost unaltered after a simulated GI digestion, and the analysis of the GI digests showed that almost 100% of Se in the raw plants was found in the bioaccessible fraction (Pedrero et al., 2006). Results regarding Se(VI)-fortified green onion and chieves showed that 80–90% of the total Se content (mainly as selenate) became bioaccessible in the simulated GI digests. Otherwise, a lower Se bioaccessible percentage was obtained in Se(IV)-fortified green onion and chives (12–30%), and also in SeMet-fortified green onion and chives (20–22%) (Kápolna and Fodor, 2007). Se bioavailability was also assessed by SHIME simulator in Se(IV) and Se(VI)-fortified leek (Lavu et al., 2012). The lowest Se uptake was observed when using Se(IV)-based fertilizers. When soil was amended with Se(VI)-based fertilizers, a percentage of 55% of total Se was found as inorganic forms in leek; whereas, only 21% was inorganic Se when fertilizing with Se(IV)-based fertilizers. Most of Se in the Se-fortified leek was found to be bioaccessible in the stomach (~60%), and in the small intestine (~80%). Regarding simulated colon digestions, Se bioaccessibility in Se-enriched leek samples started to decrease to below 50% after a 24 h incubation period. The decrease on Se bioaccessibility in the colon can probably be attributed to sorption phenomena between Se and organic matter, or to uptake processes by the colon microbial biomass.

Se in naturally selenized peas was found to be highly bioavailable after in vivo rat assays (Yan and Johnson, 2011). The overall Se bioavailability was ~88% higher in peas than that observed when testing SeMet solutions.

Selenium bioavailability in seafood

Several in vivo (Gabrielsen and Opstvedt, 1980; Levander et al., 1981; Alexander et al., 1983; Mutanen et al., 1986a; Meltzer et al., 1993; Wen et al., 1997; Ros et al., 1998; Hagmar et al., 1998; Bügel et al., 2001; Ørnsrud and Lorentzen, 2002; Fox et al., 2004; Haratake et al., 2007) and in vitro (Crews et al., 1996; Shen et al.,1997; Cabañero et al., 2004; 2007; Calatayud et al., 2012; Moreda-Piñeiro et al., 2013a; 2013b, Laird and Chan, 2013) studies have been carried out to assesses the bioavailability of Se in seafood (Table 2).

In vivo human tests showed that fish is a highly bioavailable source of dietary Se (Fox et al., 2004). Animal supplementation with Se rich seafood increased Se tissue concentration and apparent Se absorption and retention (Meltzer et al., 1993; Hagmar et al., 1998; Bügel et al., 2001; Fox et al., 2004). Several in vivo rat approaches (Mutanen et al., 1986a; Ros et al., 1998; Ørnsrud and Lorentzen, 2002; Haratake et al., 2007) also reported high Se bioavailability in fish and shellfish (salmon, anchovy, crab, oyster, shrimp, and fish based food). However, low Se bioavailability in tuna and flounder, especially when comparing to that obtained with other foodstuff such as wheat (Alexander et al., 1983; Wen et al., 1997), were reported.

Results from in vitro tests showed that Se contents in the bioaccessible fraction in raw/cooked fish (Crews et al., 1996; Cabañero et al., 2004; Cabañero et al., 2007; Calatayud et al., 2012); and raw shellfish (Calatayud et al., 2012) were dependent on the type of seafood (see Section 6). Higher Se solubility percentages in the bioaccessible fraction in butter clams (98%), swordfish (76–96%), anglerfish (100%), blue whiting (91%), hake and small hake (86–99%), salmon (76–100%) and sole (84%) than those obtained for sardine (53–83%), tuna (50%), chinook salmon (52%), sockeye salmon (50%), and cod (61%) were reported. In addition, high median bioaccessibility values were found for shellfish products (Table 2). SeMet was identified in the GI digests from swordfish, sardine, and tuna (Cabañero et al., 2004; 2007).

In vitro tests also showed that SeMet was the major Se species found in the bioavailable fraction from seafood: whereas, $SeCys_2$ was detected at low concentrations while SeMeSeCys and inorganic Se species (selenite and selenate) were not detected in the dialyzates (Moreda-Piñeiro et al., 2013a). In addition, low Se bioavailability (percentages ranging from 4.0% to 31.3%) was found in fish (plaice, herring, hake, cod, poor cod, anglerfish, Atlantic pomfret, tuna, sardine, Atlantic

mackerel, Atlantic horse mackerel), and mollusk (shrimp, edible cockle, razor shell, variegated scallop, carpet-shell clam) (Shen et al., 1997; Moreda-Piñeiro et al., 2013a; 2013b). Differences between bioaccessible and bioavailable percentages are attributed to the additional dialysis stage in the in vitro bioavailability methods, which improves the simulation of the intestinal digestion and lead therefore to low bioavailable Se percentages. It can also be concluded that total Se is highly bioaccessible but it is not easily dialysed from fish and mollusk. These results are in agreement with those reported by using Caco-2 cells transport and retention simulation approaches for swordfish (Calatayud et al., 2012); i.e., high bioaccessibility percentages (96%) but low bioavailability percentages (Se transport from the food was less than 14%, and cell retention was between 8 and 12%).

