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REVIEW

Exposure to toxic chemicals in the diet: Is the Brazilian population at risk?

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In Brazil, in the last 20 years, dietary risk assessments have been conducted on pesticides, mycotoxins, food additives, heavy metals (mainly mercury), environmental contaminants (mainly DDT) and acrylamide, a compound formed during food processing. The objectives of this paper were to review these studies, discuss their limitations and uncertainties and identify the most critical chemicals that may pose a health risk to Brazilian consumers. The studies have shown that the cumulative intake of organophosphorus and carbamate pesticides by high consumers of fruits and vegetables may represent a health concern (up to 169% of the ARfD), although the benefits of consuming large portions of those foods most probably overcome the risks. High consumers of maize products may also be at risk due to the presence of fumonisin (355% of the PMTDI), a mycotoxin present at high levels in Brazilian maize. The studies conducted in the Brazilian Amazon have shown that riparian fish consumers are exposed to unsafe levels of mercury. However, this is a more complex issue, as mercury levels in the region are naturally high and the health benefits of a fish-based diet are well known. Studies conducted both in Brazil and internationally on acrylamide have shown that the exposure to this genotoxic compound, mainly from the consumption of French fries and potato chips, is of health concern. Reducing the population dietary exposure to toxic chemicals is a challenge for government authorities and food producers in all countries. Management strategies aimed at decreasing exposure to the critical chemicals identified in this review involve limiting the use or eliminating highly toxic pesticides, implementing good agricultural practices to decrease maize contamination by fumonisins, educating local fish-eating communities toward a fish diet less contaminated by mercury, and changing dietary habits concerning the consumption of fried potatoes, the main processed food containing acrylamide.
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Introduction

Humans are constantly exposed to various hazardous chemicals present in the diet, and the assessment of the risks to health from this exposure is essential to support government actions aimed at guaranteeing a safe food supply for the population. The chemical dietary risk assessment process, introduced in the 1980s by the US National Research Council (NRC, 1983), has evolved rapidly over the last 10 years, mainly with regard to the toxicological and methodological aspects.

The first two steps of the risk assessment — hazard identification and hazard characterization — involve mostly animal data, but can also include information from human studies (Renwick et al., 2003; IPCS, 2009). While the hazard identification determines the nature of the adverse effect that a given chemical causes to an organism, system or population, the hazard characterization describes quantitatively the severity of this effect through dose/response relationships (IPCS, 2009). For non-genotoxic compounds, a health-based guidance value is determined, generally by applying a safety factor to the no-observed-adverse-effect-level found in the most critical animal study. Health-based guidance values include the acceptable daily intake (ADI), the acute reference dose (ARfD), the provisional maximum tolerable daily intake (PMTDI) and the provisional tolerable weekly intake (PTWI) (IPCS, 2009). Approaches to the hazard characterization of genotoxic and carcinogenic compounds (non-threshold response) include the mathematical modeling of the dose–response curve to estimate a low effect level (benchmark dose; BMD) or the carcinogenic potency (WHO, 1999; Dybing et al., 2008; Muri et al., 2009).

In the dietary exposure assessment step, the chemical intake is estimated by multiplying its level in the food by the amount of the food consumed *per* body weight (IPCS, 2009). Depending on the objectives of the assessment and the availability of data, two methods can be used to estimate the exposure. In the deterministic model, or point estimate, fixed values of concentration and consumption *per* body weight are used to calculate intake, such as the mean or a given

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percentile. In the probabilistic model, the concentration and consumption variables are described as distributions, and a statistical model, such as Monte Carlo, is used to generate an intake distribution and characterize its variability and uncertainty (Kroes et al., 2002; van Klaveren and Boon, 2009). Cumulative exposure to multiple chemicals with the same mechanism of action can be estimated using relative potency factors (RPF) in relation to an index compound (IC) (USEPA, 2006; Boobis et al., 2008).

In the risk characterization step of the risk assessment of non-genotoxic compounds, the exposure to a chemical is compared with its health-based guidance value; risk may exist when the exposure exceeds this value (IPCS, 2009). One approach to characterize the risk from the exposure to genotoxic carcinogenic substances is to calculate the margin of exposure (MOE), defined as the ratio between a toxicological reference (such as the BMD) and the estimated intake (Barlow et al., 2006; Benford et al., 2010). It has been hypothesized that an MOE of 10,000 or higher, if based on the BMDL10 (BMD lower confidence limit of a 10% response) from an animal study would be of low concern from a public health point of view (EFSA, 2005).

Brazil is one of the largest food producers in the world, supplying most of the dietary needs of its population. In this review, dietary exposure assessment studies conducted in the country on pesticides, mycotoxins, food additives, mercury, environmental contaminants and acrylamide, a substance formed during food processing, are presented and discussed. The objective is to identify and discuss their limitations and uncertainties with the aim of defining the main risks that the Brazilian population is exposed to in the diet.

Pesticides

The agricultural use of pesticides is still the most used strategy in food production worldwide. By 2010, over 400 pesticide active ingredients have been registered in Brazil (ANVISA, 2011a), one of the largest pesticide users in the world. The toxicity of these compounds, however, is not always restricted to the target pest organism, and has also been demonstrated in mammals, including humans (Belpoggi et al., 2002; Mendes et al., 2005).

One first-tier approach to estimate exposure to pesticide residues in food is to use the maximum residue limit (MRL), established by the government as a parameter for concentration in food, to calculate the national theoretical maximum daily intake (TMDI) (WHO, 1995). Brazilian MRLs are established based on supervised pesticide residue trials conducted throughout the country and reflect the good agricultural practices used nationally, according to the registered product labels specifications. The TMDI is highly conservative as it assumes that all individuals in a population will consume daily all the food for which there is an established MRL, that the residues will always be present at the MRL level, and that no decrease in the pesticide level will occur during storage, transportation and processing of the food. However, this first-tier approach is important to identify pesticide/commodity combinations of possible health concern and to help authorities set priorities to generate residue data to refine the estimation.

Caldas and Souza (2000, 2004) estimated the national TMDI using MRLs established by the Brazilian Government and consumption data estimated from a national household budget survey (HBS) (*Pesquisa de Orçamento Familiar* (POF); IBGE, 2010). In the 2004 study, the intake of eight of the 275 compounds evaluated (2.9%) exceeded the respective ADI in at least one Brazilian region (the organophosphorous (OP) insecticides prothiofos, ethion, fenitrothion, methidathion and dimethoate, the dithiocarbamate metam sodium, the acaricide dicofol, and the fumigant methyl bromide) (Caldas and Souza, 2004). Citrus, tomato and beans were the commodities that most contributed to the intakes. The intake estimations were refined for six compounds by replacing the MRL by the mean residues found in food commodities analyzed by the Brazilian Monitoring Program on Pesticide Residues (PARA) in 2664 samples collected from 2001 to 2003 (ANVISA, 2011a). Refined intakes ranged from $<1\%$ (dimethoate) to 90% (ethion) of the ADI, not indicating a health risk concern. The refinement was not possible for methyl bromide (8000% of the ADI) and fenitrothion (140% of the ADI) due to the lack of residue data.

