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Review

Current knowledge in species-related bioavailability of selenium in food

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ABSTRACT

Selenium is an essential trace element that has raised interest because of its antioxidant and anticancer properties. The beneficial or toxic effect of Se is not only dose-dependent, but also relates to the chemical form of the element and its bioavailability. In this review, recently published data is summarised concerning both Se speciation and Se relative bioavailability in various foodstuffs. In addition, Se bioavailability is discussed in relation to the species-dependent metabolism in humans. In this way, the understanding of the potential health impact of Se species in commonly consumed food is aimed to be improved. It is strongly suggested on the basis of a higher retention and a lower toxicity, that organic Se (especially SeMet, the major species in food) is more recommendable than inorganic Se in the frame of a balanced diet. Further research is however desirable concerning the characterisation of unidentified Se species and determination of their health effects.

Contents

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1. Introduction

Like other trace elements, selenium (Se) is a natural constituent of the earth's crust. After its discovery by Berzelius in 1817, this element became firstly known for its toxic properties. Adverse effects were reported in seleniferous areas of the world such as parts of Canada, Ireland, western USA or some zones of China (Navarro-Alarcon & Cabrera-Vique, 2008). Symptoms observed in poisoned cattle grazing on Se-rich vegetation include loss of vitality, loss of hair, elongated and disfigured hooves, degeneration of internal organs (Fishbein, 1991) and death in the worse cases (Dumont, Vanhaecke, & Cornelis, 2006). Selenium intoxication can also happen in humans, even though it is not very common. The disease is known as "selenosis", and causes symptoms similar to those observed in cattle, i.e. hair and fingernails brittleness, skin and liver damage, and neurotoxicity at higher levels (Lederer, 1986).

Schwarz and Foltz (1957) suggested for the first time that Se might be an essential element despite its toxic properties at high concentrations. Rotruck et al. (1973) were able to demonstrate that Se is part of the active centre of glutathione peroxidase (GPx), an enzyme whose role is to protect tissues against oxidative stress by catalysing the reduction of peroxides responsible of various cellular damages (Zhang, 2009). During the following years, several additional proteins and enzymes were recognised as "selenoproteins", all of them containing Se exclusively as selenocysteine (SeCys) residue. At least 25 different selenoproteins and a variety of subsequent isoforms exist in the human body (Gromer, Eubel, Lee, & Jacob, 2005). Among these, glutathione peroxidases (GP1–4), thioredoxin reductases (TR1–3), desiodases and selenoprotein-P serve particularly important functions. Similarly to GPx, TRx regulate the cellular redox balance by catalysing the reduction of oxidised compounds and playing a role in apoptosis (Gromer et al., 2005). Desiodases are involved in the thyroid function by controlling triiodothyroine (T_3) and thyroxine (T_4) levels (Tapiero, Townsend, & Tew, 2003). Selenoprotein P (SeP) is the most abundant selenoprotein in plasma and probably acts as a Se transporter between the liver and other organs such as the brain, kidneys and testes (Burk, Hill, Motley, Austin, & Norsworthy, 2006; Hill et al., 2007). The selenoprotein pool also includes minor molecules whose role is less clear but which seem to have structural functions, notably in sperm and muscles (Alexander, 2007). The essentiality of Se as a trace nutrient for humans and animals is nowadays widely recognised and is illustrated by the occurrence of specific diseases in some areas with low environmental Se levels. Keshan disease is a well known example of an endemic cardiomyopathy that has been observed in children, adolescents and pregnant women in the Keshan region of China, a place where Se levels in soil and food are extremely low (Lederer, 1986). Similarly, Kashin-Beck disease is an osteoarthropathy reported to occur in Se-deficient populations (Navarro-Alarcon & Lopez-Martinez, 2000). These two pathologies have been shown to disappear with an appropriate Se supplementation (Fairweather-Tait, 1997). Moreover, when consumed at supranutritional level, Se has revealed anticancer properties in several epidemiological studies and experimental and clinical trials (for a review see Combs and Gray (1998)). In particular, it has been suggested that an intake of 200 μg Se day^{-1} could prevent the incidence and mortality of carcinomas in several sites of the body (Clark et al., 1996). The anti-cancer action of supranutritional Se intake has not yet been utterly explained, however it is likely that in this case Se plays a pro-oxidative role instead of anti-oxidative. At this level of intake, activity of antioxidant selenoproteins is already at its maximum (Combs & Gray, 1998) and the excess of Se acts as oxidising agent, able to induce apoptosis of cancerous cells (Brozmanova, Manikova, Vlckova, & Chovanec, 2010; Letavayová, Vlcková, & Brozmanová, 2006). This mechanism that normally induces oxidative selenotoxicity to healthy people, becomes thus beneficial in particular cases of cancer. Additionally, specific anticarcinogenic properties have been attributed to a particular selenocompound, methylselenol, which is able to directly affect the metabolism of cancerous cells (Ip, Thompson, Zhu, & Ganther, 2000). Besides antioxidant and anti-cancer action, beneficial effects of Se include prevention of cardiovascular diseases (Rayman, 2000), detoxification of heavy metals (Navarro-Alarcon & Lopez-Martinez, 2000; Rayman, 2000), and involvement in cerebral functions, reproduction (Renko et al., 2008) and immune system response (Kiremidjian-Schumacher, Roy, Wishe, Cohen, & Stotzky, 1992).

The particularity of Se is that the margin between toxicity and deficiency is very narrow. According to the US Food and Nutrition Board, the recommended dietary allowance has been set at 55 μg Se day^{-1} for both sexes, whereas the tolerable upper intake level is advised at 400 μg Se day^{-1} in the USA (Goldhaber, 2003; Pedrero & Madrid, 2009) and 300 μg Se day^{-1} in Europe (SCF, 2000). Dietary Se intake of people is highly influenced by Se content of the local soil, which is heterogeneous throughout the world. In Europe, Se intake ranges approximately from 28 to 70 μg Se day^{-1} (Navarro-Alarcon & Lopez-Martinez, 2000; Rayman, 2000) while in the US, the daily intake almost reaches 100 μg (Rayman, 2000). Due to the essentiality of Se and its reported beneficial effects, nutritionists recommend more and more to increase Se intake, especially in regions where environmental Se levels are low. This supplementation can be achieved by different means: by addition of food supplements to the usual diet (Dumont, Vanhaecke, et al., 2006), by consumption of food that is naturally rich in selenium (e.g. brazil nuts) (Dumont, De Pauw, Vanhaecke, & Cornelis, 2006; Thompson, Chisholm, McLachlan, & Campbell, 2008) or by consumption of food that has been previously enriched in Se, such as vegetables fertilised with inorganic Se or cattle fed with Se supplemented feed (Jiakui & Xiaolong, 2004; Varo, Alfthan, Ekholm, Aro, & Koivistoinen, 1988). The problem is that the recommended values for consumption or supplementation of Se do not take into account the fact that Se can be present in food under different chemicals forms, organic or inorganic, and that these forms do not exert the same effect on the organism (Amoako, Uden, & Tyson, 2009; Meltzer, Norheim, Bibow, Myhre, & Holm, 1990). Some Se species have been reported to be particularly beneficial (e.g. anticancer action of methylselenol) while some others show no effect or have toxic properties (Tiwary, Stegelmeier, Panter, James, & Hall, 2006). This "uncorrelation" between Se intake and Se health effects may be explained, at least partially, by differences in bioavailability of distinct Se species (Meltzer et al., 1990). For this reason, it is not sufficient to recommend a healthy Se intake on the basis of the total Se content of a category of food or food supplement, but attention must also be given to the speciation of Se, which is determinant for its action on the body. In order to allow an efficient Se supply to Se-deficient individuals or populations, an in-depth insight is needed into the bioavailability and bioactivity of different chemical Se species and, correspondingly, into the Se species composition of distinct food sources. In this context, the present paper aims to provide a summary of existing information about Se speciation and Se bioavailability in food.

2. Selenium speciation

2.1. General points

Speciation refers to the physical and chemical form of an element, namely its oxidation state, stoichiometry or the possible

presence of various ligands (Reeder, Schoonen, & Lanzirotti, 2006). Despite the analytical advances made in this field of analysis during the last decades, there is still a relatively limited number of studies dealing with the quantification of Se species in food. Moreover, existing studies usually focus exclusively on the few most abundant and easily measurable species, even though dozens of different selenocompounds can occur in biological tissues (for a list, see, for example, B'Hymer and Caruso (2006)). Best known species are two oxyanions, selenite (SeO_3^{2-} or Se(IV)) and selenate (SeO_4^{2-} or Se(VI)), and some organic species, selenomethionine (SeMet), selenocysteine (SeCys) and the methylated form of the latter: methylselenocysteine (MeSeCys). SeMet and SeCys are analogues of sulphur aminoacids methionine (Met) and cysteine (Cys) with an atom of Se replacing the atom of S, these two elements having similar physical and chemical properties (Johansson, Gafvelin, & Arner, 2005).

2.2. Methods of extraction and speciation analysis

The scarcity of the data concerning Se species content in food can be explained by the complexity of the methods allowing their detection and quantification, especially when looking for low concentrations (often in the range of the ng g^{-1}). Over the past few years, powerful analytic devices have been developed to make speciation of Se possible. However, this kind of analysis remains difficult to be conducted in routine fashion, which is still the case today.

A difficulty to overcome is the total extraction of Se from the food matrix, without affecting its original speciation. Due to the lack of reference materials certified for Se species (at the moment the only existing reference material is a selenised yeast certified for SeMet (Selm-1, INMS-NRCC)), which otherwise could be added during the extraction procedure and consecutively used for quality control during the measurements, the presence/absence of speciation changes during the extraction process cannot be shown easily. To assess the integrity of other species during extraction, spiking experiments, followed by a check of the recovery, are probably the best alternative. Among Se species, SeCys is particularly difficult to be extracted because its selenol group is so reactive that it can hardly exist under its free form (Suzuki, 2005). The fact that, in addition, there is no commercially available standard for this compound makes that SeCys is not often analysed. On the contrary, selenocystine (SeCys2), a diselenide oxidation product of SeCys, is a well more stable molecule and is regularly measured, without being a correct estimation of SeCys quantities as SeCys2 is not necessarily the only product of SeCys reactions. Enzymatic extraction procedures are usually chosen to extract Se from food matrices. Thanks to the soft conditions of the enzymatic reactions (37 °C; pH 7.5), this method minimises the risk of species transformation while allowing high extraction efficiencies. As most of the time Se is trapped into proteins – often as SeMet nonspecifically incorporated in place of methionine – extraction is typically made with proteolytic enzymes, sometimes combined with ultra-sounds (Pedrero, Madrid, & Camara, 2006; Siwek, Bari Noubar, Bergmann, Niemeyer, & Galunsky, 2006; Vale, Rial-Otero, Mota, Fonseca, & Capelo, 2008) or micro-wave energy (Peachey, McCarthy, & Goenaga-Infante, 2008; Reyes et al., 2009) to shorten the extraction time. For selenocompounds that are not incorporated into proteins, such as MeSeCys, γ-glut-MeSeCys or free inorganic Se, aqueous extraction methods (with eventual addition of an acid or a surfactant) can also be efficient (Montes-Bayon, Molet, Gonzalez, & Sanz-Medel, 2006).