Selenium bioavailability in algae and seaweeds

Since algae and seaweeds are naturally Se rich foodstuffs, several in vivo animal (Cases et al., 2001; 2002) and in vitro (Moreda-Piñeiro et al., 2013b; García-Sartal et al., 2013; Laird and Chan, 2013) approaches have been developed (Table 2).

In vivo approaches showed that Se bioavailabilty in *Spirulina* was less effective than selenite and SeMet solutions in restoring Se concentration in the rat liver but not in kidney. Similarly, *Spirulina* was less effective than the other forms of Se in restoring GPx activity, except in plasma and RBC for which no differences were noted among the three Se sources (Cases et al., 2001). However, Se in the retentate fraction of *Spirulina* (resulting from ultra-filtration of the supernatant through a 30 kDa exclusion membrane) was highly bioavailable. The retentate fraction allowed full replenishment of Se concentration in liver and kidney (as did selenite) and GPx activity in liver, kidney, plasma, and erythrocytes (Cases et al., 2002).

A simulated gastric and intestinal digestion/dialysis method was used to assess the Se bioavailability in different raw and cooked edible seaweed (Dulse, Nori, Kombu, Wakame, Sea spaghetti, and Sea lettuce), and canned/cooked seaweed (*Himanthalia elongata* and *Saccorhiza polyschides*) (Moreda-Piñeiro et al., 2013b; García-Sartal et al., 2013). High bioavailability percentages (48–100%) for raw edible seaweed (Moreda-Piñeiro et al., 2013b), and moderate dialyzable percentages (15–61%) for cooked edible seaweed were assessed (García-Sartal et al., 2013). Dialyzability percentages were dependent on the type of seaweed. This can be due to the different seaweed composition (fat, protein, carbohydrates and fiber contents, among other dietary factors) (see Section 6). In general, Wakame followed by Kombu. was the seaweed exhibiting the highest Se bioavailability percentage. Finally, a Se bioaccessibility percentage of 65% was also assessed in laver seaweed (Laird and Chan, 2013).

Selenium species bioavailability in yeast and yeast-based nutritional supplements

Se bioavailability in yeast (Smith and Picciano, 1987; Wang and Lovell, 1997; Yoshida et al., 1999; Fox et al., 2004; Dumont et al., 2004; Mateo et al., 2007; Wang and Xu, 2008; Rider et al., 2009; 2010; Jang et al., 2010; Hall et al., 2012; Briens et al., 2013), Se-fortified yeast (Levander et al., 1983; Bogye et al., 1988; Nève et al., 1988a,b; Alfihan et al., 1991; Xia et al., 1992; Thompson et al., 1993; Alfthan et al., 2000; Larsen et al., 2004; Hinojosa-Reyes et al., 2006a; 2006b; Bügel et al., 2008; Bost and Blouin, 2009; Thiry et al., 2013a; Le and Fotedar, 2014) and yeast-based nutritional supplements (Finley et al., 1999; Hinojosa-Reyes et al., 2006a,2006b; Mahmoud, 2012) have been extensively assessed (Table 3).

In vivo human tests showed that Se levels in plasma and/or whole blood increased significantly after supplementation with yeast and selenized tablets (Levander et al., 1983; Bogye et al., 1988; Nève et al., 1988a, 1988b; Alfihan et al., 1991; Xia et al., 1992; Thompson et al., 1993; Alfthan et al., 2000; Larsen et al., 2004; Bost and Blouin, 2009; Bügel et al., 2008; Mahmoud, 2012), which indicated that Se in yeast was high bioavailable. However, Fox et al. (2004) reported lower Se apparent absorption in yeast (53%) than that achieved in fish (88–90%).