Caldas et al. (2006a, b) applied the probabilistic approach to estimate the exposure of the Brazilian population to dithiocarbamates and the acethylcholinesterase (AChE) inhibitors OP and carbamate insecticides. Residue monitoring data from the PARA program for 4001 samples of nine fruit and vegetables (2001–2004), and additional residue data for rice and beans (dithiocarbamates only; Caldas et al., 2006a) were used to estimate the exposure. The chronic intake of dithiocarbamates did not represent a health risk to consumers at the higher percentiles of exposure (Caldas et al., 2006a). The cumulative acute intake of AChE inhibitors exceeded the ARfD at the 99.9th percentile of exposure (acephate as IC) or higher (methamidophos as IC) (Caldas et al., 2006b).

The intakes estimated in these studies probably underestimate the total exposure of the Brazilian population to pesticides in the diet as they include only a limited number of commodities. Currently, the PARA program analyses a total of 20 food commodities, including rice and beans, staple foods in the Brazilian diet. Additionally, the Ministry of Agriculture launched its own fruit and vegetable monitoring program in 2006. Between 2001 and 2010, over 13,000 samples of 22 food commodities were analyzed for pesticide residues by both programs (data not published), and this

Table 1. Probabilistic risk assessment of pesticides in Brazil and other countries.

Country	Compounds	Risk characterization at P99.9 of exposure		
		Children	General population	Reference
Brazil[a]	OPs and carbamates	169% ARfD[b] 80.2% ARfD[c]	70.2% ARfD[b] 33.6% ARfD[c]	Caldas et al. (2006a)
Denmark[d]	OPs and carbamates	31.3% ARfD[c] 1.8% ARfD[e]	13.8% ARfD[c] 0.83% ARfD[e]	Jensen et al. (2009)
The Netherlands[d]	OPs and carbamates	114% ARfD[b] 16.3% ARfD[f]	46% ARfD[b] 7.1% ARfD[f]	Boon et al. (2008)
USA[d]	OPs	23% ARfD[c]	11% ARfD[c] adults	USEPA (2006)
Brazil[a]	Dithiocarbamates	17.2% ADI[g,h]	8.8% ADI[g,h]	Caldas et al. (2006b)
Denmark[b]	Dithiocarbamates	1.5% ADI[g]	0.7% ADI[g]	Jensen et al. (2008)

[a]HBS data (*Pesquisa de Orçamento Familiar* – POF) used as food consumption.
[b]Acephate as index compound (IC).
[c]Methamidophos as IC.
[d]Individual consumption data.
[e]Chlorpyrifos as IC.
[f]Oxamyl as IC.
[g]ADI for mancozeb.
[h]30% of CS_2 residues are assigned to propineb.

larger residue data set will allow a more sound pesticide risk assessment in the future.

Table 1 summarizes the results of the probabilistic dietary risk assessments conducted on AChE inhibitors insecticides and dithiocarbamates in Brazil and other countries. The studies conducted on AChE inhibitors followed the approach used by the USEPA (2006), in which the equivalent AChE inhibitor residue level in a sample is calculated by multiplying the level detected by its RPF, and expressed as the IC. The risk characterization is performed by comparing the total intake with the IC ARfD. In addition to the limited residue database discussed previously, another major difference between the Brazilian and other studies shown in Table 2 is the source of the consumption data used in intake estimations. While individual consumption data were used in the United States, the Netherlands and Denmark, in the Brazilian studies, the food consumption data were estimated from HBS data (2002/2003 POF). The use of HBS data in dietary risk assessments presents some limitations, the main one being that the data reflect the food that is available in the household that will be consumed by the family members, not the food actually consumed by each individual. Also, real consumption levels may be underestimated by not considering outside consumption, or overestimated by not accounting for wasted food (Serra-Majem et al., 2003).

Results of the studies conducted in Brazil and Denmark shown in Table 1 illustrate the impact of the chosen IC on the cumulative acute risk assessment of OPs and carbamates. In the Brazilian study (Caldas et al., 2006b), the associated risk was approximately two times higher when acephate was used as the IC when compared with methamidophos. In Denmark, the risk when methamidophos was used as the IC was almost 20 times higher than that associated to chlorpyrifos (Jensen et al., 2009). In general, the selected IC in a cumulative assessment should have the largest available toxicological database of acceptable quality (USEPA, 2006), but these may differ, depending on the study. Furthermore, the uncertainties in the toxicological database used in selecting the IC and estimating the RPF should also be considered when evaluating the possible health impact from the exposure to AChE inhibitor pesticides (Bosgra et al., 2009). One limitation present in the dithiocarbamate dietary risk assessments regards the residue data, which refers to the CS_2 formed during the analytical procedure from any dithiocarbamate present in the sample. In order to compensate for this limitation, Caldas et al. (2006a) considered a more conservative situation, where 70% of the CS_2 detected in the sample came from mancozeb and 30% from propineb, a compound with a higher toxicity. In their assessment in Denmark, Jensen et al. (2008) considered only the use of mancozeb.

Dietary intake assessments can also be conducted using total diet studies (TDS). In a TDS, the actual dietary exposure is assessed by analyzing the chemical in all foods in a population diet that are in a ready-to-eat form, thus taking into consideration the impact of preparation and processing on the final chemical concentration (WHO, 2005). In a study conducted in a Brazilian university restaurant using a TDS deterministic approach, food meal samples were collected from the restaurant production line and analyzed for dithiocarbamate and organophosphorus pesticides (Caldas et al., 2011). Food consumption data and body weight information were obtained directly from the restaurant users. The chronic intake of dithiocarbamate represented $<10\%$ of the ADI (mancozeb) for both vegetarian and non-vegetarian users. However, the cumulative acute exposure to OP reached 116% of the ARfD (acephate as IC) for the vegetarians. Nevertheless, the authors pointed out that this exceedance might not be relevant in light of all the uncertainties involved in the estimation and the health benefits of consuming large portions of vegetables by vegetarians.

Table 2. Human exposure to aflatoxins and fumonisins in Brazilian food.