Following extraction, Se species must be unambiguously separated, identified and correctly quantified. Separation process is typically made by chromatography, which allows separation of compounds according to their differential affinity and interactions with a mobile phase and a stationary phase. Owing to the fact that most of the selenocompounds of interest are non-volatile, liquid chromatography (HPLC – high performance liquid chromatography) is usually practiced. Gas chromatography (GC) is nevertheless possible for the few volatile selenocompounds (methylselenol, dimethylselenide, dimethyldimethyldiselenide), or with a previous derivatisation step (B'Hymer & Caruso, 2006). The HPLC separation mode is dependent on the species of interest and the desired degree of selectivity. Size exclusion chromatography (SEC) separates molecules according to their size and their shape. The resolution of SEC is generally too poor to allow discrimination of Se species with a molecular weight inferior to 10 kDa, which makes this technique more suitable as a purification step preceding a complementary chromatographic separation in a multidimensional scheme (Bierla et al., 2008; Wrobel et al., 2004). Ion (anion or cation) exchange chromatography (IEC) is based on the interactions of charged species with an oppositely charged stationary phase. It is particularly efficient for separation of inorganic species that are often charged or easily ionisable (B'Hymer & Caruso, 2006), but it is also customarily used for the analysis of some organo-Se compounds such as selenoamino acids which, depending on the pH of the mobile phase, can be present as cations, anions or zwitterions (Stadlober, Sager, & Irgolic, 2001). Reverse-phase chromatography (RPC) allows separation of compounds according to their polarity and their corresponding affinity with a non-polar stationary phase (C8 and C18) usually coated with an ion-pairing agent added to the relatively polar mobile phase. This method is appropriate for separation of many organic compounds, charged or not, but present the inconvenient to necessitate considerable amounts of organic solvent in mobile phases, which can destabilise and alter ionisation ability of the argon plasma of the ICP-MS (B'Hymer & Caruso, 2006; Bird et al., 1997; Chen, Wang, Mallavarapu, & Naidu, 2008; Szpunar, 2000). Unfortunately, whatever the kind of separation, resolution of Se species in food samples remains quite low and a single column is often not able to clearly distinguish more than three or four species in a complex matrix. To quantify more species, or to validate a result obtained with another column, it becomes necessary to combine successively two or more kinds of separation (Szpunar, 2000).

Finally, to identify and quantify the separated species, hyphenated techniques have been developed based on the coupling of the chromatography device with a particularly sensitive detector. Today, ICP-MS (inductive coupled plasma-mass spectrometer) is a very powerful detector to demonstrate the presence of an element, well more sensitive than other detection methods such as AAS (atomic absorption spectrometry) or ICP-OES (optical emission spectrometry) (B'Hymer & Caruso, 2006). The ICP-MS device is easily coupled to the HPLC system by simply connecting the end of the chromatography column to the ICP-MS nebuliser with PEEK tubing. ICP-MS generates a plasma, typically made of argon (Ar), that leads to the ionisation of the nebulised sample. The identification of the element is made according to the "mass/charge" ratio of the ions. As Se has six different isotopes ($Se^{74, 76, 77, 78, 80, 82}$), the analyst must choose the most appropriate mass to measure the element. This choice depends upon several parameters: the isotopic abundance, the detector sensitivity and the spectral interferences generated by the formation of polyatomic ions in the plasma of Ar. The less sensitive an ICP-MS is, the more abundant the analysed isotope must be to allow a correct quantification. Se^{80} is the most abundant isotope of the element (49.6%) but it is not likely chosen because its signal overlaps with a huge interference of the polyatomic ion $^{40}Ar^{40}Ar^{+}$. For this reason, despite their lower abundance, Se^{78} (23.5%), Se^{76} (9.4%), Se^{82} (9.2%) or Se^{77} (7.6%) are often preferred for analysis (B'Hymer & Caruso, 2006; May & Wiedmeyer, 1998). To a certain extent, polyatomic interferences can be eliminated with an appropriate collision/reaction cell

(CRC) or interface (CRI) which injects post plasma a specific gas into the ionic beam (Paucot, 2006). In collision devices, molecules of this gas (e.g. He, Xe) enter in collision with the interferents and split them into masses that do not impair the analysis of the mass of interest. In reaction devices, the gas (e.g. H_2, NH_3, CH_4, O_2) chemically reacts with the interferent or the analyte itself to modify its mass or its charge (Paucot, 2006). The major limitation of HPLC-ICP-MS is that the system only allows measurement of species for which a standard exists, and it is therefore not applicable to minor or unknown Se species. In this case, ESI-MS (electrospray ionisation mass spectrometry) can represent a valuable alternative. By opposition to ICP-MS whose plasma completely destroys the molecular information, the soft mode of ionisation of ESI-MS permit the conservation of Se species for analysis and the exact molecular mass of the ion can be used to identify this species (Casiot et al., 1999; Goenaga-Infante et al., 2004). In addition, with ESI tandem MS mode, fragmentation patterns can be obtained to get information about the structure of the molecule and isotopic composition can be determined (Casiot et al., 1999). The problem is that ESI-MS sensitivity is well inferior (up to 100 times) to ICP-MS sensitivity, mostly because of the matrix load of the samples. This technique is thus not suitable for determination of very low concentrations or analysis of complex matrices (Kotrebai, Birringer, Tyson, Block, & Uden, 2000).

2.3. Results of speciation analyses

In 2008, an important compilation was realised about the principal data published until then concerning Se speciation in food (Rayman, 2008). Table 1 is a complement and update of this work with the most relevant data found in recent literature. As far as possible, attention was paid to select studies in which total Se concentrations after mineralisation and in the extract were both reported, together with data on Se speciation in the extract. The presence of all these data allows the best interpretation of what is really known about the species composition of a food item.

According to Table 1, meat products contain exclusively organic Se (Bierla et al., 2008). Inorganic Se is almost never found in food, except in cereals (Cubadda et al., 2010), mushrooms (Stefanka, Ipoly, Dernovics, & Fodor, 2001) and some vegetables (Pedrero et al., 2006) exposed to high quantities of selenite or selenate. Plants take up inorganic Se from the soil and are able to convert it into organic forms. Selenite is rapidly transformed and stored as SeMet or SeCys directly in the roots, while selenate, highly mobile in the xylem, is previously translocated in above ground plant parts (Sager, 2006) and less readily transformed (Terry, Zayed, de Souza, & Tarun, 2000). Selenium concentrations in most plants remain usually low except in "Se-accumulator" plants, typically those who belong to *Brassica* and *Allium* families, which can tolerate Se concentrations up to 10^3 times higher than usual plants (Dumont, Vanhaecke, et al., 2006). This particularity comes from the fact that these plants can convert mineral Se into two non-protein selenoamino acids, MeSeCys and γ-glutamyl-MeSeCys (γ-glut-MeSeCys) (Ogra, Ishiwata, Iwashita, & Suzuki, 2005). In this way, Se-accumulator plants avoid usual selenotoxicity induced by replacement of Met and Cys by their seleno-analogues into proteins, allowing the accumulation of very high quantities of Se (Pedrero & Madrid, 2009; Terry et al., 2000). In Table 1, most of the values concerning cereals and vegetables arise from enrichment experiments because the natural Se content of these products is very low. It must therefore be kept in mind that the results do not necessarily reflect the natural Se status of these plant species. Brazil nuts probably represent the food with the highest natural content of Se as two nuts provide about 100 μg of Se, mostly under the form of SeMet (Thompson et al., 2008). In sesame seeds, quite rich in Se as well, SeMet accounts for 80% of total Se (Kapolna, Gergely, Dernovics, Illes, & Fodor, 2007). The majority of cereal products collected in Table 1 contains between 80% and 96% of SeMet. Se-enriched yeast, which is consumed widely as a food supplement, is also essentially constituted of SeMet (Ip, Birringer, et al., 2000; Moreno, Quijano, Gutierrez, Perez-Conde, & Camara, 2004; Yoshida et al., 2002). Finally, Se speciation in fish seems to be rather case-dependent as Cappon and Smith (1981) reported a selenate content varying between 14% and 36% in muscle of various species of fish, while Reyes et al. (2009) could identify only SeMet in samples of three different fish species.

In most commonly consumed foods, SeMet seems to be the principal species of Se. However, the presence of particular compounds such as MeSeCys, trimethylselenonium ($TMSe^+$), or other unknown species, even in lower quantities, also might be determinant for the assessment of the Se health effect of food. Remarkably, in several matrices (fish, enriched onions, and enriched mushrooms), the species that could be identified and quantified represent only a limited fraction of the total Se. The characterisation of this 'unidentified' fraction of Se species, remains a challenge for future speciation studies, and may be of high importance to solve the scientific Se-puzzle.