In vivo studies by using rat, catfish, yellowtail kingfish, ewe, trout, boiler chicken and pig as animal models (Smith and Picciano, 1987; Wang and Lovell, 1997; Yoshida et al., 1999; Mateo et al., 2007; Wang and Xu, 2008; Rider et al., 2009; 2010; Jang et al., 2010; Hall et al., 2012; Briens et al., 2013; Le and Fotedar, 2014) also confirmed the high Se availability in yeast. Yeast increased Se status to a similar extent to SeMet (this finding can be attributed to the fact that Se-yeast contains more than 90% of its Se in the form of SeMet). In addition, Se in yeast was almost twice as bioavailable as Se from selenite and selenate for restoration of depleted GPx activity (Smith and Picciano, 1987; Wang and Lovell, 1997; Yoshida et al., 1999; Mateo et al., 2007; Wang and Xu, 2008; Rider et al., 2009; 2010; Jang et al., 2010; Hall et al., 2012; Briens et al., 2013). However, Le and Fotedor, 2014 (Le and Fotedor, 2014) reported no difference in GPx activity of kingfish fed with any supplemented diet (Se-yeast, Se (IV), SeMet, and SeCys), although muscle Se concentration was increased when Se supplementing with SeCys, SeMet or Se-yeast, but not with selenite.

In vitro approaches showed that SeMet was the major compound identified in the GI extracts from yeast (Dumont et al., 2004), selenized yeast (Hinojosa-Reyes et al., 2006a; 2006b, Thiry et al., 2013a), and yeast-based nutritional supplements (Hinojosa-Reyes et al., 2006a; 2006b); whereas, SeOMet was its main degradation product after medium and long-term sample storage (Dumont et al., 2004; Hinojosa-Reyes et al., 2006a; 2006b). Other minor Se compounds were identified as $SeCys_2$, SeMeSeCys, and Se low molecular weight compounds. High bioaccessible percentage (55–89%) was achieved for total Se in yeast. Concerning SeMet, low bioaccessible percentages (26–41%) were observed in yeast and yeast-based nutritional supplements (Dumont et al., 2004; Hinojosa-Reyes et al., 2006a; 2006b). The bioavailable fraction was also assessed by using a Caco-2 cell model. Overall Se bioavailability in the three food supplements was low (7.0, 14 and 1.0% for S-enriched yeast, selenate-based food supplements, and selenite-based food supplements) (Thiry et al., 2013a). Differences among the three types of food supplements could be attributed to the Se species pattern because selenate-food supplements and selenite-food supplements contain exclusively Se(VI) and Se(IV), respectively, but speciation studies in the bioavailable fractions in

selenized yeast showed the presence of SeMet, Se(IV), and SeMeSeCys.

Selenium bioavailability in meat

Several in vivo human and animal tests have been developed for Se bioavailability in beef (Van der Torre et al., 1991; Wen et al., 1997; Hawkes et al., 2003; Finley et al., 2004), pork (Wen et al., 1997; Bügel et al., 2004), chicken (Christensen et al., 1983; Wen et al., 1997), sheep (Butler et al., 1991) and lamb (Wen et al., 1997) (Table 4). Concerning beef, pork, chicken, sheep and lamb, the increase of GPx activity and Se concentration in several tissues was similar to those observed when using a selenite reference diet. However, Se in beef, veal, chicken, pork and lamb was not sufficient to restore liver and muscle Se after 9 weeks of recovery following a 6 weeks period of Se depletion (Wen et al., 1997). Finally, regarding sheep meat, Se levels and GPx activity were found to be higher (more available) when feeding rats with the sheep muscle than when feeding with the liver and hemoglobin (Butler et al., 1991).

Bioaccessibility of Se was studied in meat from chicken (feed with Se-enriched wheat (Govasmark et al., 2010; Brandt-Kjelsen et al., 2012), in un-aged and aged beef (Ramos et al., 2012) and in wild game meat (rabbit, deer, and moose) and wild game organs (deer and moose liver and moose kidney) (Laird and Chan, 2013). The in vitro gastric digestion increased the Se yield to 62–91% (Brandt-Kjelsen et al., 2012; Ramos et al., 2012). The main Se form in beef was SeMet, which is much more bioavailable than other forms such as selenite (Ramos et al., 2012). Finally, higher Se bioaccessibility percentages were assessed in deer and moose organs (52, 62 and 35% for deer liver, moose kidney, and moose liver) than in deer (37%) and moose (29%) meat (Laird and Chan, 2013).

Selenium bioavailability in other foodstuffs

Brazil nuts

Several in vivo (Chansler et al., 1986; Thompson et al., 2008) and in vitro (Dumont et al., 2006b; Gomes da Silva et al., 2013) studies have been carried out to assess the bioavailability of Se in naturally Se rich foodstuffs such as Brazil nuts (Table 4). Based on Se contents and GPx activity restoring in plasma and liver from human and rodents, Se can be considered as fully available in the Brazil nuts (Chansler et al., 1986; Thompson et al., 2008). Similarly, bioaccessibility studies (Dumont et al., 2006b; Gomes da Silva et al., 2013) showed high Se bioaccessible percentages (74%) from Brazil nuts (Gomes da Silva et al., 2013), and $SeCys_2$ and SeMet were the main identified compounds in simulated GI digests (Dumont et al., 2006b).