Mycotoxin	Food commodities, consumption data	Mean concentration, samples analyzed	Total daily intake, mean	Cancer risk or % PMTDI	Reference; region
Aflatoxins (B1, B2, G1, G2)	*Paçoca* and *pé-de-moleque*, 25 g/person, 50 kg bw	84 μg/kg, 92 samples	42 ng/kg bw	—	Caldas et al. (2002); DF
	Peanut and peanut products; 2002/2003 POF, 50 kg bw	6 μg/kg[a], 106 samples	0.23 ng/kg bw	—	Oliveira et al. (2009); SP
	25 g/person; 50 kg bw	6 μg/kg, 106 samples	3 ng/kg bw	0.036 cancers/year/10^5	This study
Fumonisins[b]	Corn meal, individual consumption data for rural population (highest level); 70 kg bw	2290 μg/kg, 9 samples (FB1)	1.3 μg/kg bw (RP)	—	Machinski and Soares (2000); SP
	Corn meal, individual consumption data (highest level); 70 kg bw	5200 μg/kg, 30 samples (FB1)	0.9 μg/kg bw (SP)	45%	Bittencourt et al. (2005), SP
			2.9 μg/kg bw (RP)	145%	
	Corn meal, 40 g/12 month child, 10 kg bw	1673 μg/kg, 89 samples (FB1)	6.7 μg/kg bw	335%	Castro et al. (2004), SP
	Corn products, 2002/2003 POF (consumption and bw)	Not reported, 534 samples (FB1 + FB2)	26 μg/person (Tp)	24.1%	Caldas and Silva (2007); Brazil
			376 μg/person (Co)	365%	

Abbreviations: Co, consumers only; RP, Brazilian rural population; SP, São Paulo state; Tp, total population.
[a]Mean of positive samples.
[b]PMTDI of 2 μg/kg bw for FB1, FB2, and FB3.

Mycotoxins

Mycotoxins are secondary metabolites produced by fungi that grow in food under certain conditions worldwide (WHO, 2002; Wagacha and Muthomi, 2008). The incidence of various mycotoxins in food in Brazil is well reported (Rodrigues-Amaya and Sabino, 2002; Caldas and Silva, 2007), but exposure assessment studies have only been conducted for aflatoxins and fumonisins.

Aflatoxins

Aflatoxins (AFB1, AFB2, AFG1 and AFG2; AFs) are mycotoxins produced by *Aspergillus* species that contaminate a variety of crops, mainly peanuts, tree nuts, dried fruit, spices, figs and maize. In Brazil, the highest incidence of aflatoxin contamination is found in peanuts and peanut products (Rodrigues-Amaya and Sabino, 2002; Oliveira et al., 2009). Dietary exposure assessments of AFs through the consumption of these products have been conducted in the country using monitoring data for samples collected in local commercial establishments. Caldas et al (2002) estimated that, in the Federal District area, the mean intake of aflatoxins from the daily consumption of *paçoca* and *pé-de-moleque*, peanut products popular among youths in the country, was 42 ng/kg bw/day. The mean AFs intake from the consumption of peanut and peanut products in the state of São Paulo (SP), estimated by Oliveira et al. (2009), was 0.23 ng/kg bw/day (Table 2). The large difference between these two estimates is due to the AFs concentration levels found in the samples and the consumption level used in the intake estimation. The mean AFs level (84 μg/kg) found in the Caldas et al. (2002) study (1998–2001) is much higher than that found more recently (2006–2007) by Oliveira et al. (2009) (6.0 μg/kg, mean of positive samples), probably due to the compulsory implementation of good manufacturing practices by Brazilian peanut industries since 2003 (ANVISA, 2011b). The consumption level assumed by Caldas et al. (one unit of peanut product, 25 g/person/day) considers only the population that consumes these products (consumers only), while the consumption level used by Oliveira et al. (2009), obtained from the 2002/2003 POF (1.9 g/person/day), is the mean consumption of peanut and peanut products for the entire POF survey population, which also considers non-consumers of these commodities. The latter consumption level probably underestimates the consumption of peanut products by children and teenagers. A more realistic estimation of exposure to AFs from the consumption of peanut products may be made if we assumed a contamination level of 6 μg/kg, as calculated by Oliveira et al. (2009), and a daily consumption of 25 g of peanut products (0.5 g/bw), as assumed by Caldas et al. (2002). Considering these levels, the AFs intake by Brazilian children and teenagers can be estimated as being 3 ng/kg bw/day.

Aflatoxins are a known genotoxic carcinogen to humans and are classified as group 1 compound by the International Agency for Research in Cancer (IARC), with the hepatitis B virus being an important risk factor for the development of hepatic cancer (IARC, 2011). There are no health-based guidance values for aflatoxins to compare with the estimated intake and risk characterization requires information on the prevalence of the hepatitis virus in the population. This assessment has not been conducted so far in Brazil.

The Joint FAO/WHO Expert Committee on Food Additives (JECFA) estimated the carcinogenic potency of aflatoxins for individuals with the hepatitis B virus ($HBsAg^+$) to be 0.3 cancers/year/100,000 individuals (P $HBsAg^+$), 30 times higher than the potency for non-infected individuals (0.01 cancers/year/100,000 individuals; P $HBsAg^-$) (WHO, 1999). The cancer risk estimated by the JECFA in Europe (1% of the population is $HBsAg^+$) from the intake of 0.32 ng/kg bw/day of aflatoxins was 0.0041 cancers/year/100,000 individuals, calculated from Eqs. (1) and (2) (WHO, 1999).

$$P_{estimated} = [P\ HBsAg^+ \times \%\ pop.\ HBsAg^+] + [P\ HBsAg^- \times \%\ pop.\ HBsAg^-] \quad (1)$$

$$\text{Cancer risk} = P_{estimated} \times \text{intake} \quad (2)$$

In Brazil, the prevalence of $HBsAg^+$ is estimated to be 0.6% (Pereira et al., 2009). Using the mean aflatoxin intake of 3 ng/kg bw/day calculated previously, we can estimate a population risk of 0.036 cancers/year/100,000 individuals for the country, almost 10 times higher than that estimated for the European population (as expected from an exposure $\sim$10 times higher) (Table 2). Although higher, the estimated Brazilian risk to aflatoxins from the consumption of peanut and peanut products is below what is considered the cancer risk associated with background exposure ($1/10^5$ or $1/10^6$ individuals). This estimation, however, needs to be refined based on real individual consumption data for peanut and peanut products and concentration data nationwide.

Aflatoxin M1 (AFM1) is a hydroxylated metabolite of AFB1 found in milk from animals consuming feed contaminated with AFB1, and also in human milk from exposed mothers (Oliveira et al., 2006; Gürbay et al., 2010). AFM1 is a genotoxic and carcinogenic compound classified as possibly carcinogenic to humans (group 2B) (IARC, 2011), and the exposure of newborn and children to this mycotoxin might be of health concern. Oliveira et al. (2006) estimated the AFM1 intake of 4-month-old children (assuming 6 kg bw) in the city of SP to be 3.7 ng/kg bw/day. More recently, also in SP, Shundo et al. (2009) estimated an intake of 1 ng/kg bw/day for children (assuming 400 ml consumption and 23 kg bw), and concluded that the occurrence of AFM1 in milk does not seem to indicate a health risk concern. In both studies, samples of whole milk powder consumed by infants at municipal schools and nurseries were analyzed. Oliveira et al. found much higher levels of AFM1 in milk using the immunoassay method (mean 0.27 μg/kg) than Shundo et al. (mean of 0.061 μg/kg), using the more selective HPLC/fluorescence method. The AFM1 levels found in both studies are lower than the maximum permitted level (ML) for AFM1 in milk set by Brazilian legislation and by the Codex Alimentarius, which is 0.5 μg/l (CODEX STAN 193, 1995). Currently, there is no methodology available for the characterization of the risks from the exposure to AFM1.

