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Reassessment of the acrylamide risk: Belgium as a case-study

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ABSTRACT

Acrylamide is a food process contaminant with carcinogenic and genotoxic properties. As a result of intensive research, numerous mitigation initiatives to prevent its formation were suggested and various of them were implemented in the food chain. To evaluate if the mitigation strategies applied were significant, a comparison was made between two time periods (2002–2007 versus 2008–2013) in terms of acrylamide food levels and dietary exposure in Belgian.

The most important changes observed are a significant decrease of the acrylamide content in potato crisps and gingerbread, and a significant increase in (instant) coffee. Additionally, the acrylamide content of breakfast cereals, bread and rolls, chocolate and baby biscuits showed a downward trend, whereas for coffee substitute and ready-to-eat French fries (mainly obtained from catering facilities), an upward, although not significant, trend was observed. These changes resulted in only a slight, but insignificant decline of the overall dietary exposure of adults, adolescents and children.

The mean and P95 intake estimated in the 2008–2013 period for these consumer groups corresponded to margins of exposure (MOE) ranging between 515 and 236 and between 155 and 71, respectively, when based on the endpoint for neoplastic effects ($BMDL_{10}$ = 0.17 mg/kg bw per day). Such low MOE values indicate that acrylamide remains an issue for public concern, requiring renewed attention.

1. Introduction

Acrylamide (AA, CH_2=CHCONH$_2$, CAS nr. 79-0601) is almost exclusively used for the synthesis of polyacrylamides, which have various applications, such as in waste water treatment and paper processing. Its presence was also reported in tobacco smoke (ECB, 2002). In 2002 unexpected high levels of this chemical substance were found in various foods, leading to intensive research encompassing its occurrence, analysis, chemistry and toxicology (Claeys, De Vleeschouwer, & Hendrickx, 2005; EFSA, 2008; Friedman & Levin, 2008; JECFA, 2005; Stadler & Scholz, 2004; Taeymans et al., 2004; Tardiff, Gargas, Kirman, Carson, & Sweeney, 2010).

AA is neurotoxic and probably also carcinogenic and genotoxic for humans (IARC, 1994; JECFA, 2005). It is naturally formed at temperatures above 120 °C (and low moisture), principally via the Maillard reaction and mainly in foods containing free asparagine and reducing sugars.

Since its finding in food, AA levels were monitored in various countries and the observed results indicated a public health concern. As such, several research-based initiatives were taken in order to reduce the level of this process contaminant in food, amongst which the regular organization of stakeholder meetings, workshops and forums (Busk, 2010). For instance, the European Commission (EC) and Food Drink Europe (FDE, formerly the Confederation of the European Food and Drink Industry or CIAA)

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organized a joint workshop in 2006, where government, industry and academia discussed the issue. The workshop resulted in a number of concrete actions, including a European monitoring program of the AA levels in a number of foods (Recommendation 2007/331/EC, later extended by Recommendation 2010/307/EU) and the development and dissemination of sector-specific brochures and leaflets offering small to medium sized food companies guidelines for minimizing the AA content of their products (EFSA, 2009). The FDE developed additionally the "AA Toolbox", a regularly updated guide for industry with possible intervention steps for reducing AA levels in food (FDE, 2014). Similarly the European Potato processors' association launched the multilingual GoodFries initiative (www.goodfries.eu) for food services and consumers.

From the onset of the AA issue, the Belgian Federal Agency for the Safety of the Food Chain (FASFC) monitors the AA content of different foodstuffs. Based on monitoring results obtained between 2002 and 2007, the AA intake of the Belgian population was evaluated a first time and potential mitigation strategies were evaluated (Claeys et al., 2010). Since then, a large amount of new data on AA levels in food and on AA toxicity became available (EFSA, 2015). Therefore, AA data measured in food between 2008 and 2013 are reassessed in the present paper, not only to reevaluate the risk related to the AA intake of the Belgian population, but also to discuss the potential progress at the industrial and food service level regarding the minimization of AA in food products.

2. Material & methods

2.1. Acrylamide levels

AA levels were monitored in various foodstuffs on the Belgian market within the framework of the control programme of the FASFC (Maudoux et al., 2006). The AA content of the food samples was determined by a liquid chromatography-mass spectrometry (LC-MS)-ISO 17025 accredited method in the Federal Laboratory for the Safety of the Food Chain (FLVVG, 9050 Gentbrugge, Belgium), with a limit of quantification (LOQ) of 50 μg/kg and a limit of detection (LOD) of 25 μg/kg. More details are provided in Claeys et al. (2010).