Milk

Se bioavailability in cow milk (Mutanen et al., 1986b; Shen et al., 1996; Alférez et al., 2003; Barrionuevo et al., 2003; Chen et al., 2004; Muñiz-Naveiro et al., 2006; Zohoori et al., 2009; Alzate et al., 2010), goat milk (Shen et al., 1996; Alférez et al., 2003; Barrionuevo et al., 2003), sheep milk (Shen et al., 1996), human milk (Shen et al., 1996) and milk-based infant formulas (Van Dael et al., 2002) were also assessed. In vivo results showed that Se in milk was highly bioavailable (Table 4). In addition, Se bioavailability in milk from cows fed with selenite-barley was higher than Se bioavailability in cows fed with selenite when the plasma Se level was used as the response criterion. However, Se biovailability was not different when plasma or liver GPx activities were used as the response criteria (Mutanen et al., 1986b). The apparent absorption of Se was higher for goat milk than for cow milk (Alférez et al., 2003; Barrionuevo et al., 2003). These findings showed that milk can be a readily available source of dietary Se.

Simulated in vitro GI digestion (Zohoori et al., 2009; Alzate et al., 2010) and in vitro GI digestion plus dialysis procedure (Shen et al., 1996; Muñiz-Naveiro et al., 2006) showed high bioaccessible percentages (76%) after GI digestion (Alzate et al., 2010), but low bioavailable ratios (2–11%) (Shen et al., 1996; Muñiz-Naveiro et al., 2006). As previously discussed for seafood, it can be concluded that Se is highly bioaccessible but it is not easily bioavailable in milk. Dialysate percentages in human milk (11.1%) were significantly higher than those observed for cow (6.8%), goat (6.2%), and sheep (~2%) milk. On the other hand, Se bioavailability increased when removing the milk fat fraction (15, 10, 12, and 2.4% for human, cow, goat, and sheep milk, respectively) (Shen et al., 1996). $SeCys_2$ and SeMetSeCys were the most abundant compounds in the bioaccessible fraction in Se-fortified fermented milk. Two unknown Se compounds were also detected in the bioaccessible fraction (Alzate et al., 2010).

Eggs

In vivo human results showed a high Se bioavailability in intrinsically labeled-Se eggs (Sirichakwal et al., 1985). In vitro tests were also applied to selenized eggs (eggs fortified with health-promoting additives, including Se, and eggs obtained from hens fed with organic Se feed supplement) (Lipiec et al., 2010); but bioaccessibility percentage data were not given in the study. On the other hand, the forms of Se initially present were degraded under the gastrointestinal conditions to low molecular weight peptides, amino-acids (SeMet and SeCys), and inorganic Se.

Selenium bioavailability in chemically pure forms

Se chemically pure forms (Se(IV), Se(VI) and SeMet mainly) have been also used to assess Se bioavailability by several in vivo human (Thompson and Stewart, 1974; Griffiths et al., 1976; Thompson et al., 1978; 1982; 1988; 1993; Levander et al., 1983; Christensen et al., 1983; Kasper et al., 1984; Mutanen, 1986; Thompson and Robinson, 1986; Nève et al., 1988a, 1988b; Clausen et al., 1989; Martin et al., 1989; Xia et al., 1989; 1992; Alfihan et al., 1991; 2000), in-vivo animal (Thompson et al., 1975a; 1975b; Gabrielsen and Opstvedt, 1980; Mutanen and Mykkänen, 1984; Smith and Picciano, 1987; Waschulewski and Sunde, 1988; Whanger and Butler, 1988; Ip and Lisk, 1993; Shi and Spallholz, 1994a; Serra et al., 1997; Wang and Lovell, 1997; Ros et al., 1998; Cases et al., 2001; Peng et al., 2007; Wang et al., 2007; Jaramillo et al., 2009; Küçükbay et al., 2009; Zhou et al., 2009; Rider et al., 2010; Díaz-Castro et al., 2011; Hall et al., 2012; Briens et al., 2013; Le and Fotedor, 2014) and also in vitro tests (Wang and Fu, 2012; Thiry et al., 2013b; Pick et al., 2013) (Table 5).