In contrast with the scarcity of Se speciation data, a great number of papers report on total Se concentrations in food. These data are useful to have a global idea about the Se richness or poorness of various food categories. Se content in food all over the world has been reported or reviewed by numerous authors such as Finley, Matthys, Shuler, and Korynta (1996), Matos-Reyes, Cervera, Campos, and de la Guardia (2010), Navarro-Alarcon and Cabrera-Vique (2008), Smrkolj, Pograjc, Hlastan-Ribic, and Stibilj (2005), Thompson and Robinson (1990), Wyatt, Melendez, Acuña, and Rascon (1996). Since the Se content of local foods depends largely upon the selenium soil composition, it is important to specify the origin of the foodstuff in parallel to its Se content. It is also relevant to detail the food processing applied to the food. For example, cooking, boiling or freezing might induce changes in Se species initially present, or favour the loss of certain selenocompounds (Navarro-Alarcon & Lopez-Martinez, 2000; Pedrero & Madrid, 2009; Pedrero et al., 2006). Despite these factors of variation, some general trends can be observed. Typically, high protein-containing foodstuffs tend to be particularly rich in Se, which means that animal products generally contain more Se than plant materials. Among animal products, fish and crustaceans are the richest in Se (from 200 to >1000 ng Se g^{-1} wet weight). Offal such as liver and kidneys show the highest concentrations (>1000 ng Se g^{-1} wet weight), while Se in muscles usually ranges from 100 to 400 ng Se g^{-1} wet weight. As previously mentioned, plant products are ordinarily poor in Se (<10 ng Se g^{-1} wet weight) except some particular cases: brazil nuts and some species of mushrooms, that reach >5000 ng Se g^{-1} wet weight (Dumont, De Pauw, et al., 2006); and Se-accumulator plants that can exceed 10 μg Se g^{-1} wet weight when grown on seleniferous soil (Diaz Huerta, Fernandez Sanchez, & Sanz-Medel, 2006; Wrobel et al., 2004).

3. Selenium bioavailability

3.1. General points

At the start of this section about Se bioavailability, it seems useful to elaborate on the definition of "bioavailability" because, depending on the author, this term seems often to be defined and interpreted differently. Some authors define bioavailability as the fraction of an ingested element that is absorbed through the intestinal barrier and that passes into the bloodstream or an organ (Ruby et al., 1999). Other authors give a more restrictive definition and consider the bioavailable fraction as the quantity of Se

Table 1
Typical concentrations of Se species in various food sources.

Matrix	Food	Origin	Sample preparation	Extraction method	Quantification method	Typical concentrations						References
						Total Se (μg Se g^{-1})	Extracted Se (μg Se g^{-1})	Extraction efficiency (%)	Se species in extract	Se species concentration in extract (μg Se g^{-1})	Fraction of Se species to total Se (%)	
Marine products	Tuna		Lyophilised	Enzymatic	HPLC-ICP-MS	4.6 ± 0.1	4.4* ± 0.2	96	SeMet	0.28 ± 0.02	6[a]	Quijano et al. (2000)
									TMSe+	0.51 ± 0.07	12[a]	
	Tuna		Lyophilised	Enzymatic	HPLC-ICP-MS	3.9 ± 0.2			SeMet	1.61*	41*,b	Moreno et al. (2004)
	Tuna	Portugal	Dried	Enzymatic	HPLC-ICP-MS	2.32 ± 0.03			SeMet	1.07 ± 0.03	46*,b	Cabañero, Carvalho, Madrid, Batoreu, and Camara (2005)
	Canned tuna	USA	Freeze-dried	Enzymatic	HPLC-ICP-MS	5.6 ± 0.2	5.5 ± 0.2	98 ± 3	SeMet	1.6 ± 0.1	29 ± 2[a]	Reyes et al. (2009)
	Trout		Lyophilised	Enzymatic	HPLC-ICP-MS	2.9 ± 0.1			SeMet	0.76*	26*,b	Moreno et al. (2004)
									TMSe+	0.083*	3*,b	
	Shark	USA	Freeze-dried	Enzymatic	HPLC-ICP-MS	2.0 ± 0.1	0.9 ± 0.1	46 ± 5	SeMet	0.5 ± 0.1	56 ± 11[a]	Reyes et al. (2009)
	Marlin	USA	Freeze-dried	Enzymatic	HPLC-ICP-MS	3.9 ± 0.1	3.5 ± 0.1	89 ± 5	SeMet	1.6 ± 0.1	46 ± 3[a]	Reyes et al. (2009)
	Swordfish	Portugal	Dried	Enzymatic	HPLC-ICP-MS	2.09 ± 0.04			SeMet	1.95 ± 0.12	93*,b	Cabañero et al. (2005)
	Sardine	Portugal	Dried	Enzymatic	HPLC-ICP-MS	1.81 ± 0.02			SeMet	0.51 ± 0.02	28*,b	Cabañero et al. (2005)
	Oyster	IRMM (Belgium)	Lyophilised	Enzymatic	HPLC-ICP-MS	3.6 ± 0.3			SeMet	0.88*	24*,b	Moreno et al. (2004)
									TMSe+	0.30*	8*,b	
	Mussel	IRMM (Belgium)	Lyophilised	Enzymatic	HPLC-ICP-MS	1.7 ± 0.02			SeMet	0.48*	28*,b	Moreno et al. (2004)
									TMSe+	0.13*	8*,b	
Meat	Chicken breast		Freeze-dried	Enzymatic	HPLC-ICP-MS	0.54 ± 0.03			SeMet	0.36 ± 0.09	67*,b	Bierla et al. (2008)
									SeCys	0.11 ± 0.04	20*,b	
	Chicken leg		Freeze-dried	Enzymatic	HPLC-ICP-MS	0.57 ± 0.09			SeMet	0.32 ± 0.05	56*,b	Bierla et al. (2008)
									SeCys	0.18 ± 0.01	32*,b	
	Lamb heart		Freeze-dried	Enzymatic	HPLC-ICP-MS	1.26 ± 0.12			SeMet	0.25 ± 0.07	20*,b	Bierla et al. (2008)
									SeCys	0.75 ± 0.07	59*,b	
	Lamb liver		Freeze-dried	Enzymatic	HPLC-ICP-MS	1.41 ± 0.1			SeMet	0.25 ± 0.07	18*,b	Bierla et al. (2008)
									SeCys	0.85 ± 0.2	60*,b	
	Lamb kidney		Freeze-dried	Enzymatic	HPLC-ICP-MS	4.53 ± 0.6			SeMet	0.40 ± 0.26	9*,b	Bierla et al. (2008)
									SeCys	4.0 ± 0.07	88*,b	
Cereals	Spring wheat grains	Austria	Powder	Enzymatic	HPLC-ICP-MS		0.0122 ± 0.0007		SeMet	~0.011	~90*,a	Stadlober et al. (2001)
	Spring wheat grain	India	Freeze-dried	Enzymatic	HPLC-ICP-MS	83.1 ± 0.1		70–90	SeMet	55.1 ± 2.8	66*,b	Cubadda et al. (2010)
									MeSeCys	0.303 ± 0.026	<1*,b	
									Selenate	1.18 ± 0.07	1*,b	
	Summer barley grains	Austria	Powder	Enzymatic	HPLC-ICP-MS		0.0065 ± 0.0011		SeMet	~0.005	~77*,a	Stadlober et al. (2001)
	Durum wheat	Austria	Powder	Enzymatic	HPLC-ICP-MS		0.0241 ± 0.0032		SeMet	~0.015	~62*,a	Stadlober et al. (2001)
	Winter wheat grain	India	Freeze-dried	Enzymatic	HPLC-ICP-MS	29.5 ± 0.2		70–90	SeMet	17.1 ± 0.8	58*,b	Cubadda et al. (2010)
									MeSeCys	0.041 ± 0.005	<1*,b	
									Selenate	0.352 ± 0.041	1*,b	

(*continued on next page*)

Table 1 (*continued*)

Matrix	Food	Origin	Sample preparation	Extraction method	Quantification method	Typical concentrations						References
						Total Se (μg Se g^{-1})	Extracted Se (μg Se g^{-1})	Extraction efficiency (%)	Se species in extract	Se species concentration in extract (μg Se g^{-1})	Fraction of Se species to total Se (%)	
	Selenate enriched spring wheat grains	NA	Powder	Enzymatic	HPLC-ICP-MS		0.183 ± 0.014		SeMet	0.155 ± 0.008	85[a]	Stadlober et al. (2001)
									SeCys2	~0.016	~9[*,a]	
	Selenate enriched summer barley grains	NA	Powder	Enzymatic	HPLC-ICP-MS		0.188 ± 0.008		SeMet	0.148 ± 0.004	79[a]	Stadlober et al. (2001)
									SeCys2	~0.016	~10[*,a]	
	Selenate enriched *durum* wheat	NA	Powder	Enzymatic	HPLC-ICP-MS		0.218 ± 0.03		SeMet	0.187 ± 0.004	86[a]	Stadlober et al. (2001)
									SeCys2	~0.018	~9[*,a]	
	Selenate enriched buckwheat seed	NA	Air-dried	Enzymatic	HPLC-UV-HG-AFS, HPLC-ICP-MS	3.58 ± 0.80	171		SeMet	2.42[*]	68[*,b]	Vogrincic, Cuderman, Kreft, and Stibilj (2009)
	Wheat flour	IRMM (Belgium)	Lyophilised	Enzymatic	HPLC-ICP-MS	0.69 ± 0.09			SeMet	0.36 ± 0.09	52[*,b]	Moreno et al. (2004)
									SeCys2	0.13 ± 0.01	19[*,b]	
	Basmati rice	India	Powder	Enzymatic	HPLC-ICP-MS	0.67[*]	0.543 ± 0.007	81 ± 5	SeMet	0.507 ± 0.007	93[a]	Mar, Reyes, Rahman, and Kingston (2009)
	Jasmine rice	Thailand	Powder	Enzymatic	HPLC-ICP-MS	0.068[*]	0.048 ± 0.008	70 ± 7	SeMet	0.046 ± 0.006	96[a]	Mar et al. (2009)
	White rice	USA	Powder	Enzymatic	HPLC-ICP-MS	0.141[*]	0.124 ± 0.008	88 ± 8	SeMet	0.116 ± 0.007	94[a]	Mar et al. (2009)
	Rice-based cereal product	Italy	Powder	Enzymatic	HPLC-ICP-MS	0.062[*]	0.051 ± 0.009	82 ± 8	SeMet	0.046 ± 0.008	90[a]	Mar et al. (2009)
	Rice-based cereal product	USA	Powder	Enzymatic	HPLC-ICP-MS	0.323[*]	0.291 ± 0.01	90 ± 9	SeMet	0.259 ± 0.007	89[a]	Mar et al. (2009)
	Rice-based cereal product	Canada	Powder	Enzymatic	HPLC-ICP-MS	0.443[*]	0.39 ± 0.011	88 ± 10	SeMet	0.341 ± 0.012	87[a]	Mar et al. (2009)
Vegetables	Mushrooms *Agaricus*	Spain	Freeze-dried	Enzymatic	HPLC-ICP-MS	1.6 ± 0.1	0.99 ± 0.06	61 ± 3	SeMet	0.157 ± 0.007	16[*,a]	Diaz Huerta et al. (2006)
Enriched vegetables	Selenite enriched onion leaves	NA	Dried powder	Methanol:chloroform:water (12:5:3)	HPLC-ICP-MS	154 ± 6			MeSeCys	6.2[*]	4[b]	Wrobel et al. (2004)
									SeMet	0.5[*]	<1[b]	
	Selenate enriched onion leaves	NA	Dried powder	Methanol:chloroform:water (12:5:3)	HPLC-ICP-MS	601 ± 7			MeSeCys	11.4[*]	2[b]	Wrobel et al. (2004)
									SeMet	1.2[*]	<1[b]	
	Se enriched mushrooms	NA	Lyophilised	Enzymatic	HPLC-ICP-MS	51.4 ± 0.8	29 ± 4	56 ± 7	SeMet	2.32 ± 0.04	8[*,a]	Diaz Huerta et al. (2006)
	Selenite enriched mushrooms	NA	Powder	Enzymatic	HPLC-HHPN-AFS	110.2	83		SeCys2	27.7	33[*,a]	Stefanka et al. (2001)
									Selenite	46.4	56[*,a]	
	Se enriched garlic	NA	Mixed	Boiling-water bath	HPLC-ICP-MS, ESI-MS/MS				Selenate		8[a]	Ogra et al. (2005)
									MeSeCys		2[a]	
									γ-glu-MeSeCys		90[a]	
	Se enriched shallot	NA	Mixed		HPLC-ICP-MS, ESI-MS/MS				Selenate		28[a]	Ogra et al. (2005)
									MeSeCys		5[a]	