Fumosinins

Fumonisins are mycotoxins produced mainly by *Fusarium* sp and are found worldwide mostly in maize and maize products; the levels found in Brazilian maize are reported to be higher than the levels found elsewhere (CODEX, 2009). Fumonisins are classified as possibly carcinogenic to humans (group 2B) (IARC, 2011) and human dietary exposure to fumonisins has been associated with esophageal cancer (Sun et al., 2007) and neural tube defects (Gelineau-van Waes et al., 2009). A PMTDI of 2 μg/kg bw/day was allocated by the JECFA to FB1, FB2 and FB3 alone or in combination (WHO, 2002).

Machinski and Soares (2000) estimated a maximum daily intake of 1.3 μg/kg bw FB1 from the consumption of corn meal by the Brazilian rural population (high consumers) using contamination data from samples collected at commercial establishments in Campinas (SP), individual consumption data reported by the IBGE in 1977, and assuming 70 kg body weight for adults (Table 2). Using the same consumption data and bw, and also laboratory data, Bittencourt et al. (2005) estimated a daily intake of 0.9 μg/kg bw FB1 by the population of the city of SP, and of 2.9 μg/kg bw by the rural population, exceeding the fumonisin PMTDI by 45%. Castro et al. (2004) analyzed 89 samples of corn meal, and assuming a daily consumption of 40 g, the authors estimated that 12-month-old babies in the state of SP (10 kg bw child) were exposed to 6.7 μg/kg bw/day of fumonisins, exceeding 3.4 times the PMTDI (Table 2). However, the daily consumption of corn meal products by infants used in this study may have overestimated the intake. According to data from the 2002/2003 POF, the household availability of corn meal, corn starch, corn flower and corn flakes in the state of SP is, on average, 3.7 g/person/day total (IBGE, 2010).

Caldas and Silva (2007) estimated the intake of FB1 + FB2 by the Brazilian population from the consumption of maize products, including meal, breakfast cereals and popcorn, using consumption and body weight data from the 2002/2003 POF and contamination data from samples collected in the Federal District and in the states of SP, Santa Catarina and Pernambuco. The total intake represented 24% of the PMTDI for the total population, which considered all the households surveyed by the POF, and 355% PMTDI for the consumer-only population (Table 2).

This higher exposure level mostly refers to certain groups of the population, such as those with low availability of other sources of carbohydrates, local maize producers and individuals with gluten tolerance.

The co-exposure to aflatoxins, a genotoxic carcinogenic substance, and fumonisins, a possible carcinogen, may represent an additional risk to humans. In a study conducted in the state of Parana (PR), in Southern Brazil, all 300 maize samples analyzed were contaminated with fumonisins and 8% also contained aflatoxins (Moreno et al., 2009).

Food Additives

Food additives are substances intentionally added to food for a technological purpose in the manufacture, processing, preparation, treatment, packing, packaging, transport or holding (IPCS, 2009). They include preservatives, sweeteners, coloring and antioxidant agents.

Preservatives

Preservatives are substances added to food to prevent the growth of bacteria, yeast and mold. The intake of the preservatives benzoates and sulfur dioxides contained in wines and fruit juices has been associated with triggering asthmatic responses in some studies (Freedman, 1997; Vally and Thompson, 2003).

Tfouni and Toledo (2002) estimated that the intake of benzoates (ADI of 5 mg/kg bw/day) and sorbates (ADI of 25 mg/kg bw/day) by high consumers of soft drinks, juices, margarine, yogurt and cheese in Campinas (SP) contributed to a maximum of 54% and 4% of the respective ADIs (Table 3). The authors used consumption data from the 1995/1996 POF and from a market survey (Brasil Trend'99), concentration data generated in laboratory (56 samples) (Table 3). When the estimation was refined for benzoates in soft drinks using individual consumption data, the intake for the average (259 ml) and the high consumer (2 l) were 22% and 172% of the ADI, respectively (Table 2). These results show that POF data indeed underestimated the consumption of soft drinks, probably by not accounting for outside consumption.

Popolim and Penteado (2005) estimated the exposure of high-school students in the state of SP to sulfites, compounds with antioxidant and preservative properties, using consumption data from a 24-h dietary recall survey among 176 students and Brazilian MLs as concentration parameter. The intake represented, on average, 10% of the ADI (0.7 mg SO_2/kg bw/day) (Table 3). Highly exposed consumers (intake $>50\%$ ADI) accounted for 4.5% of the interviewed students, mainly due to the consumption of fruit juices and alcoholic beverages. Machado (2007) estimated that the mean and the 97.5th percentile of exposure of 11- to 17-year-old Brazilians to sulfites present in fruit juices represented 36% and 94% of the ADI, with high consumers of mango juices being potentially at risk of exceeding the ADI. Consumption data from 140 fruit juice consumers in a 24-h dietary recall study with 578 adolescents and mean sulfite concentrations found in 35 out of 39 juice samples analyzed were used in the estimation (Table 3). Moreover, Machado et al. (2009) estimated that the mean and maximum intakes of sulfites by a 60-kg person from the consumption of 150 ml of wine with sulfite concentrations found in 72 wine samples analyzed represented 36% and 84% of the ADI, respectively. The profile of sulfite exposure found in Brazil was similar to that in Belgium, where the sulfite intake, estimated using individual food consumption data and laboratory data or MLs, corresponded to 27% of the ADI, with high consumers of wine having an intake around the ADI (Vandevijvere et al., 2010).

Sweeteners

Saccharin, cyclamate and aspartame have been used in the food industry as sugar substitutes for at least 30 years, and are known as the first generation sweeteners (Weihrauch and Diehl, 2004). Saccharin, the oldest among them, can cause bladder cancer in rats, but the cancer-inducing mechanisms do not apply in humans (Cohen et al., 1998). Saccharin, as well as cyclamate, is not classifiable as to its carcinogenicity to humans (group 3) (IARC, 2011). Soffritti et al. (2006) have shown that aspartame is a multipotential carcinogenic compound in laboratory animals, whose carcinogenic effects are evident even at a daily dose of 20 mg/kg bw. However, its potential to cause cancer in humans has not yet been considered by the IARC.