In vivo human results showed that supplementation with selenite increased plasma Se concentration (plasma Se increased within the 15–100% range) (Levander et al., 1983; Mutanen, 1986; Thompson et al., 1988; Alfihan et al., 1991). Plasma GPx activity was also increased (19–30%) (Thompson et al., 1982; 1988; Alfihan et al., 1991). Moderate apparent Se absorption was also obtained (apparent absorption of Se was between 56 and 85%) (Thompson and Stewart, 1974; Thompson et al., 1978; Thompson and Robinson, 1986). Supplementation with selenate increased significantly plasma Se levels (58–530%) and GPx activity (16–300%) (Xia et al., 1989; 1992; Alfthan et al., 2000). However, Alfihan et al (Alfihan et al., 1991) reported that supplementation with selenate did not increase plasma Se above placebo while plasma GPx activity reached at 30%. Apparent Se absorption percentages were in the 35–94% range (Christensen et al., 1983; Thompson and Robinson, 1986; Xia et al., 1992). Supplementation with SeMet also increased the plasma Se level (40–196%) (Thompson et al., 1982; Nève et al., 1988a,b), plasma GPx activity (29%) (Thompson et al., 1982) and the apparent absorption (22–97%) (Griffiths et al., 1976; Thompson et al., 1978). Se levels tended to a plateau after selenite supplementation, while they continued to rise as long as dosing continued when supplementing with SeMet (Thompson et al., 1982; Kasper et al., 1984).

Despite the high Se bioavailability in chemical pure forms of Se, Se supplementation with Brazil nuts (Thompson et al., 2008) and Se-fortified yeast (Alfthan et al., 2000) showed better results in terms of the response of plasma Se concentration and GPx activity. On the other hand, although SeMet and Se-fortified yeast were equally effective in raising GPx activity, Se-yeast provided a longer lasting body pool of Se (Xia et al., 1992). In addition, the mean whole blood Se concentration obtained with synthetic SeMet was lower than that obtained with the same daily level of supplementation by Se yeast, which suggests a different degree of utilization of the yeast-based and the synthetic supplements (Larsen et al., 2004).

In vivo animal tests showed that organic chemically pure Se species were more available than inorganic species. Higher Se absorption, retention, and accumulation ratios in blood and tissues were found when SeMet was administered than those observed after inorganic species (selenite and selenate) administration. Absorption, accumulation and retention ratios increased with the dose of Se administered. On the contrary, organic Se generated a lower GPx activity than that achieved when using inorganic Se. (Thompson et al., 1975a; 1975b; Mutanen and Mykkänen, 1984; Smith and Picciano, 1987; Whanger and Butler, 1988; Serra et al., 1997; Ros et al., 1998; Küçükbay et al., 2009).

In vitro models of the intestinal barrier (Caco-2 cell model) (Zeng et al., 2008; Wang and Fu, 2012; Thiry et al., 2013b, Pick et al., 2013) also confirmed in vivo conclusions; Se absorption efficiency was shown to be dependent on the Se species (56 ± 4, 46 ± 2, 33 ± 2, and 12 ± 1% for SeMet, SeMeSeCys, Se(VI), and Se(IV), respectively after 3 h (Thiry et al., 2013b); and ~8.0, ~4.0, and ~1.0% for seleno nanoparticles (nano-Se), SeMet, and Se(IV), respectively, after 2 h (Wang and Fu, 2012). Higher transport efficiencies of organic Se and nano-Se than those for sodium selenite and selenate were observed (Wang and Fu, 2012; Thiry et al., 2013b, Pick et al., 2013). Also, no transport for selenite from apical to basolateral side was observed (Pick et al., 2013). These findings showed that transcellular might be the main pathway for SeMet and nano-Se and suggest that interactions between Se species could change the transport behavior (Pick et al., 2013). In addition, the uptake efficiency of Se in the Caco-2 cells varied with the chemical form, which might be associated with differences in Se transport and uptake. Se uptake resulted in a twofold lower retention for selenate than for selenite. The uptake of SeMet and SeMetSeCys was 6- to 20-fold higher than for inorganic species, whereas the SeMet content was threefold higher than SeMetSeCys content (Pick et al., 2013). Therefore, a higher accumulation for the organic species SeMet and SeMetSeCys compared to selenite and selenate was assessed. Se(IV) seemed to be of particularly low nutritional value, as most of Se(IV) was directly excreted (Thiry et al., 2013b). Finally, SeMet and MeSeCys transport was significantly inhibited by their respective sulfur analogues methionine and methylcysteine, which suggests a common transport system for both kinds of compounds (Thiry et al., 2013b). Caco-2 cell models also showed that no significant differences were observed in GPx activities when using standards (Se(IV), SeMet, and SeMeSeCys) and seleno-broccoli extracts, and a period of 72 h was required to obtain high Se-induced GPx activity in Se-deficient Caco-2 cells (Zeng et al., 2008).