							γ-glu-MeSeCys		66[a]	
Yeast	Se enriched yeast	NA	Enzymatic	HPLC-ICP-MS	>90		SeMet		75[a]	Yoshida et al. (2002)
							SeCys2		10[a]	
							Selenite		5[a]	
	Se enriched yeast	NA	Lyophilised	HPLC-ICP-MS	1180 ± 40		SeMet	672[*]	57[*,b]	Moreno et al. (2004)
							Selenite	3.44[*]	<1[*,b]	
							Selenate	8.3 ± 0.3	<1[*,b]	
	Se enriched yeast	NA	Powder	Enzymatic	HPLC-ICP-MS	1922	SeMet		85[c]	Ip, Birringer, et al. (2000)
					80–90					

~, Approximate value.
NA, not applicable (concerning origin of selenised samples).
[a] Percentage of Se species compared to total extracted Se.
[b] Percentage of Se species compared to total Se content of the sample.
[c] Percentage of Se species compared to total eluted Se.
[*] Refers to data that have been calculated by author.

that is effectively used by the organism and transformed into a biochemically active form. Since a good terminology is the basis of good understanding, it is ideal, in this article, to differentiate between bioaccessibility, bioavailability and bioactivity, which each correspond to a different level of interaction that Se species can have with the organism. According to the scheme presented in Fig. 1, the bioaccessible fraction of an element is the fraction that is soluble in the intestine and that is therefore available for subsequent processes of absorption through the intestinal mucosa (Ruby et al., 1999; Shen, van Dyck, Luten, & Deelstra, 1997; Stahl et al., 2002). This soluble element fraction can be estimated by *in vitro* simulation of the gastro-intestinal digestion. Typically such simulation is conducted in two steps: a gastric digestion with pepsin at pH 2, followed by an intestinal digestion with amylase, pancreatin and bile salts at neutral pH (Kapolna & Fodor, 2007). The extract resulting from this procedure is considered to be the bioaccessible fraction. By opposition, non-accessible Se cannot be absorbed through the intestinal membrane and is directly excreted without having affected the body mechanisms. To define bioavailability we refer to Schümann's et al. definition (1997) designed for antioxidants. The bioavailable fraction is the fraction of an element that is absorbed and that reaches the systemic circulation in order to be distributed to organs and tissues, where it can eventually become bioactive. Bioavailability can be estimated by measuring Se level in blood and in body tissues. The bioactive part of Se is the one that is converted into biologically active selenometabolites. Most of the time bioactivity is estimated by measuring selenoprotein activity in blood and tissues.

It must be mentioned that, typically, studies dealing with Se bioavailability measure both Se levels and GPx activity induced by a given category of food or a given Se species. They estimate therefore both bioavailability and bioactivity of Se, but they usually do not make the distinction between these two notions and bioavailability is used as the general term.

Bioavailability of Se is tightly related to its chemical form but certain other components of the food matrix can have a considerable influence. For example Vitamins E and A have been shown to

Fig. 1. Ingested Se has to be separated into different fractions: (1) non-bioaccessible fraction: Se passing through the organism without affecting it and finishing in the faeces. (2) Bioaccessible fraction: Se soluble in the intestine. (3) Bioavailable fraction: Se absorbed through the intestinal barrier and reaching the systemic circulation. (4) Bioactive fraction: Se transformed into active selenometabolites.

increase Se bioavailability, while heavy metals and fibres tend to reduce it (Fairweather-Tait, 1997; Ralston, Ralston, Blackwell, & Raymond, 2008; Reeves et al., 2007). The nature of the lipids present in food can also affect Se bioavailability (Mutanen & Mykkänen, 1984), as well as dietary sulphur (especially from methionine (Met)) which competes with Se for absorption and utilisation (Waschulewski & Sunde, 1988). Finally, parameters related to the person itself have to be taken into account i.e. Se status, age, sex, lifestyle (Thompson, 2004).

3.2. Species-dependent metabolism of Se

Species-dependent bioavailability of Se is related to the distinct metabolic pathways that different species follow in the body. Fig. 2 gives a global view of what is known about metabolism of Se species in human body. The aim of this paragraph is not to describe in detail the complete biochemistry of Se, but rather to allow a better understanding of the impact of speciation on Se bioavailability.

3.2.1. Absorption

Organic and inorganic dietary Se are both commonly well absorbed through the intestinal membrane (70–95%) (Finley, 2006). Since Se and S (sulphur) have very similar properties, certain selenocompounds can be absorbed through the same pathways as their sulphur analogues. This is the case of selenate, that shares an active way of transport with sulphate, and of SeMet, that passes the intestinal barrier using the same Na^+-dependant process than Met (Schrauzer, 2000). Due to these shared transporters, there can be competition for absorption between the sulphur compounds and their seleno-analogues if they are both present in abundance in food. In contrast, selenite is taken up by a passive process that is not affected by sulphite; neither seems selenocysteine to be influenced by Cys (Stahl et al., 2002).

3.2.2. Transport

Following their absorption, Se species are translocated towards different organs and tissues with the help of proteic transporters, most of the time albumin or SeP. It seems that SeP enters the transport chain after albumin. Suzuki et al. (2009) suggested that ingested Se is firstly bound to albumin which transports the element to the liver, where Se is released and serves for the synthesis of selenoproteins, notably SeP. Selenoprotein P is then released into the bloodstream to become itself a Se transporter between the liver and various other organs and tissues. The liver is the organ with the highest Se content, followed by the kidneys. These organs are the two principal places of synthesis for most selenoproteins, especially SeP and cellular GPx in the liver and extracellular GPx in the kidneys (Suzuki et al., 2009). Other Se-containing organs/tissues are spleen, pancreas, blood, plasma, erythrocytes, skeleton, muscles and fat (Stahl et al., 2002).

3.2.3. Utilisation

Tissues with high rate of protein synthesis, especially skeletal muscles, seem to be a kind of storage place for Se under the form of SeMet. Methionine-tRNA is not able to discriminate between Met and SeMet (Schrauzer, 2000), and SeMet can therefore substitute non-specifically for Met into the muscles proteins to form Se-containing proteins (Ducros & Favier, 2004; Navarro-Alarcon & Cabrera-Vique, 2008). In contrast, the other forms of Se cannot be stored and they enter the pathway of selenoprotein synthesis. This requires the transformation of the original species into hydrogen selenide (H_2Se) (or its equivalent), the common precursor of selenoproteins for all nutritional sources of Se and, at the same time, the checkpoint intermediate for utilisation and excretion of this element (Suzuki, Doi, & Suzuki, 2006; Suzuki, Kurasaki, Ogawa, & Suzuki, 2006). The reaction of formation of selenide depends on the original form of Se. Selenite can be directly reduced into selenide by cellular glutathione (GSH). This reaction occurs in red blood cells, immediately after passage of selenite through the intestinal membrane. The freshly synthesised selenide is then transported to an organ of selenoprotein synthesis, usually the liver. Selenate reaches the liver under its original form and is afterwards reduced to selenite and selenide. Organic Se species as well reach the liver "as they are" and undergo their transformation once

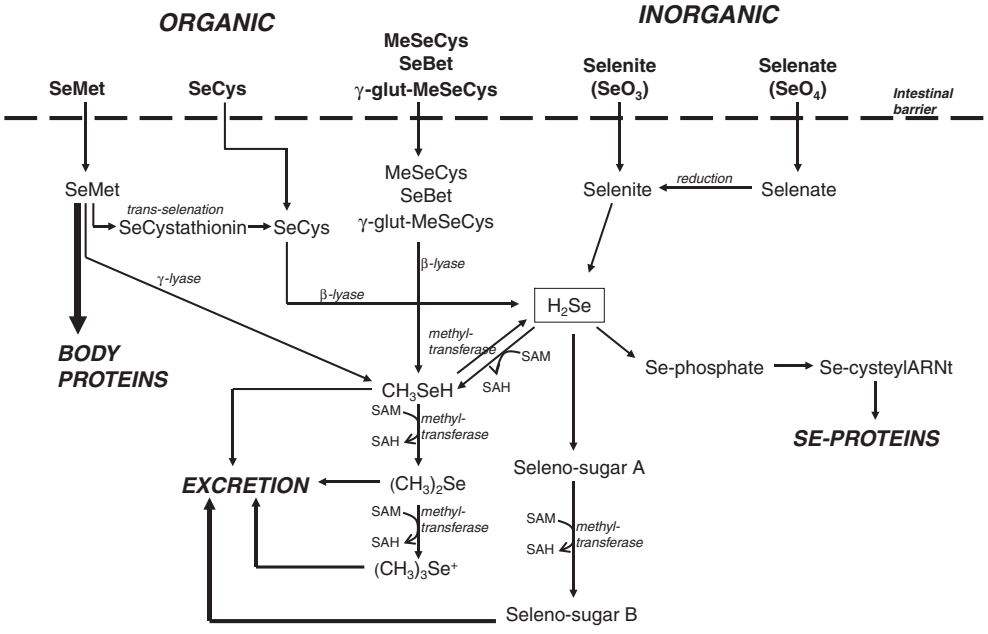

Fig. 2. Proposed schematic view of Se metabolism in humans (adapted from Suzuki, Doi, et al., 2006; Suzuki, Kurasaki, et al., 2006; Suzuki et al., 2008). CH_3SeH: methylselenol; $(CH_3)_2Se$: dimethylselenide; $(CH_3)_3Se^+$: trimethylselenonium; γ-glut-methylselenocysteine: gamma glutamine methylselenocysteine; GSH: glutathione; H_2Se: hydrogen selenide; MeSeCys: methylselenocysteine; SAH: S-adenosylhomocysteine; SAM: S-adenosylmethionine; SeBet: selenobetaine; SeCys: selenocysteine; SeMet: selenomethionine.