Foods were grouped in different categories according to their AA level and in line with the grouping applied in other studies (Boon et al., 2005; Claeys et al., 2010; EFSA, 2012a, 2015; Matthys et al., 2005; Mestdagh et al., 2007) and as specified in Recommendation 2013/647/EU on investigations into the levels of AA in food. AA concentrations below the LOQ were replaced by LOQ/2 (middle-bound scenario).

Most samples concerned ready-to-eat foods. Regarding French fries, most samples were taken at the level of catering (e.g. chip shops, community kitchens). Only 10 samples were taken in 2011 of pre-cooked, frozen fries, which were fried according to the instructions mentioned on the packaging prior to analysis.

The distributions of AA levels analyzed in the period 2008–2013 and in the period 2002–2007 were compared with the Kruskall-Wallis test (SPSS 21; SPSS Inc., USA).

2.2. Food consumption data

To estimate the AA intake of the Belgian population, three different food consumption databases were used.

Food consumption data of adults were obtained from the Belgian Food Consumption Survey (BFCS) performed in 2004 by the Scientific Institute of Public Health (Devriese et al., 2005). The survey involved 3214 participants of 15 years or older, which were interviewed twice about their consumption during the last 24 h (repeated non-consecutive 24 h recall) in combination with a self-administered food frequency questionnaire.

Consumption data of adolescents were received from the Department of Public Health, Ghent University. The Belgian chapter of the survey, which was performed in the framework of the European HELENA project (Moreno et al., 2008), involved 245 adolescents aged between 12.5 and 17.5 years (Ghent region) who completed a 24-h recall twice (once by self-report and once by interview) using the YANA-C tool (Young Adolescents Nutrition Assessment on Computer) (Vereecken et al., 2008).

Consumption data of children were obtained from a dietary pattern study in pre-school children conducted in Flanders (Dutch-speaking part of Belgium) between 2002 and 2003 by the Department of Public Health, Ghent University. Diets of 662 pre-school children between 2.5 and 6.5 years old were assessed with parentally reported estimated dietary records for 3 days (Huybrechts & De Henauw, 2007).

The individual intakes over the different survey days were considered without the application of a usual-intake model. As a consequence, exposure estimates are probably more conservative, particularly with regard to the upper tail of the distribution (Kettler et al., 2015; Van Klaveren, Goedhart, Wapperom, & van der Voet, 2012). However, recent findings on the impact of usual intake models on the dietary exposure estimation of AA have shown that the use of usual-intake model did not bring any changes in terms of risk assessment (Mancini, Sirot, Busani, Volatier, & Hulin, 2015).

2.3. Estimation of the acrylamide intake

The dietary exposure to AA was determined by a probabilistic approach considering all data or the full distribution of the different variables (i.e. AA content and consumption). The variability of the consumption and contamination levels was characterized by a non-parametric, discrete, uniform distribution. By means of Monte Carlo simulations, individual consumption data and AA concentration data were sampled randomly by the Latin Hypercube method from the databases and combined for the relevant food consumed. Summing over foods provided an estimate of the exposure distribution. All estimated exposures were adjusted for the individual's body weight reported in the surveys and expressed as μg/kg bw/day. The number of Monte Carlo simulations was 100,000. To quantify the uncertainties in the exposure calculations due to sampling uncertainty of consumption and concentration data, the bootstrap method was used. With this method a bootstrap database is generated of the same size as the original database for both consumption and concentration by a theoretical resampling from the original databases. These two bootstrap databases are then used for the Monte Carlo exposure calculation and derivation of the relevant percentiles. By repeating this process 500 times, 500 'bootstrap' datasets are obtained, on which the same statistical calculations (e.g. 97.5th, 99.9th percentile, etc.) can be applied as on the original dataset. As such a 'bootstrap' distribution of 500 97.5th, 99.9th percentiles, etc. is created that characterizes the uncertainty of the original dataset (Vose, 2008). Calculations were performed by the software @Risk® (Palisade Corporation, Version 6, NY, USA).

For the conversion of the AA level of roasted or grounded coffee beans (coffee surrogate) to liquid coffee (coffee surrogate) a conversion factor of 0.046 was applied (van Dooren, Boeijen, van Klaveren, & van Donkersgoed, 1995). Variability in preparation conditions (e.g. deep-frying of crisps, toasting of bread, etc.) was not taken into account.

3. Results & discussion

3.1. Acrylamide levels in food

Evaluating changes over time by means of a yearly comparison of AA levels in the different food categories was difficult since some food categories were quite heterogeneous. Moreover, not every year the same number and types of products were analyzed within a food category. For example, the category of breakfast cereals contained samples with and without chocolate pieces, with toasted or with puffed cereals. The category of biscuits included hard, dry as well as soft biscuits that are made from various kinds of cereals to which various additives could have been added. By merging the data over a larger period of years, it can be assumed that the heterogeneity in terms of sampling is more or less leveled. Furthermore, given the fact that around 2007 a lot of initiatives were taken for reducing the AA level in food (Busk, 2010; EFSA, 2009; FDE, 2014), it was chosen to compare two time periods, namely the periods 2002–2007 and 2008–2013. The AA levels monitored during these two time periods in different foodstuffs on the Belgian market are presented in Table 1. The number of samples analyzed was 771 for the period 2002–2007 and 954 for the period 2008–2013.