Effect of food processing on selenium bioavailability

Foodstuffs are usually processed (cooking, frying, boiling, baking, grilling; ripening; ageing, or fermentation treatments) to improve the palatability of the product. Food processing might affect the digestive enzymes efficiency. In addition, major constituents of foodstuff (i.e., protein, fat, or carbohydrate content) and/or nutrient (i.e., Se) degradation or transformation could also occur, which affect nutrient bioavailability. During food processing nutrient could also be removed from the foodstuff (i.e., nutrient could be releasing into cooking water).

During heat and ripening processing, loss of water and protein degradation occurs. Protein degradation can improve protein digestibility, which will facilitate Se release and enhance the bioaccessibility ratio (Cabañero et al., 2004). Therefore, Se bioaccessible percentages in cooked tuna [microwave oven at high power (100%, 650 W) for 4 min] were slightly higher than those found in uncooked tuna (Cabañero et al., 2004). However, in vivo rat tests showed that no differences were found among Se bioavailability of raw, precooked, and canned tuna (Alexander et al., 1983). There was a significant Se content increase in liver, kidney, blood, and muscle of rats fed with canned tuna than those found in rats fed with the same levels of Se in raw and precooked tuna. Although Se levels were different when feeding with cooked, raw, precooked, and canned tuna, the similar GPx activity values measured did not allow conclusive results regarding Se bioavailability in cooked and uncooked tuna (Alexander et al., 1983). Similarly, in vivo human tests showed that Se bioavailability in cooked trout fish (87.8%) and salted trout fish (90.4%) (Fox et al., 2004) was not significantly different. Milk fermentation procedure (with lactic acid bacteria such as *Lactobacillus, Lactococcus*, and *Leuconostoc*) also improved the digestibility of milk proteins, which

increased Se availability in milk (Shen et al., 1996). The bioavailability percentage in ripening herring (maatjesherring: herring ripened for a couple of days in oak barrels in a salty solution) was higher (31.3%) than those found in unripened herring (21.3%) (Shen et al., 1997). On contrary, in vivo rat models showed higher bioavailability in raw salmon than for cured salmon. These results suggested that curing salmon altered the utilization of Se. Further work is necessary to clarify the effect of processing on fish fillets with regard to the bioavailability of Se (Ørnsrud and Lorentzen, 2002).

Regarding in vivo human tests, plasma Se concentrations increased to 59% and 15% after supplementation with Se-fortified wheat with process-fortified Se biscuits, respectively. This difference may be due to the presence of SeOMet in process-fortified biscuits. SeMet in fortified wheat is degradated to SeOMet after wheat processing (Kirby et al., 2008). Thus, the low bioavailability of SeOMet could explain the low Se bioavailability in process-fortified Se biscuits.

During cooking, Se could also be released from foodstuff into the cooking water. García-Sartal et al. (García-Sartal et al., 2013) achieved low dialyzability percentages in cooked (following the manufacturer's recommendations, i.e., seaweed were cooked in 300 mL of de-ionised water for 5–60 min) Dulse, Nori, Kombu, Wakame, Sea spaghetti, and Sea lettuce. The dialyzability percentage obtained for canned seaweed (mixture of Sea spaghetti and *Saccorhiza polyschides*) was higher (~100%) than the bioavailability percentages obtained for raw Sea spaghetti (~80%) (Moreda-Piñeiro et al., 2013b).

Aging affected negatively Se bioaccessibility in beef (Ramos et al., 2012). Natural enzymes present in beef break down the fibrous tissues during aging time; and, in addition, moisture is partially lost. Se bioaccessibility ratios ranged between 75 and 91% in un-aged beef. After aging, Se bioaccessibility was significantly lowered (65 to 78%). Finally, processes to isolate protein from soybean and the tofu production do not modified the high Se bioavailability in soybean, isolate protein, and tofu (Yan et al., 2010).

Effect of sample composition on selenium bioavailability

Major (carbohydrate, proteins, dietary fiber, and fat content) and minor (oligoelements/toxic metals and vitamins) food components can influence nutrient bioavailability in foods. Table 1–4 showed that Se bioavailability depends on the foodstuff. As an example, high Se absorption in pork (94%) (Bügel et al., 2004) and milk (73–97%) (Van Dael et al., 2002) by in vivo tests reported; whereas, low values in chicken meat (71–72%) (Christensen et al., 1983) and wheat (62%) (Levander et al., 1981) were found.