there (Beilstein & Whanger, 1988; Suzuki, Doi, et al., 2006; Suzuki, Tsuji, Ohta, & Suzuki, 2008). The fraction of SeMet that has not been unspecifically incorporated into body proteins, follows the *trans*-selenation pathway (similar to *trans*-sulphuration pathway) to be transformed in SeCys. β-Lyase is then able to cleave its carbon–Se bound to generate selenide. In the case of very excessive Se intake, SeMet can also be directly transformed into methylselenol (CH_3SeH) via γ-lyase, and then be demethylated into H_2Se (Suzuki, Doi, et al., 2006; Zeng, Botnen, & Johnson, 2008). Selenide is afterwards transformed into Se-phosphate, and Se-cysteyl-tRNA inserts a SeCys residue into the aminoacid sequence in order to form the active centre of selenoproteins. Although selenoproteins contain Se under SeCys form (Suzuki, 2005), it is interesting to mention that intact SeCys from food cannot be immediately used for selenoprotein synthesis, but has to be digested and transformed into H_2Se in order to synthesise *de novo* molecules of SeCys that will be incorporated into selenoproteins (Suzuki, Doi, et al., 2006; Suzuki, Kurasaki, et al., 2006). Methylated species, MeSeCys and γ-glut-MeSeCys, as well as selenobetaine, follow a different pathway than others selenospecies. After their absorption, they enter directly the methylated pool of Se by being transformed into CH_3SeH by β-lyase. This methylated pathway is above all destined to the excretion of Se. Only a fraction of methylselenol will be demethylated and transformed in H_2Se to join the selenoprotein synthesis pathway (Pedrero & Madrid, 2009; Suzuki, Doi, et al., 2006). As methylselenol has been recognised as an anti-cancer compound, the consumption of foodstuff containing MeSeCys, the main precursor of CH_3SeH, is generally recommended.

3.2.4. Excretion

Excretion of Se is regulated by its intake, and methylation and demethylation reactions between selenide and methylselenol seem to be a critical step in this process (Suzuki et al., 2008). When Se intake is poor, its excretion is reduced and Se supply is maintained to priority organs (brain, reproductive organs, and endocrine glands) while cellular GPx content of the rest of the body quickly decreases (Ducros & Favier, 2004; Steinbrenner & Sies, 2009). On the contrary, when Se intake is important, excretion of Se increases. Urine is the principal way of excretion in humans and Se is generally eliminated as a selenosugar (Se-methyl-*N*-acetylgalactosamide or selenosugar B). Under conditions of higher Se intake, also methylated forms can be excreted, either in urine (as trimethylselenonium ion or sometimes as methylselenol), either by breathing (as dimethylselenide) leading to a characteristic odour of garlic on the breath (Suzuki, Doi, et al., 2006; Suzuki, Kurasaki, et al., 2006). However, despite an increased excretion, in case of huge Se excess, part of ingested Se is retained and can accumulate (van der Torre, Van Dokkum, Schaafsma, Wedel, & Ockhuizen, 1991), which can lead on the long term to selenotoxicity.

3.3. Methods for study of selenium bioavailability

To study bioavailability of Se, different approaches can be considered: experiments can be conducted *in vitro* on cultivated intestinal cells, or *in vivo* on laboratory animals or directly on persons.

In vitro experiments have the advantage to be faster, cheaper and simpler compared to *in vivo* experiments. In addition, it is a manner to spare laboratory animals, or to carry out experiments that might be not feasible or practical on living models (Zeng et al., 2008). Recently a cell culture model making use of Caco-2 cells has been developed to study *in vitro* bioavailability of Se. Caco-2 cells derive from a human colon carcinoma cell line and have similar structural and functional characteristics that colon enterocytes, including polarity and induction of specific enzymes. This makes this model suitable to study various processes such as cellular uptake, retention, transport, metabolism and selenoprotein synthesis (Zeng et al., 2008). However, when working directly on intestinal cells, gastro-intestinal tract is by-passed. Gastro-intestinal tract is a place where, most of the time, Se species undergo several transformations because of the hard stomach conditions and the interactions that they can create with various functional groups of other food components (Cabañero, Madrid, & Camara, 2004). To reproduce natural conditions of ingestion in these kind of studies, it is therefore necessary to previously digest artificially the food to be tested, and then to add it in the cell culture medium (Glahn, Lee, Yeung, Goldman, & Miller, 1998).

Animal models were the firsts to be used and are still nowadays the most common models for bioavailability studies. Most of the time, lab animals are rodents or, less frequently, chicks (Gabrielsen & Opstvedt, 1980; Wang & Xu, 2008) or fishes (Rider et al., 2009; Wang, Han, Li, & Xu, 2007). Typically, animals are firstly made Se-deficient with a low-Se diet, to be fed afterwards with the food of interest. During the period of repletion, the extent and the rapidity of the Se status recovery are measured. In parallel, a group of animals is often fed with a reference selenocompound in order to compare the results obtained for Se from the food tested with these, obtained for the single species. More precisely, in the "slope-ratio assay" method that has been reported by Suttle (1974), the slope of the regression line generated by a dose–response relationship of a tested food item is divided by the slope of the regression line generated by the reference selenocompound. The resulting ratio can then be used to quantitatively compare different food matrices (Alexander et al., 1983). This method has been largely applied for analysis of bioavailability in beef (Shi & Spallholz, 1994), tuna (Alexander et al., 1983), salmon (Ornsrud & Lorentzen, 2002), wheat (Alexander et al., 1983; Reeves et al., 2007), buckwheat bran (Reeves et al., 2005), Se-enriched algae (Cases et al., 2002) or Se-enriched broccoli (Finley, 1998).

Studying bioavailability directly on human people is obviously the most appropriate method, compared to animal or cellular models, but it involves additional problems. On the one hand, people are not as standardisable as animals or cells and, on the other hand, they cannot release as much information as animals that are usually sacrificed at the end of the experiment, allowing the analysis of biopsied tissues and organs. In studies conducted on humans, the food tested can be labelled with stable isotopes of Se in order to differentiate the Se contained in the tested food items from the Se coming from other constituents of the diet or from endogenous excretions (Fairweather-Tait, 1997). It is recommended to also administer a reference dose with different isotopic label so that differences in individual absorption capacities can be eliminated (Fairweather-Tait, 1997).

In humans and animals, after the Se treatment, different parameters can be measured in order to evaluate the nutritional impact of Se on the organism. The most frequently analysed parameters are GPx activity and Se levels, usually measured in faeces, urine, blood and various body tissues. These measurements actually allow the determination of the three successive levels of interaction that can occur between Se and the human body, i.e. bioacccessibility (analysis of Se in faeces), actual bioavailability (analysis of Se in urine and tissues) and bioactivity (analysis of GPx activity). As mentioned before, in practice, authors rarely make the distinction between the different notions and include all the analysed parameters into the term "bioavailability" of Se.

3.4. Data about selenium bioavailability

The following paragraph is a compilation of the relevant outcomes that have been published during the last decades concerning Se bioavailability. This work is, to our knowledge, realised for the first time. This is probably due to the fact that, because of the multiple methods, models and experimental designs that have

been used to measure Se bioavailability, it is not evident to compare all the data that have been published. To allow a comparison to a certain extent, it was chosen to not express the results as absolute values such as these presented in the original articles, but the results were 'standardised' by calculation of the relative bioavailability of a tested foodstuff compared to the reference selenocompound used in the study, usually selenite or selenate. For each different kind of matrix the results were summarised in separate tables (Tables 2–4).

3.4.1. Selenium bioavailability of single species

Many studies have tried to compare bioavailability of individual organic and inorganic Se species, or Se supplemented yeast. Table 2 gives an overview of results obtained in these studies. In some cases, a control was made with non-supplemented subjects. The response of this control was always inferior or equal to the reference compound, illustrating that all the tested Se species were able to affect positively the Se status of the subjects. Among the parameters considered, apparent digestibility (calculated as the difference between Se intake and Se excretion in faeces) was significantly higher in animals supplemented with Se yeast (known to be essentially constituted of SeMet) than selenite (Rider, Davies, Jha, Clough, & Sweetman, 2010). In addition, in each body location, Se concentrations were higher after SeMet supplementation than with inorganic Se, and this with an order of magnitude increasing with the dose of Se administered (Rider et al., 2009; Whanger & Butler, 1988). Without surprise, Se yeast increased Se status to a similar extent to SeMet (Wang & Lovell, 1997). In two different studies conducted on humans, the Se concentration in blood reached a plateau at the end of the supplementation period in seleni(/a)te-supplemented subjects, while it was still rising in SeMet-supplemented people (Levander et al., 1983; Thompson, Robinson, Campbell, & Rea, 1982). This would suggest that the long term effects of SeMet are larger than those of mineral Se. On the other hand, based on selenoenzyme induction, no real difference was observed between inorganic and organic Se, and there was no effect of the quantity of administered Se. Rider et al. (2009) suggested that this absence of a concentration effect could be due to the fact that Se requirements were already met by the basal diet. Finally, concerning MeSeCys, it is surprising to notice that this organic species shows a behaviour closer to inorganic Se than SeMet when tested on Caco-2 cells, i.e. a lower Se accumulation but a higher GPx induction than SeMet (Zeng et al., 2008).