Most important findings were a significant decrease of the mean AA level in potato crisps and gingerbread, and a significant increase in coffee (Table 1). Additionally, a significant decrease of the mean AA level was observed in breakfast cereals, breads & rolls, chocolate and baby biscuits, but these changes appear to be less pronounced.

Parallel to the significant decrease observed for the mean AA level measured in potato crisps when comparing the periods 2002–2007 and 2008–2013 (on average − 38%), an annual comparison of AA levels showed a steady decreasing trend as well, with a decline of both the average and the variance on the values (Fig. 1a). A similar trend was observed by Powers, Mottram, Curtis, and Halford (2013) in a compilation of industrial data (40,455 samples in total), with a significant decrease of the average level from 763 μg/kg in 2002 to 358 μg/kg in 2011.

In contrast to potato crisps, the AA content of French fries did not change much. A yearly comparison even suggested an upward trend of AA levels, although it was not significant (Fig. 1b). A similar slight increase was also observed in the European monitoring data collected from 2007 to 2010 (EFSA, 2012a). Remarkably, the variance on the AA levels measured in French fries was relatively small between 2005 and 2009, but widened from 2012 onwards. Given that the fries analyzed were primarily sampled at the level of catering (e.g. chip shops, community kitchens), the question arises whether operators have adopted a lax attitude about minimizing the AA content of their fries. Regarding pre-cooked French fries for home cooking (frozen fries), the Belgian dataset contained only 10 samples (in 2011) that were fried according to the instructions mentioned on the package prior to analysis. These samples all had an AA level below the LOQ of 100 μg/kg. This illustrates the fact that the time temperature conditions applied during the final baking process have major impact on the AA level, and as such the responsibility of caterers, but also of consumers, for paying attention to the baking conditions applied in order to keep AA levels as low as possible.

For comparison, AA levels measured between 2007 and 2013 by the British Food Safety Agency (FSA) indicate overall a downward trend for the AA content of crisps, prefabricated potato snacks and pre-cooked fries/potato products for home preparation (FSA, 2014).

Whereas the AA level remained similar in toast, biscuits and sweet spiced biscuits, it dropped significantly in gingerbread (−67% of the 6-year average) with a steady decrease of both the annual averages and the variance (Fig. 1c). A similar downward trend of the AA level of gingerbread was not observed in the European AA database (EFSA, 2012a, 2015). Moreover, the AA values reported between 2010 and 2013 to the European Food Safety Authority (EFSA) were higher (average of 407 and P95 of 1600 μg/kg) compared to the levels measured in this study. This difference is presumably related to the fact that gingerbread samples in this survey mainly concerned the bigger brands in retail, whereas the European dataset contained additionally many samples of gingerbread manufactured according to traditional recipes and in smaller (local) companies where AA reduction strategies are probably less implemented than in larger food businesses.

With respect to coffee, the average AA level measured between 2008 and 2013 was almost twice as high than the average measured between 2002 and 2007, which seemed to be mainly due to a rise of the AA level in instant coffee and not so much in roasted

Table 1
Acrylamide levels (μg/kg) of food on the Belgian market between 2002 and 2013.