Toledo and Ioshi (1995) estimated the intake of the three sweeteners using food consumption data obtained from a Food Frequency Questionnaire (FFQ) with 673 respondents in the cities of Campinas (SP) and Curitiba (PR). Concentration levels were determined in laboratory for beverages and table-top sweeteners, and obtained from the product labels of other food commodities, such as jellies and yogurts. The mean intake represented $<20\%$ of the respective ADIs (Table 3), but the intake exceeded the cyclamate ADI (11 mg/kg bw/day) for six consumers, the saccharin ADI (5 mg/kg bw/day) for two consumers and for both compounds for eight consumers. The consumption of table-top sweeteners contributed the most to the total intake, followed by beverages; diabetics were more likely to exceed the ADI in their diet. These results supported the decision made by ANVISA to decrease the Brazilian ML in beverages for cyclamate (from 1300 to 400 mg/l) and saccharin (from 300 to 150 mg/l) (RDC 18/2008; ANVISA, 2011b) and guarantee that the intake would be below the ADI even for high consumers of food containing these compounds.

A similar experience occurred in Europe. A study conducted in Denmark in 1999 found that many of the

Table 3. Human exposure to food additives in Brazilian food.

Food additive	Food commodity; consumption data[a]	Mean concentration, samples analyzed	Total daily intake, mg/kg bw	% ADI	Reference; region
Benzoates	Soft drinks, juices, margarine, yogurt and cheese; 1995/1996 POF[b] and Brazil Trend'99[b,c], 60 kg bw	Not reported, 56 samples	0.31 (POF)[b]	18%	Tfouni and Toledo (2002); SP
			0.90 (BT)[b]	54%	
Sorbates		Not reported, 56 samples	0.17 (POF)[b]	2.1%	
			0.33 (BT)[b]	4.0%	
Benzoates	Soft drinks; individual consumption data[d]	259.2 mg/l, 15 samples	1.1 (Mean)	22%	
			8.6 (High consumers)	172%	
Sulfites	All 33 food items in which sulfites are allowed; 24-h recall[e]	Brazilian MLs (0.02–1.5 mg/g or ml)	0.07 (Mean)	10%	Popolim and Penteado (2005); SP
			0.52 (Max)	74.3%	
	Fruit juices; 24-h recall[f]	34.1 mg/l[g], 39 samples	0.25[g,h] (Mean)	35.7%	Machado (2007); SP
			0.66 (97.5th P)	94.3%	
	Wine; 150 ml, 60 kg bw	98.5 mg/l	0.25 (Mean)	35.7%	Machado et al. (2009); SP
		235 mg/l (max), 72 samples	0.59 (Max)	84.2%	
Saccharin	Beverages, table-top sweeteners and other food; 24-h recall[i]	Not reported MLs	0.82 (Mean)	16.4%	Toledo and Ioshi (1995); SP
			10.8 (Max)	216%	
Cyclamate			1.7 (Mean)	15.4%	
			17.9 (Max)	163%	
Aspartame			1.2 (Mean)	3%	
			18.8 (Max)	47%	
Amaranth	Jellies, juice, soft drink, syrup, candy; FFQ/individual interview[j]	Not reported, 140 samples	Not reported	8–38%	Toledo et al. (1992); SP
Sunset yellow				1–5%	
Tartrazine				0.2–0.7%	
Indigotive				0–0.4%	
BHA	Oils, margarine; 1995/1996 POF and Brazil Trend'99[c], 60 kg bw	Brazilian MLs	0.09 (POF) (mean)	18%	Maziero et al. (2001)
			0.15 (BT) (mean)	30%	
BHT			0.05 (POF) (mean)	16.7%	
			0.10 (BT) (mean)	33.3%	
TBHQ			0.07 (POF) (mean)	10%	
			0.12 (BT) (mean)	17.1%	

Abbreviations: BHA, butylated hydroxyanisole; BHT, butylated hydroxytoluene; ML, maximum permitted level in the food; TBHQ, TBHQ.
[a]Consumption and body weight were given when not based on data but assumed by the authors.
[b]High consumers = 3 × mean.
[c]Based on market survey.
[d]From 600 individuals in Campinas (SP), 9–80 years old.
[e]With 176 high-school students (14–19 years old) in São Caetano do Sul (SP).
[f]With 578 adolescents (11–17 years old) in Piracicaba (SP).
[g]Mean of 35 positive samples.
[h]Only considering the 140 fruit juice consumers.
[i]With 673 individuals in Campinas (SP) and Curitiba (PR).
[j]Food frequency questionnaire (FFQ) over a 2-week period with 242 children at the household (3–14 years old) and interview with 428 children at the school to detect candy consumption, in Campinas (SP).

116 samples of non-alcoholic beverages analyzed had cyclamate levels close to the ML of 400 mg/l (Leth et al., 2007). Using individual consumption data, the authors estimated that the 90th percentile of exposure to cyclamate for 1- to 3-year-old children was close to the ADI value of 7 mg/kg bw/day; the 99th percentile for 1–10 year olds far exceeded the ADI value. These results led the Government of Denmark to argue at the European Commission for a lower ML for cyclamate in beverages, and in January 2004, the permitted level was reduced to 250 mg/l (Leth et al., 2007). It should be pointed out that the ADI for cyclamate used in this study, and recommended by the European Commission, is lower than that current JECFA ADI (11 mg/kg bw/day).

Food Coloring

A recent randomized, double-blinded study conducted in the United Kingdom demonstrated that artificial food coloring agents or sodium benzoate (or both) in the diet result in increased hyperactivity in children and in the general population (McCann et al., 2007). This study prompted the European Commission to require that food containing one or more of the food colorings tested (sunset yellow, carmoisine, tartrazine, ponceau 4R, allura red and quinoline yellow) should be labeled to indicate that they "may have an adverse effect on activity and attention in children" (EC, 2008). This association, however, is still controversial (Connolly et al., 2010; FDA, 2011). Tartrazine has been described as also being responsible for triggering attacks of urticaria and asthma (Freedman, 1997; Nettis et al., 2003). However, these reactions may be overestimated, and the pathogenic mechanisms involved remain poorly understood (Elhkim et al., 2007).

The intake of artificial food coloring agents by 3- to 14-year-old children living in Campinas (SP) was estimated using food consumption data from a FFQ conducted over a 2-week period in the households of 242 children and individual interview with 428 children in the school, with concentration levels determined in laboratory (Toledo et al., 1992) (Table 3). The total intake represented a maximum of 38% of the ADI for amaranto (0.5 mg/kg bw/day), 5% ADI for sunset yellow (2.5 mg/kg bw/day), 0.7% ADI for tartrazine (7.5 mg/kg bw/day) and 0.4% ADI for indigotine (5 mg/kg bw/day), with male low-income children having the highest intake. The results indicate no health concern from the exposure to these food coloring agents by children. In France, the intake of tartrazine by children, calculated using ML and individual consumption data, also did not represent a health concern, accounting for 37.2% ADI at the 97.5th percentile of consumption (Elhkim et al., 2007). In Kuwait, however, the intake of tartrazine, sunset yellow, carmoisine and allura red by 5- to 14-year-old children was identified as exceeding their ADIs by factors of 2–8 (Husain et al., 2006). The authors used 24-h dietary recall on 3141 children from 58 schools and the determination of the coloring agents in 344 foods items consumed.