3.4.2. Selenium bioavailability in fish

The first part of Table 3 shows the synthesises of Se bioavailability in several fish species. As a matter of comparison with other matrices, authors often included a sample of wheat or yeast in addition to the fish samples. Tuna has been studied in multiple studies and its ability to increase Se concentration in body tissues and RBC seems similar or slightly lower than selenite and wheat. Concerning GPx induction, tuna was less efficient than both wheat and selenite, being frequently inferior to 50% of selenite (Alexander, Whanger, & Miller, 1983; Douglass, Morris, Soares, & Levander, 1981; Wen et al., 1997). It has been suggested that bioaccessibility of Se in tuna is low (about half of Se would be accessible), and this could be part of an explanation of the low Se bioavailability in this fish (Cabañero et al., 2004). For none of the measured parameters, an effect of the dose of Se intake was observed (Alexander et al., 1983). Regarding other fish species, no clear trend could be put forward. Scientific literature is often pessimistic concerning Se bioavailability in marine products and does not consider them as a recommendable source of dietary Se, despite their usually high Se content. Curiously, however, according to the data shown in Table 3, marine products seem to be particularly well absorbed and retained by the body. Selenium in trout, salmon and shrimps is better or equally absorbed and retained compared to inorganic Se. Fish might become a more interesting source of Se when it has previously been enriched with organic Se. Ornsrud and Lorentzen (2002) compared on rats Se bioavailability of SeMet-enriched salmon with selenite. They found, despite an equal absorption, that Se from enriched salmon was almost two times better retained than selenite, that its accumulation in organs ranged from 113% to 274%, and that GPx activity was 141% more important compared to selenite.

3.4.3. Selenium bioavailability in meat

The second part of Table 3 is concerned with meat products. Most meat-related studies focus on beef and pork, both commonly eaten by western populations. Concerning beef, different pieces have been tested and usually exhibit a Se bioavailability similar to the selenite reference. One study was particularly positive concerning Se from beef and considered it as "highly bioavailable" (Shi & Spallholz, 1994). Concerning pork meat, despite a low Se excretion, sign of a high absorption and retention, Bügel, Sandström, and Skibsted (2004) could not measure any increase in Se concentration nor in selenoenzymes activity. Based on these results, the authors suggested that Se in pork meat is not in the form of selenite nor SeMet that have been shown to both increase these parameters. The other kinds of meat analysed all showed a GPx induction similar to selenite (Wen et al., 1997). Finally, van der Torre et al. (1991) concluded that Se from naturally Se-rich meat (with no detail concerning the kind of meat) was available to a similar extent than Se from Se-rich bread.

3.4.4. Selenium bioavailability in vegetables

Few studies have dealt with vegetables, probably because of the low Se content in plants which makes speciation analysis difficult or even impossible. Some studies could nevertheless be conducted on Se-accumulator plants, previously enriched with Se fertilisers in order to reach appropriate concentrations for analysis. For example, in Table 4, Finley (1998) studied the ability of Se-enriched broccoli to restore Se and GPx levels in rats compared to SeMet, selenite and selenate. The three single Se species had a better capacity than broccoli to restore these parameters. However, because of this lower accumulation, plasma and tissues of rodents were less quickly saturated after high-Se-broccoli administration than after administration of the single species. These results show that Se is present in broccoli in a different form that selenite, selenate or SeMet, and that this form participates less intensively to selenoprotein synthesis but on a longer term, as reflected by the monitored GPx induction. With reference to speciation studies in *Brassica* plants, this form is probably MeSeCys or γ-glut-MeSeCys, a compound that is more likely to be transformed into CH_3SeH rather than in H_2Se. Se-enriched broccoli was also investigated through the Caco-2 cell model (Zeng et al., 2008) after having been previously artificially digested *in vitro* with pepsin, pancreatin and bile extract (Glahn et al., 1998). Caco-2 cells were grown in medium depleted in Se and then supplemented with increasing concentrations of either digested broccoli, either directly MeSeCys. After 72 h of incubation, they found that GPx enzymes were more induced by MeSeCys than by broccoli but with a difference becoming less significant with increasing quantities of Se. Owing to the fact that MeSeCys is the major Se species is broccoli, this shows that, at least at low concentrations, complexity of food matrix may significantly affect Se bioavailability. A few experiments were also made on naturally Se-rich plant products. Brazil nuts, for example, were able to increase Se content in the liver of rodents (Chansler, Mutanen, Morris, & Levander, 1986) and to activate GPx activity in human blood (Thompson et al., 2008). Mushrooms, on the other hand, had a very low capacity in stimulating these parameters, which makes the authors suggest that mushrooms may contain slightly available Se species such as methylated com-

Table 2
Selenium species bioavailability.

Monitored parameter	Location	Model of study	Duration of supplementation	Dose of supplementation	Relative bioavailability						References
					Selenate	Selenite	SeMet	Se yeast	MeSeCys	Control	
Se apparent digestibility		Trout	10 weeks	~2 μg Se g^{-1} food		1[a]		~1.2[b]		~1.1[a]	Rider et al. (2010)
Se concentration	Intestinal cell	Caco-2 cells	72 h	15.6 nmol l^{-1}		1[a]	~0.6[ab]		~0.8[a]	~0.2[b]	Zeng et al. (2008)
				31.2 nmol l^{-1}		1[a]	~1[a]		~0.8[a]	~0.1[b]	
				62.5 nmol l^{-1}		1[a]	~1.5[b]		~0.9[b]	~0.1[c]	
				125 nmol l^{-1}		1[a]	~2.2[b]		~1.1[a]	~0.1[c]	
	Whole body	Trout	10 weeks	~2 μg Se g^{-1} food		1[a]		1.75[b]		0.56[c]	Rider et al. (2009)
				~4 μg Se g^{-1} food		1[a]		1.71[b]		0.32[c]	
				~8 μg Se g^{-1} food		1[a]		1.34[b]		0.15[c]	
	Whole blood	Rats	9 weeks	0.2 μg Se g^{-1} food		1*	1.2*			0.06*	Whanger and Butler (1988)
				1 μg Se g^{-1} food		1*	1.75*			0.05*	
				2 μg Se g^{-1} food		1*	2.47*			0.05*	
				4 μg Se g^{-1} food		1*	4.13*			0.04*	
		Human	17 weeks	100 μg day^{-1}		1*	1.61*				Thompson et al. (1982)
	Plasma	Trout	10 weeks	~2 μg Se g^{-1} food		1[a]		1.12[b]		0.82[c]	Rider et al. (2010)
		Human	17 weeks	100 μg day^{-1}		1*	1.5*				Thompson et al. (1982)
		Human	11 weeks	200 μg day^{-1}	1*		~1.5*				Levander et al. (1983)
	RBC	Human	17 weeks	100 μg day^{-1}		1*	1.7*				Thompson et al. (1982)
		Human	11 weeks	200 μg day^{-1}	1*		~1.8*				Levander et al. (1983)
	Liver	*Channel catfish*	*9* weeks	*0–0.4* μg Se g^{-1} food		*1*	*1.97*	*1.84*			Wang and Lovell (1997)
		Trout	10 weeks	~2 μg Se g^{-1} food		1[a]		0.97[a]		0.54[b]	Rider et al. (2010)
		Rats	8 weeks	0.115 μg Se g^{-1} food		1[a]	1.16[b]				Shi and Spallholz (1994)
		Broiler chicken	21 days	~0.2 μg Se g^{-1} food		1[a]		1.26[b]		0.63[c]	Wang and Xu (2008)
		Rats	9 weeks	0.2 μg Se g^{-1} food		1*	1.24*			0.03*	Whanger and Butler (1988)
				1 μg Se g^{-1} food		1*	1.94*			0.02*	
				2 μg Se g^{-1} food		1*	2.94*			0.02*	
				4 μg Se g^{-1} food		1*	3.35*			0.01*	
	Muscles	*Channel catfish*	*9* weeks	*0–0.4* μg Se g^{-1} food		*1*	*4.78*	*4.53*			Wang and Lovell (1997)
		Crucian carp	30 days	~0.5 μg Se g^{-1} food		1[a]	1.29[b]			0.54[c]	Wang et al. (2007)
		Trout	10 weeks	~2 μg Se g^{-1} food		1[a]		1.61[b]		0.91[a]	Rider et al. (2010)
		Rats	8 weeks	0.115 μg Se g^{-1} food		1[a]	1.49[b]				Shi and Spallholz (1994)
		Broiler chicken	21 days	~0.2 μg Se g^{-1} food		1[a]		1.11[a]		0.70[b]	Wang and Xu (2008)
		Rats	9 weeks	0.2 μg Se g^{-1} food		1*	2.80*			0.20*	Whanger and Butler (1988)
				1 μg Se g^{-1} food		1*	9.36*			0.14*	
				2 μg Se g^{-1} food		1*	16.80*			0.13*	
				4 μg Se g^{-1} food		1*	26.41*			0.12*	
	Kidneys	Broiler chicken	21 days	~0.2 μg Se g^{-1} food		1[a]		1.22[b]		0.46[c]	Wang and Xu (2008)
		Trout	10 weeks	~2 μg Se g^{-1} food		1[a]		1.08[b]		0.91[c]	Rider et al. (2010)
		Rats	9 weeks	0.2 μg Se g^{-1} food		1*		1.08*		0.21*	Whanger and Butler (1988)
				1 μg Se g^{-1} food		1*		1.79*		0.14*	
				2 μg Se g^{-1} food		1*		2.59*		0.13*	
				4 μg Se g^{-1} food		1*		3.00*		0.10*	
GPx activity	Intestinal cell	Caco-2 cells	72 h	15.6 nmol l^{-1}		1[a]	~0.2[b]		~0.8[c]	~0.1[b]	Zeng et al. (2008)
				31.2 nmol l^{-1}		1[a]	~0.2[b]		~0.9[a]	~0.1[c]	
				62.5 nmol l^{-1}		1[a]	~0.5[b]		~1[a]	~0.1[c]	

(*continued on next page*)

Table 2 (*continued*)