Food category	2002–2007					2008–2013					2002–2013					p-value *
	n° of samples (<LOQ)	Mean[a]	P50	P95	Max	n° of samples (<LOQ)	Mean[a]	P50	P95	Max	n° of samples (<LOQ)	Mean[a]	P50	P95	Max	
Breakfast cereals	71 (13)	185 ± 167	120	518	674	89 (39)	145 ± 144	63	456	670	160 (52)	163 ± 155	95	481	674	0.041
Potato crisps	97 (5)	609 ± 555	485	1500	3200	54 (1)	375 ± 229	310	725	1300	151 (6)	525 ± 477	408	1410	3200	0.016
Fries	137 (28)	236 ± 326	170	604	3300	136 (43)	268 ± 325	218	630	2500	273 (71)	252 ± 325	180	608	3300	0.231
Coffee	73 (8)	293 ± 375	200	957	2522	108 (3)	548 ± 626	330	1730	3800	181 (11)	445 ± 552	270	1326	3800	0.000
Roasted coffee	52 (5)	285 ± 59	170	1303	2522	56 (0)	269 ± 33	220	488	1800	108 (5)	277 ± 33	200	1064	2522	0.007
Instant coffee	21 (3)	313 ± 45	290	792	810	52 (3)	847 ± 106	612	3170	3800	73 (6)	694 ± 81	530	2610	3800	0.000
Coffee substitute	29 (0)	2621 ± 895	2600	3920	4700	55 (0)	2915 ± 1111	2956	4652	5400	84 (0)	2814 ± 1045	2800	4598	5400	0.255
Bread & rolls	71 (53)	38 ± 30	25	83	230	121 (97)	32 ± 44	25	66	400	192 (150)	34 ± 40	25	69	400	0.000
Toast	39 (12)	130 ± 102	120	312	430	26 (5)	129 ± 120	80	390	460	65 (17)	130 ± 109	100	326	460	0.813
Biscuits[b]	50 (13)	167 ± 244	116	316	1514	53 (18)	142 ± 190	70	524	1113	103 (31)	154 ± 217	96	510	1514	0.281
Sweet spiced biscuits	17 (0)	346 ± 187	270	694	760	10 (1)	339 ± 273	297	806	860	27 (1)	344 ± 217	284	754	860	0.900
Gingerbread	47 (1)	689 ± 568	450	1770	2100	59 (9)	225 ± 150	240	454	530	106 (10)	431 ± 455	320	1673	2100	0.000
Chocolate	21 (6)	198 ± 202	112	700	750	26 (8)	74 ± 57	57	210	249	47 (14)	129 ± 153	66	338	750	0.010
Cereal bars	20 (14)	61 ± 49	50	181	190	37 (27)	104 ± 138	50	264	820	57 (41)	88 ± 116	50	260	820	0.056
Popcorn	45 (7)	229 ± 242	150	802	1100	57 (8)	212 ± 130	180	422	470	102 (15)	220 ± 187	163	468	1100	0.363
Baby biscuits	54 (17)	240 ± 303	135	1022	1217	123 (58)	117 ± 155	50	362	1200	177 (75)	155 ± 218	63	608	1217	0.002

*Significant differences ($p < 0.05$) between pooled 2002–2007 and 2008–2013 data based on the Kruskall–Wallis test.
[a] Mean ± standard deviation.
[b] Sweet spiced biscuits are excluded.

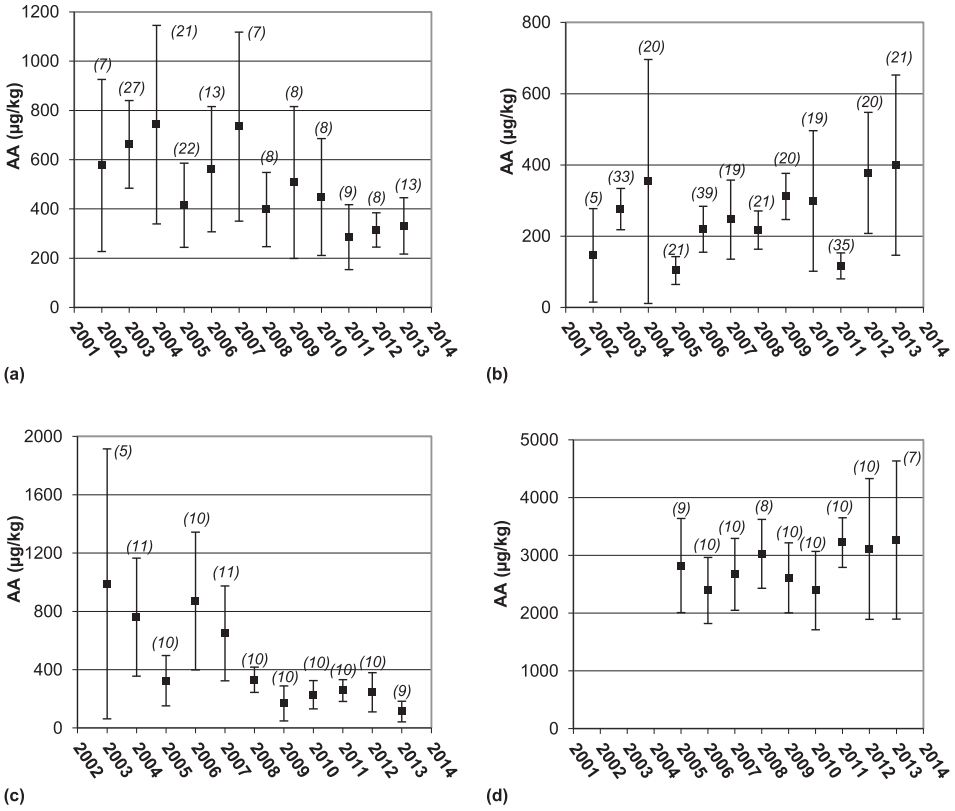

Fig. 1. Yearly comparison of AA levels (µg/kg, mean + 95% confidence interval) measured in (a) potato crisps, (b) French fries, (c) gingerbread, and (d) coffee substitute on the Belgian market between 2002 and 2013 (with between brackets the number of samples analyzed each year).