Antioxidants

Butylated hydroxyanisole (BHA), butylated hydroxytoluene (BHT) and tert-butyl hydroquinone (TBHQ) are phenolic antioxidants used in products containing fats or oils, alone or in conjunction for a synergistic effect. BHA and BHT are classified by the IARC as group 2B and group 3, respectively, and TBHQ has not yet been considered by the Agency (IARC, 2011).

Maziero et al. (2001) estimated the intake of the phenolic antioxidants through the consumption of oils and margarines. Consumption data were obtained from the 1995/1996 POF and from the Brasil Trend'99 survey and MLs were used as concentration. The intake represented a maximum of 33.3% of the ADI (Table 3). Analytical determinations in selected food categories showed that BHT and TBHQ concentrations were below the respective MLs, and BHA was not detected in any of the analyzed samples. Based on the conservative approach and on the analytical data, the authors concluded that it is unlikely that the ADI for BHA (0.5 mg/kg bw/day), BHT (0.3 mg/kg bw/day) and TBHQ (0.7 mg/kg bw/day) would be exceeded by the average Brazilian consumer through the consumption of oils and margarines.

Mercury

All humans are exposed to some low levels of mercury, by either inhalation, ingestion or dermal contact. The factors that determine the occurrence and severity of adverse health effects from the exposure to mercury include its chemical form and the age or developmental stage of the person exposed; the fetus is considered to be the most susceptible segment of the population (WHO, 2008). Methylmercury (MeHg) has long been known to affect neurodevelopment in both humans and experimental animals (WHO, 2004; Castoldi et al., 2008a, b), having a PTWI of 3.3 μg/kg bw (0.47 μg/kg bw/day) for the general population and 1.6 μg/kg bw (0.23 μg/kg bw/day) for women of child-bearing age (WHO, 2004). The previous PTWI for total mercury (THg) (5 μg/kg bw; 0.71 μg/kg bw/day) was recently withdrawn by the JECFA (FAO/WHO, 2010).

The Brazilian Amazonian soil is naturally rich in mercury and soil erosion, intensified by deforestration, biomass burning and gold mining, having been shown to increase the mercury burden in local aquatic systems (Berzas Nevado et al., 2010). Fish consumption is the main source of MeHg and, on average, at least 90% of THg found in the Amazonian fish is present as MeHg (Malm et al., 1995; Kehrig et al., 1998), with the carnivorous fish having the highest concentrations (>10 μg/g MeHg) (Dórea and

Barbosa, 2007; Berzas Nevado et al., 2010). Two recent reviews reported mean hair THg levels in the Brazilian Amazon basin population ranging from 3.3 to 38.6 μg/g (Passos and Mergler, 2008; Berzas Nevado et al., 2010), higher than the levels found in the other South American Amazon basin populations (4.9–11.4 μg/g) (Passos and Mergler, 2008). Kehrig et al. (1998) estimated that over 90% of THg in hair is in the MeHg form.

Passos and Mergler (2008) conducted an extensive review of the neurobehavioral studies in Amazonian fish-eating communities, showing that child populations with a higher mean hair THg level (>10 μg/g) tended to show a significantly lower performance in certain tests (manual dexterity, fine motricity, digit span, visual-spatial functions and leg coordination). However, according to the JECFA, there is no consistent evidence of neurodevelopmental effects in children of women whose MeHg intakes had resulted in burdens of THg in hair of 20 μg/g and below (WHO, 2004).

Many studies have been conducted to evaluate the exposure of the Brazilian Amazon basin population to mercury (Table 4). In general, they involve the analysis of THg or MeHg in fish and consumption data estimated or collected among the local population. Some studies have estimated mercury intake based on mercury concentration found in hair of the affected population using kinetic models (Boischio and Henshel, 1996; Kehrig et al., 1998). The mean THg levels found in the studies were within a small range (0.24–0.39 μg/g), and do not seem to have changed considerably over the last 10–15 years. Mean THg intake estimations for adults and the general population ranged from 0.48 to 2.6 μg/kg bw/day, 67% to 3.7 times higher than the previous JECFA safe level (0.71 μg/kg bw/day). Boischio and Henshel (1996) estimated that children from the Alto Madeira region (<5 years old) have a mean THg intake of 6.4 μg/kg bw/day (Table 2), with 60% of this subpopulation being at risk of neurological damage from mercury exposure.

Sanga et al. (2001), using a two-dimensional Monte Carlo analysis, compared the predictive model estimates of dietary MeHg exposures using dietary recall and biomarker data from fish-eating populations in Brazil (Alto Madeira) and other countries. The mean MeHg intakes by the Alto Madeira population were 453 and 509 μg/day for the dietary recall and biomarker (hair) models, respectively, corresponding to 6.5 and 7.3 μg/kg bw/day for a 70-kg bw person (Table 4). Using the dietary recall model, the authors estimated that 42–67% of the fish-eating Brazilian population exceeded the safe exposure level for MeHg of 0.47 μg/kg bw/day set by the USEPA in 1997, the same as the current JECFA safe level. When the biomarker model was used, this estimation ranged from 28% to 75%, suggesting less precision than the dietary model. The estimated intake (dietary recall) found for the Alto Madeira

Table 4. Dietary intake exposure of the Brazilian Amazonian population to mercury (Hg) or methylmercury (MeHg) from fish consumption.

Location, mercury species	Daily intake	Mercury level, μg/g	Mean fish consumption, g/day; body weight	Reference
Alto Madeira, RO; THg	2.2 μg/kg bw (Total popul.)	Fish: 0.36; $N=245$	200; 35.5 kg	Boischio and Henshel (1996)
		Hair: 17.2; $N=237$		
	1.2 μg kg bw (15–48 years)	Hair: 14.7; $N=57$	53.5 kg	
	6.4 μg kg bw (<5 years)	Hair: 18.3; $N=31$	12.1 kg	
Tapajos River, PA; THg	0.48 μg/kg bw	Fish: 0.24[a] (0.06 NC; 0.42 C); $N=238$	200; 70 kg	Bidone et al. (1997)
Balbina Reservoir, AM; MeHg	35.2 μg/person 0.50 μg/kg bw[b]	Fish: 0.24 (0.03–0.09); $N=32$ Hair: 8.76; $N=20$	110	Kehrig et al. (1998)
Alto Madeira, RO; THg	2.6 μg/kg bw	Fish: 0.39 (nd-11.5); $N=576$	243; 36 kg	Boischio and Henshel (2000a, b)
Alto Madeira, RO; MeHg	453 μg/person 6.5 μg/kg bw	Fish: 0.38 ± 0.84	200; 70 kg	Sanga et al. (2001)
	509 μg/person 7.3 μg/kg bw	Hair: 13.7 ± 9.0 (Hg)		
Tapajos River, PA; THg	0.92 μg/kg bw 0.59 μg/kg bw[c]	Fish: 0.33 (0.11 NC; 0.52C); $N=1123$	141 (115–171); 57 kg	Passos et al. (2008)

Abbreviations: C, carnivorous; NC, non-carnivorous.
[a]95th percentile upperbound estimate of the mean.
[b]Assuming a 70-kg bw.
[c]Median.

population was about one-third lower than that found in Sweden (1580 μg/day), but much higher than in the United Kingdom (74 μg/day) and Bangladesh (9 μg/day).