Effect of carbohydrate content

Findings showed that Se bioavailability increased with the carbohydrate content in fish, shellfish, and seaweed (Moreda-Piñeiro et al., 2013b) due to carbohydrate is capable of forming micelles, which can enhance the partition of hydrophobic molecules in the aqueous solution (Yu et al., 2010; Moreda-Piñeiro et al., 2013b). On the contrary, Se-containing polysaccharides from mushrooms could not be solubilized after GI digestion due to Se-enriched mycelia is associated with chitin-containing structures in cell walls (Bhatia et al., 2013). The formation of Se-containing polysaccharides might explain the low Se bioavailability found in mushroom. Similar conclusions have been addressed from mushroom after in vivo human tests (Mutanen, 1986), and from Brazil nuts and mushroom after in vivo animal assays (Chansler et al., 1986).

Effect of protein content

Although there are few data about the effect of protein content on Se bioavailability, some studies have indicated that Se bioavailability decreases when increasing the protein content in foodstuff such as fish, shellfish and seaweed samples (Moreda-Piñeiro et al., 2013b). This fact has been attributed to the effect caused by the ionic strength. Proteins are hydrolyzed to amino acids during the GI digestion. Most of these amino acids are soluble and carry positive or negative charges at the physiological pH used during the digestion. These amino acids exist therefore as a sort of salts which increases the ionic strength of the aqueous phase (GI digest). The increased ionic strength results in a lower solubility of Se species (the "salting-out" effect) in the aqueous phase, and hence lower bioaccessibility is observed (Yu et al., 2010; Moreda-Piñeiro et al., 2013b).

Effect of fat content

The effect of dietary fat on the availability of Se was investigated in chicks fed either 4 or 20% butter, olive oil, rape oil, corn oil, and sunflower oil in the diet (Mutanen and Mykkänen, 1984). Plasma GPx levels were increased with increasing proportions of the polyunsaturated fatty acids in the diet. The absorption of Se tended to be lower in chicks fed with corn oil and sunflower oil than that observed in animals receiving butter in their diet. On the contrary, the type of dietary fat did not appear to affect the absorption of orally administered selenite (Mutanen and Mykkänen, 1984). In vitro tests suggested that Se bioavailability in fish and shellfish is not affected by the fat content (Moreda-Piñeiro et al., 2013b). Se species released from the food matrix during the GI digestion are not expected to be bound to the fat due to Se species are poorly lipophilic molecules (Moreda-Piñeiro et al., 2013b). On the contrary, fat removal from milk (human, cow, goat and sheep milk) increased significantly Se bioavailability (Shen et al., 1996; Zohoori et al., 2009). Se mainly exists in protein-bound forms in milk, and almost 80% of Se in cow's milk is associated with casein. It has been suggested that protein digestibility is the main determinant factor for Se bioavailability in milk. Thus, the high Se bioavailability in skimmed milk might be due to the better protein digestibility of skimmed milk (Zohoori et al., 2009). However, other studies showed that the resulting differences in fatty acid composition on human diets, with a higher content of polyunsaturated fatty acids (pork meat), appeared not to affect Se absorption or retention (Bügel et al., 2004).

Effect of fibre content

Fiber (soluble fibre or non-soluble fibre) content seems to differently affect Se bioavailability. Findings when analyzing seaweed have demonstrated that Se bioavailability was increased with increased soluble fibre concentrations (Moreda-Piñeiro et al., 2013b). This result agrees with those reported by Vitali et al. (2008), which have attributed high mineral bioavailability ratios in vegetables with high soluble dietary fiber. On the other hand, in vivo tests with rats showed that non-digestible fiber content in wheat and bran decreased Se bioavailability (Reeves et al., 2007). The low solubility of this fiber likely effectively encapsulates cellular proteins within intact cells in the bran fraction and renders them unavailable for digestion in the upper gut. Because most of Se is incorporated primarily into these proteins, Se itself becomes unavailable.