Monitored parameter	Location	Model of study	Duration of supplementation	Dose of supplementation	Relative bioavailability						References
					Selenate	Selenite	SeMet	Se yeast	MeSeCys	Control	
				125 nmol l^{-1}		1[a]	~0.7[b]		~1.1[a]	~0.1[c]	
	Plasma	*Channel catfish*	*9* weeks	*0–0.4* μg Se g^{-1} food		*1*	*1.16*	*1.16*			Wang and Lovell (1997)
		Crucian carp	30 days	~0.5 μg Se g^{-1} food		1[a]	1.91[b]			0.50[c]	Wang et al. (2007)
		Broiler chicken	21 days	~0.2 μg Se g^{-1} food		1[a]		1.14[b]		0.66[c]	Wang and Xu (2008)
	Serum	*Chicks*	*9* days			*1*	*0.78*				Gabrielsen and Opstvedt (1980)
		Human	17 weeks	100 μg day^{-1}		1*	1.1*				Thompson et al. (1982)
		Human	11 weeks	200 μg day^{-1}	1*		~1*				Levander et al. (1983)
	RBC	Rats	9 weeks	0.2 μg Se g^{-1} food		1*	0.96*			0.11*	Whanger and Butler (1988)
				1 μg Se g^{-1} food		1*	0.99*			0.09*	
				2 μg Se g^{-1} food		1*	1.04*			0.08*	
				4 μg Se g^{-1} food		1*	0.96*			0.08*	
	Muscles	Rats	9 weeks	0.2 μg Se g^{-1} food		1*	1.11*			0.10*	Whanger and Butler (1988)
				1 μg Se g^{-1} food		1*	1.20*			0.08*	
				2 μg Se g^{-1} food		1*	1.00*			0.08*	
				4 μg Se g^{-1} food		1*	1.14*			0.10*	
	Liver	*Channel catfish*	*9* weeks	*0–0.4* μg Se g^{-1} food		*1*	*1.47*	*1.49*			Wang and Lovell (1997)
		Crucian carp	30 days	~0.5 μg Se g^{-1} food		1[a]	~0.9[a]			~0.7[b]	Wang et al. (2007)
		Trout	10 weeks	~2 μg Se g^{-1} food		1		1.02		0.88	Rider et al. (2010)
		Trout	10 weeks	~2 μg Se g^{-1} food		1		0.81		0.94	Rider et al. (2009)
				~4 μg Se g^{-1} food		1		1.01		1.09	
				~8 μg Se g^{-1} food		1		0.95		1.17	
		Broiler chicken	21 days	~0.2 μg Se g^{-1} food		1[a]		1.04[a]		0.74[b]	Wang and Xu (2008)
		Rats	8 weeks	0.115 μg Se g^{-1} food		1[a]	1.34[b]				Shi and Spallholz (1994)
		Rats	9 weeks	0.2 μg Se g^{-1} food		1*	1.02*			0.01*	Whanger and Butler (1988)
				1 μg Se g^{-1} food		1*	0.81*			0.01*	
				2 μg Se g^{-1} food		1*	1.03*			0.01*	
				4 μg Se g^{-1} food		1*	0.98*			0.01*	
	Kidneys	Rats	9 weeks	0.2 μg Se g^{-1} food		1*	1.05*			0.05*	Whanger and Butler (1988)
				1 μg Se g^{-1} food		1*	1.18*			0.04*	
				2 μg Se g^{-1} food		1*	0.92*			0.05*	
				4 μg Se g^{-1} food		1*	0.92*			0.04*	
TRx activity	Liver	Trout	10 weeks	~2 μg Se g^{-1} food		1[ab]		1.39[a]		0.93[b]	Rider et al. (2010)
		Trout	10 weeks	~2 μg Se g^{-1} food		1[a]		0.92[a]		0.81[b]	Rider et al. (2009)
				~4 μg Se g^{-1} food		1[a]		1.18[a]		0.64[b]	
				~8 μg Se g^{-1} food		1[a]		1.03[a]		0.66[b]	

Letters superscript indicates when difference between Se species is significant.
Results in italic have been calculated with slope-ratio method.
* Refers to results without indication on statistical significance.

pounds (Chansler et al., 1986). The behaviour of Se in wheat is similar to SeMet, which is not surprising as it is known that SeMet can represent almost the totality of Se species in wheat grain (Stadlober et al., 2001). Selenium from wheat has been shown to be particularly well retained by various body tissues in many studies (Douglass et al., 1981; Levander et al., 1983; Meltzer et al., 1993), especially when it is milled in meal, as this process disrupts the fibrous cell walls that can impede Se bioaccessibility (Reeves et al., 2007). GPx activity induced by wheat-Se seems also to be satisfactory, even if the results are less clear than for Se retention (Alexander et al., 1983; Douglass et al., 1981). Finally, bioavailability of Se from soybean and corn gluten meal was found to be very low (Gabrielsen & Opstvedt, 1980).

4. Discussion

Most of the studies dealing with "Se bioavailability" focus on two parameters: Se status and induction of selenoenzyme activity.

Table 3
Selenium species bioavailability in animal products.

Monitored parameter	Location	Model of study	Duration of supplementation	Dose of supplementation	Relative bioavailability										References
FISH					Selenate	Selenite	Tuna	Trout	Capelin	Mackerel	Flounder	SeMet-enriched salmon	Wheat	Yeast	
Se apparent absorption		Human	2 days		1[a]			0.96[a]						0.58[b]	Fox et al. (2004)
		Rats		0.1 μg Se g^{-1} food		1						1.04			Ornsrud and Lorentzen (2002)
Se body retention		Human	2 days		1[a]			1.44[b]						0.95[ab]	Fox et al. (2004)
		Rats		0.1 μg Se g^{-1} food		1[a]						1.84[b]			Ornsrud and Lorentzen (2002)
Se concentration	Whole blood	*Rats*	*4* weeks	*0.05–0.15* μg Se g^{-1} food		*1*	*0.6*						*0.8*		Alexander et al. (1983)
	Plasma	*Rats*	*30* days	*0–0.2* μg Se g^{-1} food		*1*						*1.25*			Ornsrud and Lorentzen (2002)
	Young RBC	Rats	4 weeks	0.2 μg Se g^{-1} food		1	1						1.14		Douglass et al. (1981)
	Liver	Rats	4 weeks	0.2 μg Se g^{-1} food		1	0.69						0.79		Douglass et al. (1981)
		Rats	*4* weeks	*0.05–0.15* μg Se g^{-1} food		*1*	*0.9*						*1*		Alexander et al. (1983)
		Rats	9 weeks	0.8 ± 0.4 μg Se g^{-1} food		1[a]	1.09[ab]				1.21[ab]				Wen et al. (1997)
		Rats	*30* days	*0–0.2* μg Se g^{-1} food		*1*						*1.15*			Ornsrud and Lorentzen (2002)
	Muscles	*Rats*	*4* weeks	*0.05–0.15* μg Se g^{-1} food		*1*	*1*						*2*		Alexander et al. (1983)
		Rats	9 weeks	0.8 ± 0.4 μg Se g^{-1} food		1	0.91				1.47				Wen et al. (1997)
		Rats	*30* days	*0–0.2* μg Se g^{-1} food		*1*						*2.25*			Ornsrud and Lorentzen (2002)
	Kidneys	*Rats*	*4* weeks	*0.05–0.15* μg Se g^{-1} food		*1*	*0.75*						*1*		Alexander et al. (1983)
	Femur	*Rats*	*30* days	*0–0.2* μg Se g^{-1} food		*1*						*1.13*			Ornsrud and Lorentzen (2002)
GPx activity	Whole blood	*Rats*	*4* weeks	*0.05–0.15* μg Se g^{-1} food		*1*	*0.44*						*1.08*		Alexander et al. (1983)
	Serum	*Chicks*	*9* days			*1*			*0.48*	*0.34*					Gabrielsen and Opstvedt (1980)
		Rats	*30* days	*0–0.2* μg Se g^{-1} food		*1*						*1.39*			Ornsrud and Lorentzen (2002)
	Young RBC	Rats	4 weeks	0.2 μg Se g^{-1} food		1[a]	0.54[b]						0.72[c]		Douglass et al. (1981)
	Liver	Rats	4 weeks	0.2 μg Se g^{-1} food		1[a]	0.49[b]						0.75[a]		Douglass et al. (1981)
		Rats	*4* weeks	*0.05–0.15* μg Se g^{-1} food		*1*	*0.38*						*0.7*		Alexander et al. (1983)
		Rats	9 weeks	0.8 ± 0.4 μg Se g^{-1} food		1	1.26				1.32				Wen et al. (1997)
	Kidneys	*Rats*	*4* weeks	*0.05–0.15* μg Se g^{-1} food		*1*	*0.34*						*1.33*		Alexander et al. (1983)
MEAT					Selenite	Veal	Chicken	Pork	Lamb	Beef	Beef kidney	Beef liver	Beef striploin	Wheat	
Se concentration	Young RBC	Rats	4 weeks	0.2 μg Se g^{-1} food	1						1			1.14	Douglass et al. (1981)

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Table 3 (*continued*)

Monitored parameter *FISH*	Location	Model of study	Duration of supplementation	Dose of supplementation	Relative bioavailability									References
					Selenate	Selenite	Tuna	Trout	Capelin	Mackerel	Flounder	SeMet-enriched salmon	Wheat	Yeast	
	Liver	Rats	4 weeks	0.2 μg Se g^{-1} food	1						0.74			0.79	Douglass et al. (1981)
		Rats	8 weeks	~0.115 μg g^{-1}	1							0.97	1.00		Shi and Spallholz (1994)
	Muscles	Rats	8 weeks	~0.115 μg g^{-1}	1							0.96	1.16		Shi and Spallholz (1994)
GPx activity	Young RBC	Rats	4 weeks	0.2 μg Se g^{-1} food	1[a]						0.65[b]			0.72[b]	Douglass et al. (1981)
	Liver	Rats	4 weeks	0.2 μg Se g^{-1} food	1						0.89			0.75	Douglass et al. (1981)
		Rats	8 weeks	~0.115 μg g^{-1}	1[a]							1.19[b]	1.16[b]		Shi and Spallholz (1994)
		Rats	9 weeks	0.8 ± 0.4 μg Se g^{-1} food	1[ab]	0.95[a]	0.96[a]	1.06[a]	0.72[b]	0.99[a]					Wen et al. (1997)

Letters superscript indicates when difference between Se species is significant.
Results in italic have been calculated with slope-ratio method.