coffee. In general, higher AA levels were measured in instant coffee compared to roasted coffee (Table 1). The average AA content of coffee substitute remained essentially unchanged, although an annual comparison of the AA levels showed a gradual upward trend of both the average and the variance of results (Fig. 1d). The samples concern mainly coffee substitutes based on chicory. European monitoring results showed higher AA levels in chicory based coffee surrogates (on average 2942 µg/kg) than in cereals based coffee surrogates (on average 510 µg/kg) (EFSA, 2015).

An increase of the AA levels in coffee, including instant coffee, and in coffee substitutes was also observed in the European AA database of 2007–2010 (EFSA, 2012a). On the British market on the other hand, the AA content of roasted coffee showed a downward trend between 2007 and 2013 (FSA, 2014). Although different practical mitigation strategies are possible for most foods, a substantial reduction of the AA content of coffee and coffee substitutes is reported to be, at present, unlikely without affecting the quality or developing additional food safety issues (EFSA, 2012a; FDE, 2014; Lineback, Coughlin, & Stadler, 2012). Nevertheless, the AA levels measured in coffee and coffee substitutes on the British market, with an average level of 430 µg/kg (maximum of 1056 µg/kg; 40 samples taken in 2012–2013), were far below the levels measured in this study or reported in the compiled EFSA database, indicating that lower levels could somehow be achievable for coffee products.

Although the mean AA level of breakfast cereals for the 2008–2013 period was significantly lower than for the 2002–2007 period (about 20%), a yearly comparison of the AA levels did not show any downward trend (data not shown). As already noted, the food category of breakfast cereals is a diverse group of products rendering the interpretation of a possible trending difficult. Consequently, it is unclear whether the decline of the pooled average level is genuinely due to efforts undertaken by the industry or to the basket of samples. In the European monitoring dataset (2010–2013) for example, higher AA levels were found in breakfast cereals based on bran and whole grains (on average 211 µg/kg) than in breakfast cereals made from wheat and rye (an average of 170 µg/kg) or based on corn, oats, spelled barley and rice (average 102 µg/kg) (EFSA, 2015).

The observed decrease for the average AA level of bread & rolls after 2008 was probably largely artificial and merely the result of a modified method of analysis. Given that >99% of the AA of bread is situated in the crust (Surdyk, Rosén, Andersson, & Åman, 2004) and that for some 80% of the samples the AA level was below the LOQ, it was decided around 2009 to analyze the AA content solely in the bread crust with conversion of the result to the full bread, in order to obtain more accurate results.

The AA content of chocolate showed a significant decrease as well. However, chocolate was not sampled every year. The merged data of 2002–2003–2004 were compared with those of 2009–2011–2012, and particularly in 2004 higher AA values were measured (an average of 312 µg/kg in 2004 compared to less than 100 µg/kg in the other years) which may have introduced some bias (data not shown).

3.2. Acrylamide intake of children, adolescents and adults

The AA intake of Belgian adults, adolescents and pre-school children is given in Table 2. Intake estimates indicate that some of the initiatives taken for reducing the AA content of food, seem to have resulted in a reduction of the AA intake. Taking the uncertainty on the data into account, this reduction is however not significant. Children, followed by adolescents, have the highest AA intake. Children have a relatively higher level of food consumption per kg of body weight, but also a diet that differs significantly from

that of adults. The contribution of the most relevant food groups to the AA intake (contribution > 1%) is shown in Fig. 2. Due to lack of recent data, the AA intake of children, adolescents and adults before and after 2007–2008 were estimated using the same consumption data, whereas meanwhile consumption patterns might have changed. Possible changes related to the intake and the contribution of the different food categories to the intake therefore reflect mainly the changes observed in AA levels between the periods 2002–2007 and 2008–2013 (Fig. 2).

The AA intake of adolescents has decreased the most (around −26%). Besides French fries, potato crisps in which AA levels decreased significantly, contributed the most to the average AA intake. The smallest change is observed for adults (around −10%). Their AA intake is mainly determined by coffee and French fries, two products for which AA levels showed an increased tendency. The main sources for the AA intake of children (that decreased with 12 à 17%) were biscuits followed by French fries and breakfast cereals. Bread and rolls, having relatively low AA levels but which are largely consumed, are an important source of AA for the lower intake percentiles of all three age groups considered.