Persistent Organochlorine Compounds

The use of persistent, fat soluble organochlorine compounds (POCs), including the insecticide 1,1,1-trichloro-bis-2,2-(4-chlorophenyl) ethane (DDT) and the industrial chemicals polychlorobiphenyls (PCBs), was prohibited in most countries in the 1970s and 1980s due to their bioaccumulation in the food chain (Ueno et al., 2003). POCs are endocrine disrupters in humans and other species, and their presence in various environmental compartments has been detected around the world (Ueno et al., 2003; den Hond and Schoeters, 2006).

Caldas et al. (1999) evaluated the exposure of the Brazilian Federal District population to DDT, its metabolites ($p,p'-DDD + p,p'-DDE$), and other persistent organochlorines through the consumption of fish from the Paranoá Lake. DDTs were detected in 95% of the 120 fish samples analyzed; the maximum intake (daily fish consumption of 500 g and 60 kg body weight) represented 0.32% of the ADI of 20 μg/kg bw/day, established by the WHO in 1994. The intake of heptachlor epoxide, detected in 10% of the samples analyzed, represented a maximum of 5% of its ADI. PCBs, endosulfan, endrin or aldrin were not found above the detection limit in any of the samples analyzed. Although low, these are overestimations of the intake, as 500 g daily fish consumption is much higher than the expected consumption by the Brazilian population.

Azevedo e Silva et al. (2007) found PCBs and DDTs ($o,p'-DDE + p,p'-DDE + o,p'-DDT + p,p'-DDT$) in all 22 muscle tissue samples of swordfish and blue shark captured on the Brazilian coast. The intake of DDTs by the general population (17 g fish/person/day) and by the fishermen and their families (200 g fish/person/day), assuming a 60-kg body weight, was considered to be safe, as it contributed to $<0.1\%$ of the WHO ADI.

Cidade dos Meninos, located in the state of Rio de Janeiro, is a known POC-contaminated area. A factory set up in 1950 for the production of hexachlorocyclohexane (HCH) and the formulation and storage of DDT and other pesticides closed its operations in 1961 and the remaining production was left in the open air. In a study conducted in 2001, Asmus et al. (2008) estimated the intake of POCs by the local population through the consumption of eggs and milk. The authors assumed a daily consumption of one egg and of 100 g milk by adults (70 kg), and of 200 g milk for children up to 11 years of age (30 kg). The intake of DDTs and HCHs exceeded the respective safe doses for both populations; for dioxins and furans, the additional cancer risk was estimated to be 0.035%. However, the authors recognized that the assessment of the effects of these compounds on this population's health was still inconclusive.

The use of DDT to control vector-borne diseases such as malaria was intense in the Brazilian Amazon region until the end of the 1990s, being sprayed on the walls of the houses in critical areas. Azeredo et al. (2008) estimated the intake of DDT and its metabolites through the breast milk of fish-eating mothers in the Madeira River basin. All 69 milk samples analyzed contained DDTs, ranging from 0.025 to 9.36 μg of total DDT/g of lipid (median = 0.369 μg/g of lipid). For 8.7% of the breast-feeding infants, the intake (1 l milk/day, 5 kg bw) exceeded the DDT WHO ADI (20 μg/kg bw/day). However, the authors emphasize that breast feeding should not be discouraged among this population since it warrants the most complete nutrient supply to children.

Acrylamide

Acrylamide is an important industrial chemical whose neurotoxicity in humans is well known from occupational and accidental exposures. Animal studies have shown its reproductive, genotoxic and carcinogenic properties (WHO, 2006a, b) and acrylamide is classified as probably carcinogenic to humans (group 2A) (IARC, 2010). Studies conducted in Sweden in the early 2000s showing the presence of acrylamide in food processed at high temperatures (Tareke et al., 2002) raised concerns about the dietary exposure of the general population to acrylamide.

Arisseto and Toledo (2008) and Arisseto et al. (2009) estimated the acrylamide exposure of the Brazilian population from the consumption of various foods, including French fries, cassava products, bread and coffee. Food samples (74) collected in Campinas (SP) was analyzed in the laboratory. In the first study (2008), total mean consumption of the critical food was obtained from the 2002/2003 POF and the national estimated intake of acrylamide was 0.14 μg/kg bw/day (total population). Furthermore, the authors refined this estimation for teenagers (11–17 years old) using individual consumption data obtained through a 24-h recall study with 464 individuals (Arisseto et al., 2009). The mean and the 97.5th percentile of exposure were 0.12 and 0.78 μg/kg bw/day, respectively; the consumption of French fries contributed the most to the intake (66% among the girls), confirming the data found in other countries (WHO, 2006a, b).

In the international dietary risk assessments of acrylamide conducted by the JECFA, the estimated intake was compared with the BMDL10 of 0.18 mg/kg bw/day for harderian gland tumors in mice (WHO, 2006a, b; FAO/WHO, 2010). The calculated MOE values (BMDL10/intake) were 180 and 45 for the mean (1 μg/kg bw/day) and high exposures (4 μg/kg bw/day), respectively. The Committee considered that for a compound that is both

genotoxic and carcinogenic, these MOEs indicate a health concern. Using the intakes estimated by Arisseto et al. (2009), we found that the MOEs for the mean (0.12 μg/kg bw/day) and high exposure (0.78 μg/kg bw/day) for the teenager population of Campinas were 1500 and 231, respectively. Although these are higher than the MOEs estimated at the international level by the JECFA, they also indicate a health concern.

Discussion and Conclusions

The quality of the information obtained from dietary risk assessments depends highly on the quality and uncertainties of the data used in each step of the process. Uncertainty occurs because of a lack of knowledge and can often be reduced by obtaining more and better data. Uncertainties may also arise from hazard characterization (in setting ADI or cancer risk, for example) and/or the intake estimation step of the risk assessment (IPCS, 2009). Hazard characterization of chemicals is normally performed by national and/or international authorities and, in general, the uncertainties involved are not available to researchers; hence, they will not be discussed here.

Uncertainty concerning the chemical concentration within the intake estimation is related to the data source (legal limits, label information or laboratory data), the food analyzed (raw commodity or ready-to-eat food), sampling protocols (if the sample is representative of the population sampled), the number of samples analyzed and the analytical method used (sensitivity, precision and accuracy) (IPCS, 2008).