Usually, Se status is estimated through Se concentration in blood (whole blood, plasma, RBC or platelet) or in body tissues (liver, muscle, kidneys). It can also be evaluated via urinary excretion, which reflects short-term Se intake, or via Se content in nail and hair, which reflects long-term status (Thompson, 2004). To estimate selenoenzyme activity, GPx is often the only selenoenzyme that is monitored, although many other selenoenzymes exist. The reason is that GPx is the most abundant selenoenzyme and that its activity is easy to be quantified. In particular, GPx activity in platelets seems to be a more sensitive indicator of Se bioavailability (Thompson, 2004) than GPx from plasma or erythrocytes which reach a plateau more rapidly (Bügel, Sandström, & Larsen, 2001; van der Torre et al., 1991).

According to a great number of studies making use of many different lab models (rodent, fish, chicken, cells, and humans), organic Se species are concluded to be more available for the organism than inorganic species, at least when they are not entrapped into a complex matrix. This conclusion is based on a better absorption (difference between Se intake and faecal excretion), a greater retention (difference between Se intake and faecal + urinary excretion) (Fox et al., 2004) and a higher accumulation of Se in blood and tissues (especially muscles) when organic species (SeMet and Se-yeast) are administered, compared to inorganic species (selenite and selenate). Conclusions are less clear when considering GPx after ingestion of one or another kind of Se species. It has been reported that organic Se accumulates better in tissues, but generates a lower GPx activity compared to inorganic Se. However, this observation seems to concern specifically SeMet, rather then being a global characteristic of organic species. MeSeCys, for instance, does not share these features with SeMet (Zeng et al., 2008). This accumulation capacity of SeMet is explained by the fact that this species, in contrast with other organic species, can be directly incorporated into body proteins instead of Met. However this important pool of SeMet is not automatically related to a greater bioactivity because, if SeMet is incorporated non-specifically to body proteins, it cannot be recognised as Se to be mobilised by the organism for selenoproteins synthesis. SeMet rather seems to be an efficient reserve of Se in case of long term shortage (Behne, Alber, & Kyriakopoulos, 2009) because of the retrieval of Se following catabolisation of SeMet during the natural protein turn-over (Fox et al., 2004; Zeng et al., 2008). Once released as free SeMet, this compound can be recognised as Se and enter the selenoprotein synthesis pathway. The biological half-time of SeMet stored in tissues appears to be largely dependent on metabolic factors of individual tissues as well as nutritional habits. Dietary intake of Met has a particularly great influence on SeMet turn-over. Methionine is a limiting aminoacid, which means that it cannot be synthesised by the human body and it must obligatory be taken up in food. As a consequence, if food contains SeMet instead of Met, SeMet risks to join almost exclusively the body protein pathway and to remain blocked under this storage form. Selenium is thus unable to be released for selenoprotein synthesis, which can lead to a Se deficiency despite an adequate intake of SeMet (Waschulewski & Sunde, 1988). By contrast, inorganic species directly join the pathway of selenoprotein synthesis and, under normal conditions, the excess is not stored but directly excreted. This might explain the observations of Thompson et al. (1982) and Levander et al. (1983), i.e. "that the Se content of blood tended to plateau rapidly on people supplemented with selenite and selenate", because of the saturation of enzymatic machinery; and "that Se level decreased rapidly after the end of supplementation", because there was no storage of this element. The question has been raised about a particular risk of toxicity generated by a long term accumulation of SeMet into proteins. It is reasonable to presume that such an accumulation could lead to a deficit in Met as well as to an excessive release of Se

Table 4
Selenium species bioavailability in plant products.

Monitored parameter	Location	Model of study	Duration of supplementation	Dose of supplementation	Relative bioavailability									References
					MeSeCys	Selenite	SeMet	Se-enriched broccoli	Mushroom	Brazil nut	Soybean	Corn gluten meal	Control	
Se concentration	Plasma	Rats	9 weeks	0.1 μg Se g^{-1} food		1*		~0.82*						Finley (1998)
		Rats	4 weeks	0.1 μg Se g^{-1} food		1[a]			0.21[b]	1.06[a]				Chansler et al. (1986)
		Human	6 weeks	~100 μg Se day^{-1}			1[a]			0.97[a]			0.62[b]	Thompson et al. (2008)
	RBC	Rats	9 weeks	0.1 μg Se g^{-1} food		1		~0.67						Finley (1998)
	Liver	Rats	9 weeks	0.1 μg Se g^{-1} food		1*		~0.9*						Finley (1998)
		Rats	4 weeks	0.1 μg Se g^{-1} food		1[a]			0.52[b]	1.36[c]				Chansler et al. (1986)
	Muscles	Rats	9 weeks	0.1 μg Se g^{-1} food		1		~0.81						Finley (1998)
GPx activity	Intestinal cell	Caco-2 cells	72 h	15.6 nmol l^{-1}	1[a]			~0.29[b]						Zeng et al. (2008)
				31.2 nmol l^{-1}	1[a]			~0.45[b]						
				62.5 nmol l^{-1}	1[a]			~0.76[b]						
				125 nmol l^{-1}	1[a]			~0.97[a]						
	Plasma	Rats	4 weeks	0.1 μg Se g^{-1} food		1[a]			0.11[b]	0.96[a]				Chansler et al. (1986)
		Human	6 weeks	~100 μg Se day^{-1}			1[a]			0.92[a]			0.89[b]	Thompson et al. (2008)
		Chicks	*9* days			*1*					*0.18*	*0.26*		Gabrielsen and Opstvedt (1980)
	Whole blood	Human	6 weeks	~100 μg Se day^{-1}			1[a]			1.20[b]			1.06[a]	Thompson et al. (2008)
	RBC	Rats	9 weeks	0.1 μg Se g^{-1} food		1*		~0.69*						Finley (1998)
	Liver	Rats	9 weeks	0.1 μg Se g^{-1} food		1*		~0.62*						Finley (1998)
		Rats	4 weeks	0.1 μg Se g^{-1} food		1[a]			0.05[b]	1.17[a]				Chansler et al. (1986)

Letters superscript indicates when difference between Se species is significant.
Results in italic have been calculated with slope-ratio method.
* Refers to results without indication on statistical significance.

during protein turnover, both able to induce toxicity (SCF, 2000). However, according to Rayman (2008), there is no evidence that this is the case since long term studies on Se supplementation with important doses (up to 800 μg Se day^{-1}) of Se yeast did not reveal toxic symptoms (Rayman, 2008). On the short term, it is generally accepted on the basis of animal studies that SeMet is less toxic than selenite (EFSA, 2009; Rayman, 2008; Schrauzer, 2000), at least at an intermediate level of intake. At very low and at highly excessive level of Se intake, there is no difference between the species, all of them inducing respectively no effect or acute toxicity. Toxicity of inorganic Se, especially in the selenite and selenide state, is believed to derive from its pro-oxidant activity on thiols, such as glutathione, leading *in fine* to the production of oxygen free radicals (Gad & Abd El-Twab, 2009; Mezes & Balogh, 2009). Methylated forms, on the contrary, are considered to be less toxic as they are easily excreted (Abdulah, Miyazaki, Nakazawa, & Koyama, 2005; Rayman, 2008).

In real foodstuffs, it has been historically stated that Se from animal sources is less available than Se originating from plants. However, it is not so easy to clearly classify Se bioavailability in complex matrices because many factors can come into the picture, e.g., Se species repartition, other food components, origin of the product. For example, several studies have put in light the high bioavailability of Se from wheat and recommended it as a good source of Se (Alexander et al., 1983; Douglass et al., 1981; Reeves et al., 2007). However, most of these studies have been conducted in the US where Se content of soils is high, such as Se content of the local wheat. In Europe, quantity and availability of Se in soil is globally lower than in America, with the consequence that wheat is not so rich in Se (Sager, 2006) so that it does not represent the major Se food source for European people. According to INRA (2004), majority of Se intake of the French population is provided by poultry, meat and mineral waters (INRA, 2004). Sea products are also globally rich in Se but bioavailability of the element has been reported to be either good or bad. The fish species and living environment of the animal play a role in Se bioavailability by influencing Se speciation and the presence/absence of components that enhance or decrease Se bioaccessibility. It was suggested that Se bioaccessibility in fish could be impaired by the presence of heavy metals, mainly mercury and arsenic, that bind very tightly with Se and make it unable to solubilise or bind anything else in the body (Bügel et al., 2001; Meltzer et al., 1993). It was also observed that part of the Se contained in fish can be present under a methylated form ($TMSe^{+}$) (Moreno et al., 2004; Quijano, Moreno, Gutierrez, Perez-Conde, & Camara, 2000) that is easily excreted and therefore not available for the organism.

In fact, the interest in a particular kind of food as Se source seems to depend on the effect criterium used. Some meat (Shi & Spallholz, 1994; Wen et al., 1997) or fish (Ornsrud & Lorentzen, 2002; Wen et al., 1997) products are able to increase efficiently GPx levels, which reinforce the antioxidant action in the body. Se yeast, on the other hand, is more efficient in favouring the storage of Se in tissues rather than in inducing selenoenzyme production (Rider et al., 2009, 2010). Se-enriched *Brassica* and *Allium* plants, finally, seem to be especially interesting for their specific anti-cancer properties by producing high quantities of MeSeCys and Glu-MeSeCys (Ogra et al., 2005). According to the needs or the desired action of Se, one or another kind of food will then be recommended.

5. Conclusions

Today determination of Se bioavailability and bioactivity in food has become a topic of great interest among researchers and nutritionists. The fact that the total Se content of a kind of foodstuff is not sufficient to predict its effect on human health makes the study of Se speciation as well as Se bioavailability and bioactivity a challenging research subject. Especially in the current context that encourages the resort to Se food supplements, it is crucial to broaden our knowledge about the efficacy or the potential toxicity of distinct Se species. On the one hand, it can be concluded with reasonable certainty that organic Se in the form of SeMet (found in cereal product or Se-enriched yeast, for example), is on the long term likely more efficient to prevent a Se deficiency, while inorganic Se in the form of selenite (found mostly in selenite-enriched vegetables or food supplements) can respond more rapidly to an acute need of Se but has a higher risk to become toxic on the long term. It is also recognised that *Brassica* and *Allium* plant products are interesting anticancer foodstuffs due to their high content in precursor species of methylselenol. On the other hand, despite the progress that has been made, many things remain unclear. The fact that large parts of Se in various foodstuffs are present under still unknown forms makes it difficult to conclude about their potential and actual health effects. Moreover, little is known about bioavailability of Se species other than SeMet and selenite. When additional forms could be identified and characterised, this would open the door for additional bioavailability and bioactivity studies that may reveal crucial information which is still lacking today.

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