A direct comparison between the intakes estimated in this study and those published in the literature should be made with caution due to methodological differences (e.g. differences in food categories covered, between sampling methods and consumption surveys). Nevertheless, the dietary exposure estimates in the present study are in the same range as those reported for adults, adolescents and children (Hirvonen et al., 2011; Mojska, Gielecinska, Szponar, & Oltarzewski, 2010; Sirot, Hommet, Tard, & Leblanc, 2012). Based on European monitoring data collected and analyzed since 2010, the EFSA calculated for children an average intake between 0.5 and 1.9 μg/kg bw per day and a P95 intake between 1.4 and 3.4 μg/kg bw per day, depending on the survey and age group. The dietary exposure of adolescents, adults and elderly was estimated to be on average between 0.4 and 0.9 μg/kg bw per day and the P95 was between 0.6 and 2.0 μg/kg bw per day, depending on the survey and age group (EFSA, 2015).

A comparison with the study of Matthys et al. (2005) might give some indication of the effect of a modified consumption pattern on the AA intake, even though a direct extrapolation is difficult. In the study, the AA intake of adolescents was estimated based on Belgian (FASFC) monitoring data from 2003 (150 samples) and a comparable consumption survey of 314 adolescents aged 13–18 years performed in 1997 (in region of Ghent, performed by the Department of Public Health, Ghent University). The estimated median (P50) intake was 0.51 μg/kg bw per day, which is about one and a half times higher than the median intake for adolescents estimated in the present study. Bread and French fries, followed by biscuits (from P55) were the main AA sources for the lower intake percentiles, whereas in the present study French fries and biscuits only become relevant AA sources starting from the P85 and the P60 intake respectively.

3.3. Risk assessment

Given that AA is a possible genotoxic carcinogen to which the ALARA principle ('as low as reasonable achievable') applies, the margin of exposure (MOE) approach might give an idea of the risks associated with its presence in food. The MOE is the ratio between a defined reference point on the dose–response curve for the adverse effect and the human intake, and makes no implicit assumptions about a "safe" intake (EFSA, 2005). In its recent opinion, the EFSA CONTAM panel selected two reference points for characterizing the hazard that AA poses (EFSA, 2015). For non-neoplastic effects the incidence of peripheral nerve (sciatic) axonal degeneration observed in male F344 rats exposed to AA in drinking water for 2 years (NTP, 2012), was selected as the most relevant and sensitive endpoint for neurotoxicity. A benchmark lower dose level or $BMDL_{10}$ of 0.43 mg/kg bw per day was derived. A comparison of this reference point with the intake values estimated for the 2008–2013 period (Table 2) results in MOE values of 1303, 896 and 597 for the average intake and of 391, 270 and 179 for the P95 intake, and this for adults, adolescents and children respectively. It is assumed that for non-genotoxic substances a MOE above 100 gives no cause for public health concern unless there are large gaps concerning toxicity data. This margin of 100 includes the uncertainties and variability with respect to both kinetic and dynamic differences between laboratory animals and humans (a factor of $4 \times 2.5 = 10$), as well as within a population (a factor of $3.2 \times 3.2 = 10$) (EFSA, 2012b).

A value of 0.17 mg/kg bw per day was selected as a reference point for neoplastic effects (EFSA, 2015). It was derived as the lowest $BMDL_{10}$ from data on incidences of Harderian gland adenomas and adenocarcinomas in male $B6C3F_1$ mice exposed to AA for 2 years (NTP, 2012). Based on this reference point, the intake estimates for the period 2008–2013 correspond to MOE values of 515, 354 and of 236 for the average intake and of 155, 107 and 71 for the P95 intake, and this for adults, adolescents and children respectively. For substances that are both genotoxic and carcinogenic it is assumed that a MOE of 10,000 or higher, based on a $BMDL_{10}$ from an animal study and taking into account overall uncertainties, would be of low concern from a public health point of view (EFSA, 2012b, 2005). The MOE values across all age groups are however, substantially lower than this theoretical maximum value of 10,000, and indicate a concern with respect to neoplastic effects. Despite the uncertainties inherent to the assessed risk, AA remains thus a high priority contaminant for risk management actions.

Sources of uncertainty are related to input data as well as to the assessment approach applied (Kettler et al., 2015). For instance, AA levels not only differ between different food products, but might differ significantly between different brands of similar food products as well. Not only food product properties (e.g. AA precursor levels which depend on the raw materials used, the heating conditions applied) but also the analytical method used contribute to

Table 2
Acrylamide intake of the Belgian population (μg/kg bw per day).