Effect of oligoelements, toxic metals, sulfur, vitamins, and ethanol

The effect of oligoelements, toxic metals on Se bioavailability is not always easy to understand. The effects are often contradictories and the major components of foodstuff play an important role. Se retention was increased in iron deficient diets (Díaz-Castro et al., 2011). This fact contributes to keep the enzymatic antioxidant activity of GPx in normal levels and to store the increase of Se in the organs, especially in kidney. Similarly, Zn had an antagonistic effect on Se absorption in Se-depleted rats (House and Welch, 1989). The antagonistic effect occurred between natural forms of Zn and Se at concentrations potentially encountered in wheat grain. Hg (Ralston et al., 2008) tends to reduce Se bioavailability in in vivo studies with rats. Therefore, selenoenzymes may be the molecular target of Hg toxicity. Regardless of the high Se bioavailability assessed in fish and seafood (Levander et al., 1981; Fox et al., 2004) other in vivo human tests also confirmed the low Se plasma/whole blood, and low GPx activity increase in Se-rich fish (trout, mackerel, wolf-fish, rosefish, and haddock) due to the presence of high levels of Hg and As (Meltzer et al., 1993). Se could not be well absorbed due to interactions between Hg/As and Se. Hg/As and Se interactions occurred in forms like Se compounds or seleno–sulfur bonds (interrupting synthesis of Se-dependent enzymes), which could explain the poor Se bioavailability in seafood (Meltzer et al., 1993). However, Hagmar et al. (Hagmar et al., 1998) reported that Hg content in erythrocytes was correlated with both Se in plasma, SeP and GPx, which minimized the antagonistic effect of Hg. On the other hand, the low bioavailability of Se in mushroom could be due to the high intake of Cd and Hg content in food. Cd and Hg would have Se complexes into a non-available form already in the gastrointestinal tract, decreasing the GPx activity (Mutanen, 1986).

Dietary sulphur (methionine, Met) (Waschulewski and Sunde, 1988) tend to reduce Se bioavailability in in vivo studies with rats. Sulfur competes with Se for absorption and utilization. The high Met and polyunsaturated fatty acids content in Se rich fish reduces deposition of Met-bound Se in tissues and increased the oxidant stress, thereby compromising Se status by increasing the turnover of Se through GPx (Meltzer et al., 1993). However, other studies showed that the resulting differences in fatty acid composition of the human diets, with a higher content of polyunsaturated fatty acids appeared not to affect Se absorption or retention (Bügel et al., 2004).

The potential interaction between Se and vitamin E (in juvenile hybrid striped bass) and between Se and fluoride (after in vitro bioaccessibility of Se in pasteurized skimmed and whole milk samples) was also evaluated. However, there was no significant effect of vitamin E on Se availability (Jaramillo et al., 2009), and a clear relationship between Se bioaccessibility and fluoride concentrations was not found (Zohoori et al., 2009).

Finally, the effect of ethanol consumption affects Se bioavailability in rats and in their progeny (Ojeda et al., 2009). Results showed that ethanol decreased Se retention, affecting the tissue Se deposits. In addition, their progeny's weight and oxidation balance was committed. The effects of ethanol were caused by a direct alcohol-generated oxidation action (Ojeda et al., 2009).

Conclusions

Several in vivo and in vitro methods have been developed and applied to assess bioavailability of Se and Se species from raw and processed foodstuffs. Despite the differences in Se bioavailability between studies are difficult to directly compare due to the lack of a generally accepted in vivo and in vitro extraction protocols; several conclusions can be achieved. Se concentration and Se species content in a foodstuff critically influence Se bioavailability and bioactivity to humans and animals. Although organic and inorganic forms of Se were equally effective in raising blood GPx activity, organic forms, especially SeMet, are more capable of increasing blood Se levels than are the inorganic selenates and selenites. In vitro approaches also confirmed the high Se bioavailability from organic forms.

The bioavailability of Se and Se species are also affected by:

(1) foodstuff-matrix composition: major (carbohydrate, fat, protein and fibre) and minor components (oligoelements and toxic metals) of foodstuff determine Se bioavailability rates;
(2) foodstuffs processing: cooking, boiling, ripening; ageing or fermentation treatments could enhancement or decrease Se bioavailability;
(3) experimental conditions: the selection of examined group (healthy subjects with a well defined basal Se status and pro- and antioxidant status (smoking, alcohol consumption, vitamin E status, etc.)) in in vivo humans approaches; the selection of animal model in in vivo animals approaches; or the selection of GI conditions (gastric plus intestinal digestion, Caco-2 cells model or SHIME model) in in vitro tests could determines the results. As a consequence of the different models used to assess bioavailability, future development in this area will focus on assessing an international standardized protocol for assessment of in vivo and in vitro approaches. The development of certified reference materials (together with laboratory guidelines for their use) could provide appropriate materials for quality control procedures in all laboratories.

In addition, most studies emphasize Se bioavailability in individual foodstuff or discrete Se compounds. However, because of the many possible interactions it is important to

study the effects of Se within the total diet. Finally, important Se species degradation and transformation could occur during the in vitro process (i.e., conversion of γ-glu-Se-MeSeCys to Se-MeSeCys, conversion of SeMet to SeOMet, organic Se species (MeSeCys, $SeCys_2$, and SeMet) degradation to inorganic Se).

Funding

The authors wish to thank Xunta de Galicia (Programa de Consolidación y Estructuración de Unidades de Investigación Competitivas 2013–2016, ref: GRC2013-047 and the Ministerio de Ciencia y Tecnología (Project number AGL-2006-11034) for financial support.

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