When the intake estimate is conducted using legal limits or label information, the final estimate usually tends toward the conservative end of uncertainty. Although the result does not reflect real exposure, it can be useful in government decision-making, such as the approval of a pesticide or food additive and the prioritizing of monitoring programs when the conclusion of the assessment indicates a possible health concern.

Uncertainties in food consumption data are related to the type of data (e.g. HBS or individual data), the number of individuals surveyed, and whether the surveyed population can be extrapolated to the rest of the population. One major limitation found in the Brazilian studies, mainly those conducted on pesticides and mycotoxins, was the consumption data used to estimate the intake, based mostly on the HBS (POF) data. These data reflect the amount of food available in the household, and individual consumption is estimated by dividing the amount of food available by the number of individuals in the household. As food consumption patterns vary considerably with age and also with sex (Muñoz et al., 1997; McLennan and Podger, 1999; AUS, 2008), that approximation may underestimate the consumption of certain foods by certain population groups, further underestimating their exposure to certain chemicals. This limitation is partially being resolved by the 2008/2009 POF, where individual consumption data were generated for individuals 12 years of age or older (IBGE, 2010). However, individual consumption data for infants and children are still lacking in the country, limiting a more sound assessment for this group of the population.

Limitation of food consumption data designed to conduct dietary exposure assessment studies is a common problem encountered in many other countries, as acquiring these data demand significant financial and human resources. Countries that still lack food consumption data may rely on the 13 GEMS/Food Consumption Cluster Diets, used by the JMPR and JECFA for their international exposure assessments. The Cluster Diets were derived from the FAO Food Balance Sheet data of 183 countries (WHO, 2006b), and although very limited, it can be used by national authorities as a first tier to estimate exposure. According to Pomerleau et al. (2003), FAO data tend to overestimate the consumption of fruit and vegetables.

The impact of the data quality on the final intake estimation can be evaluated from the two mercury studies conducted along the Tapajos River (Table 2). In the first study (Bidone et al., 1997), 238 fish samples from 15 species were analyzed for THg content, and 200 g fish consumption and 70 kg body weight were assumed in the estimation. In the study conducted by Passos et al. (2008), 1123 fish samples from 24 species were analyzed and consumption and body weight data were obtained from a 7-day recall FFQ conducted with 256 individuals over the age of 14. The estimated daily intakes were 0.48 and 0.92 μg/kg bw, respectively. It is reasonable to conclude that the uncertainties regarding the latter estimation are smaller, and that the uncertainties regarding the first estimation are mostly due to the fish consumption and body weight, information that were not derived from real data.

In spite of all the uncertainties involved in the estimations, some conclusions on the dietary risk of the Brazilian population to chemicals can be drawn from the studies reviewed in this paper. Concerning the pesticides, the intake of AChE inhibitors pesticides by high consumers of fruits and vegetables might be of health concern. However, we believe that the risks are relatively small compared with the health benefits of consuming large portions of fruits and vegetables. Moreover, the identified risks indicate the need to eliminate or decrease the use of these compounds in the country. Indeed, recent government decisions point in this direction, and methamidophos registration was recently canceled in Brazil (ANVISA, 2011a). Other countries have also found that certain AChE inhibitors pesticides can be of health concern, and have taken similar actions toward restricting or banning their use, including the United States (USEPA, 2010).

High consumers of peanut and maize products might be at a higher cancer risk due to the chronic exposure to aflatoxins

and fumonisins. Although aflatoxin contamination in Brazilian food is somewhat under control, and current exposure does not contribute significantly to the baseline cancer risk, the fumonisin levels in Brazilian maize are among the highest worldwide (Codex, 2010). This problem was recently identified by the Brazilian health and agricultural authorities, leading to the established of an ML of 5 mg/kg for fumonisins (FB1 + FB2) in maize grain, which should be complied with by producers by 2014 (ANVISA, 2011b). This long-term management strategy will not immediately affect the maize supply and the production chain in the country, while the producers take action to reduce the fumonisin contamination level. This strategy is in accordance with the Codex management principles for food safety to be implemented by governments (Codex, 2007) and may be adopted by other countries that identify similar problem in their food supply.

In general, food additive exposure is higher in industrialized countries and can exceed the ADI for individuals with high consumption of some specific products (wine and fruit juice containing sulfites, for example), and for specific groups of the population (intake of sweeteners in soft drinks by children). Specifically for children, the strategy used in Brazil and elsewhere has been to decrease the ML of certain food additives. However, it is also important to educate mothers to decrease the consumption of these industrialized products by their children.

The studies discussed in this paper show that the mercury intake by the Brazilian Amazonian population exceeds the safe exposure level and may represent a risk to health, mainly for newborns and children of exposed mothers. However, the nutritional benefits of fish consumption need to be weighed against the possibility of adverse effects (WHO, 2004). Grotto et al. (2010) found an association between the levels of mercury in hair and blood and oxidative stress in fish-eating Amazonian communities; nevertheless, they also found that fish consumption appears to have a beneficial effect regarding the same end point. Furthermore, Passos et al. (2007b) have shown an association between fruit consumption and lower hair and blood mercury levels in a study conducted among 13 riparian Tapajos River communities. Many researchers agree that one strategy to decrease mercury exposure without drastically changing the population's diet would be to educate the local population to change dietary habits toward the preferential consumption of the less-contaminated fish species (non-carnivorous) (Boischio and Henshel, 2000b; Mertens et al., 2005). Clearly, this strategy is essential for women of child-bearing age. Boischio and Henshel (2000a) have shown a direct correlation between the maternal THg hair and breast milk THg concentrations. However, to be successfully implemented, any educational program needs to involve researchers, government, as well as local authorities and community leaders.

Acrylamide exposure, mainly from potato product consumption, is also a health concern in Brazil and other countries. However, there is no global consensus regarding the management strategies to decrease this exposure. Approaches could include educational campaigns among the population aimed at controlling the degree of home-made fried or baked potatoes (lighter potatoes have lower acrylamide levels), the development of potato varieties that have lower levels of acrylamide precursors (asparagine and reducing sugar), and the introduction of asparaginase, the enzyme that degrades the precursor, during food processing by the industry (WHO, 2006a, b). Vinci et al. (2010) have shown that color measurements after blanching and two-stage frying or measuring reducing sugars in the incoming product are possible preventive tools for the French fries industry to reduce acrylamide at the start of production. In any case, it is unlikely that these strategies will be implemented in the near future, as they represent a drastic change in food production and in the dietary habits of certain groups of the population, who are not actually aware of the risks they are exposed to.

Decreasing the population's exposure to toxic chemicals in the diet is a challenge for government authorities and food producers worldwide. As the importance of the dietary risk assessment process is being increasingly recognized by scientists and managers, it is expected that the limitations pointed out in this paper will be overcome and more sound dietary risk assessments may be conducted in Brazil and other countries facing the same problems.

Conflict of interest

The authors declare no conflict of interest.

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