	Mean	P50	P75	P90	P95	P97.5	P99	P99.9
2008–2013								
Children	0.72 (0.56–0.85)[a]	0.39 (0.34–0.47)	0.85 (0.69–1.02)	1.68 (1.35–2.03)	2.42 (1.97–2.99)	3.21 (2.56–4.12)	4.53 (3.35–6.81)	9.03 (5.08–18.21)
Adolescents	0.48 (0.42–0.56)	0.27 (0.23–0.32)	0.59 (0.51–0.69)	1.11 (0.94–1.33)	1.58 (1.31–1.91)	2.09 (1.72–2.69)	2.86 (2.17–4.17)	5.36 (3.16–12.22)
Adults	0.33 (0.29–0.39)	0.19 (0.17–0.21)	0.40 (0.35–0.46)	0.76 (0.65–0.90)	1.08 (0.90–1.31)	1.47 (1.19–1.86)	2.02 (1.56–3.01)	3.88 (2.25–13.55)
2002–2007								
Children	0.87 (0.74–1.03)	0.53 (0.45–0.62)	1.04 (0.89–1.25)	1.90 (1.56–2.34)	2.73 (2.14–3.51)	3.70 (2.79–4.99)	5.21 (3.58–7.56)	9.76 (5.41–23.44)
Adolescents	0.64 (0.55–0.76)	0.37 (0.31–0.44)	0.78 (0.65–0.93)	1.47 (1.24–1.77)	2.12 (1.73–2.60)	2.86 (2.22–3.71)	3.93 (2.91–5.54)	7.39 (4.41–17.87)
Adults	0.35 (0.31–0.42)	0.20 (0.17–0.22)	0.42 (0.36–0.49)	0.81 (0.67–0.97)	1.16 (0.96–1.44)	1.57 (1.26–2.01)	2.17 (1.62–3.17)	4.01 (2.49–14.88)

[a] 95 confidence intervals are given between brackets.

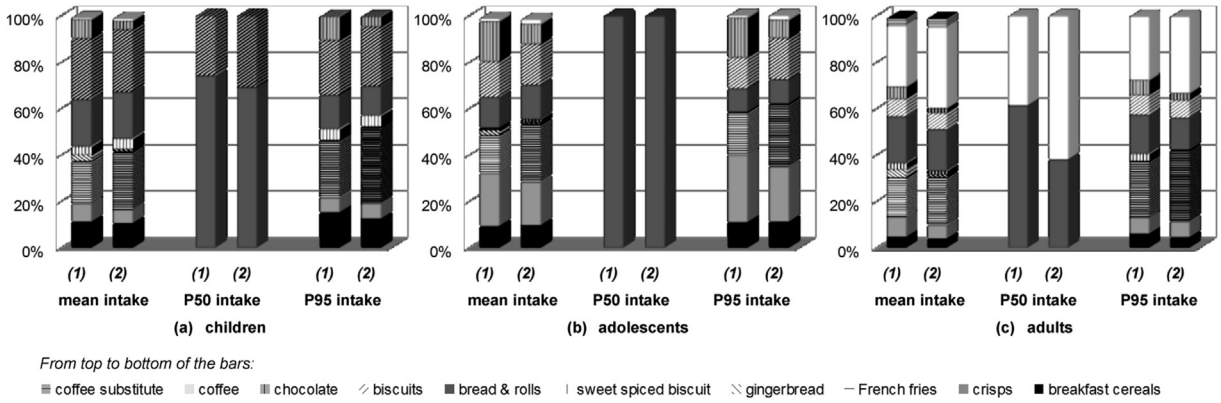

Fig. 2. Contribution (>1%) of the different food categories to the AA mean, P50 and P95 intake of (a) adults, (b) adolescents and (c) children between the period 2002–2007 *(1)* and the period 2008–2013 *(2)*.

the uncertainty. Moreover, only the products sampled within the FASFC monitoring plan were considered, whereas AA has also been detected in other foods, such as cooked seafood and dairy products. However, due to the very low AA levels measured in these foods, their contribution to the AA intake is considered negligible (Health Canada, 2012). Additional bias results from linking the consumption surveys having their own methodology, classification and detail of description of the food products, to the food categories that were selected based on the occurring AA levels. Home-cooked food, which may include potential major contributors to overall intake, were not covered in this study as this information and information about the preferences of consumers (e.g. the degree of browning of their fries or toast, the conversion of 'solid' to 'liquid' coffee) is very limited.

With respect to the toxic reference doses of AA, uncertainties include the influence of the food matrix on the AA uptake, the populations examined in epidemiological studies (e.g. regarding scale and uniformity) and the dose–response relationship (e.g. mode of action, extrapolation of the dose response relationship). According to a recent EFSA publication, one could conclude that based on a review of epidemiological data there is a lack of a consistent association between AA exposure and increased risk for the majority of the different cancer sites. However, positive associations have been found between AA intake and renal cell, endometrial and ovarion cancer. Nevertheless, according to EFSA, AA in food remains a public health concern (EFSA, 2015).

3.4. Strategies for risk policy

There are no statutory maximum AA levels of regulatory, although the latest European Commission Recommendation (2013/647/EU) on investigations into the levels of AA in food specifies 'indicative values' for AA. Depending on the food category, between 2 and 28% of the samples analyzed between 2002 and 2013 exceeded the indicative value. These indicative values were selected based on monitoring results from 2011 to 2012 in the different European member states and are not risk based. An approach for defining risk based maximum AA levels is illustrated in Table 3. These AA levels were calculated (i) assuming a maximum AA intake of 4.3 μg/kg bw/day or of 0.017 μg/kg bw/day (i.e. the ratio of the BMDL to the MOE for neurotoxicity and for neoplastic effects respectively), (ii) taking into account the procentual contribution of each food category to the average AA intake, and (iii) assuming an average consumption of each food category. It is clear that when the risk for neoplastic effects is accounted for, resulting maximum AA levels are too low to be achievable in practice. On the other hand, considering neurotoxicity as the reference point, 250 times higher limit values are

Table 3
Maximum acrylamide levels (μg/kg) at which the margin of exposure for neurotoxicity of 100 (MOE 1) and for neoplastic effects of 10,000 (MOE 2) is not exceeded (assuming an average consumption and taking the procentual contribution of each food category to the average intake into account).

		Breakfast cereals	Potato crisps	Coffee	Coffee substitute	French fries	Bread & rolls	Toast	Biscuits	Chocolate	Gingerbread	Sweet spiced biscuits	Cereal bars	Popcorn
Children														
Contribution[a]		10.6%	5.9%	1.0%	–	25.0%	20.2%	0.9%	26.9%	3.7%	1.1%	4.4%	0.2%	0.1%
AA level[b]	MOE1	848	2231	153		1595	188	770	842	436	1347	2045	644	1305
	MOE2	3	9	1		6	1	3	3	2	5	8	3	5
Adolescents														
Contribution[a]		9.7%	18.8%	1.9%	–	24.7%	14.8%	0.9%	17.6%	8.6%	0.9%	1.2%	0.3%	0.4%
AA level[b]	MOE1	1290	3333	228		2391	283	1139	1267.5	658	1993	3026	925	1891
	MOE2	5	13	1		10	1	5	5	3	8	12	4	8
Adults														
Contribution[a]		3.9%	5.8%	35.0%	3.8%	20.6%	17.9%	0.8%	7.0%	2.1%	1.3%	1.5%	0.1%	0.2%
AA level[b]	MOE1	1860	4871	330	1784	3461	413	1639	1834	963	2960	2210	1451	2847
	MOE2	7	19	1	7	14	2	7	7	4	12	9	6	11
Indicative AA values[c]		200–400	1000	450–900	2000–4000	600	80–150	450	500		1000	500		

[a] Procentual contribution of the food category to the average intake (period 2008–2013).
[b] Assuming an average consumption of each food category.
[c] Indicative AA values based on the EFSA monitoring data from 2011 to 2012; see European Commission Recommendation of 8 November 2013 on investigations into the levels of acrylamide in food (2013/647/EU) for more details.

obtained that exceed by far the indicative values set by the European Commission, except for coffee and coffee substitute. Nevertheless, the possible scenarios that can be considered and assumptions that can be made for the calculation of such risk based maximum AA levels are legion and basically depend on a risk management decision.

4. Conclusions

AA mitigation initiatives taken in 2007–2008 seem to have had only a minor impact. Although the AA content of crisps and gingerbread showed clearly a downward trend, the AA level of most other foods showed little to no change. Moreover, the AA level of coffee has increased significantly, and also for coffee substitutes and ready-to-eat French fries a gradual upward, albeit not significant, trend of the AA level was observed. An increase of the variance on AA levels, as observed for French fries from 2012 onwards, could indicate a loosened attention for AA formation and be a risk management tool for identifying where additional sensitization is required.

Accordingly, the overall effect of the observed tendencies on the AA intake appeared to be relatively small. Without accounting for possibly modified dietary patterns, the AA intake showed a slight, but not significant decrease after 2008. Based on pooled 2008–2013 data average and P95 intakes for adults, children and adolescents ranged between 0.33 and 0.72 μg/kg bw per day and between 1.10 and 2.40 μg/kg bw per day respectively. These values correspond to MOE values indicating a reason for concern regarding neoplastic effects.

Although some food sectors have undertaken efforts for reducing the AA content of their products and although in some cases there are no single solutions due to the complexity of factors to be considered, results indicate that more drastic efforts are required from both the food industry and the policy to tackle this issue.

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