ORIGINAL ARTICLE
Annals of Agricultural and Environmental Medicine 2013, Vol 20, No 2, 351–355
www.aaem.pl

Dietary acrylamide exposure in chosen population of South Poland

Joanna Zając[1], Iwona Bojar[2], Jadwiga Helbin[1], Emilia Kolarzyk[1], Artur Potocki[1], Joanna Strzemecka[3,4], Alfred Owoc[5]

[1] *Department of Hygiene and Dietetics, Jagiellonian University, Medical College, Krakow, Poland*
[2] *Department for Health Problems of Ageing, Institute of Rural Health in Lublin, Poland*
[3] *Pope John Paul II State School of Higher Education in Biała Podlaska, Institute of Public Health, Poland*
[4] *Departament of Orthopaedics and Rehabilitation, Medical University of Lublin, Poland*
[5] *Higher School of Public Health, Zielona Góra, Poland*

Zając J, Bojar I, Helbin J, Kolarzyk E, Potocki A, Strzemecka J, Owoc A. Dietary acrylamide exposure in chosen population of South Poland. Ann Agric Environ Med. 2013; 20(2): 351–355.

Abstract

Introduction: Acrylamide is used for wide range of industry purposes and it is produced in food during heating process. Foods with high acrylamide concentration include French fries, chips, bread crust, cereal, different baked goods. The electrophilic nature of acrylamide allows to interact with biological molecules. It is easily absorbed via the ingestion, inhalation or through the skin.

Objective: Evaluation of dietary exposure to acrylamide in chosen population with respect to different age groups in South Poland and assessment of health risk.

Material and Methods: Food consumption survey was conducted among 3 southern provinces in Poland. Studies involved 1470 participants. A semi-Quantitative Food Frequency Questionnaire was used. Consumption data of individuals were calculated into $\mu g/kg_{bw}$/day. Statistics was calculated for both whole group and different age groups. MOE values were calculated.

Results: Average acrylamide intake was 0.85 ± 0.82 $\mu g_{acrylamide}/kg_{bw}$ per day and calculated 95th percentile was 1.70 $\mu g_{acrylamide}/kg_{bw}$/day. In general total dietary exposure decreased with age from 1.51 $\mu g_{acrylamide}/kg_{bw}$/day for the youngest group (6–12 years old) to 0.67 $\mu g_{acrylamide}/kg_{bw}$/day for the oldest one (42–60 years old). The main contributor of acrylamide in diet in all age groups are bakery products. The MOE values calculated for average acrylamide exposure in diet was 212 and 365 for $BMDL_{10}$ 0.18 and 0.31 mg/kg_{bw}/day.

Conclusions: Young population consume the highest amount of acrylamide thus any efforts should be done to rise their nutritional knowledge and to decrease intake of high acrylamide products (crisps and French fries). The need for promotion of knowledge how to decrease acrylamide level especially in home-made food regardless of age is necessary.

Key words

semi-quantitative food frequency questionnaire, acrylamide, margin of exposure

INTRODUCTION

Acrylamide (CH_2=CH-CO-NH_2) is odourless, colourless, crystalline substance, highly water soluble. It polymerizes easily and forms another molecule–polyacrylamide with new properties. Polymers of acrylamide are used for wide range of purposes (wastewater treatment processes, pulp and paper processing, mining and mineral processing or biotechnology) [1, 2, 3, 4]. High activity of acrylamide is caused by vinyl group, which is easily prone to nucleophilic attack. The electrophilic nature of acrylamide allows to interact with biological molecules. The major targets seem to be the -SH and -NH_2 groups of proteins as well as nitrogen in nucleic acid [5]. Low molecular weight and water solubility of acrylamide enable this molecule to pass through biological membranes. *In vitro* acrylamide can cross the blood/placenta barrier and *in vivo* blood/breast milk barrier [6, 7]. Its structure and compounds created during metabolic transformation enable acrylamide to react with subcellular targets. It is quickly absorbed via the ingestion, inhalation or through the skin [8].

Address for correspondence: Joanna Zając, Kopernika 7 Street, Krakow, Poland
E-mail: joanna.jankowska@interia.eu

Received: 20 November 2012; accepted: 11 February 2013

Acrylamide is metabolised on two competing pathways: conjugation with glutathione and epoxidation to glicidiamine [7]. Acrylamide can also bind to plasma proteins, mainly hemoglobin but epoxide group of glicidamine reacts easier with haemoglobin [9]. This adduct is considered as internal dose marker of acrylamide exposure [2]. Epoxide hydrolase-1 represents the most sensitive biomarker for glicydamide exposure (induction was observed at concentration as low as 0.1 μM) and it shows dose-dependent changes in gene expression [10].

The tragedy that took place in Sweden during tunnel construction, where acrylamide was used as sealing adjuvant (1997), was the cause of many adverse health effects in exposed humans and animals. The observation and researches led to acrylamide as a cause of health problems [11]. The influence of acrylamide on peoples' health was stated first in case of studies conducted on animals and showed its carcinogenic properties [12]. Data gathered in human studies are more ambiguous and correlation between dietary acrylamide uptake and cancer were found only in some groups [13, 14]. Because of reports International Agency for Research on Cancer in 1994 considered acrylamide as "probably carcinogenic to humans" 2A group carcinogen [15, 16]. The Joint FAO/WHO Expert Committee on Food Additives mentioned 0.2 mg/kg_{bw}/day

as NOAEL (non-observed adverse effect level) of acrylamide for morphological changes in rat nerves [17]. Such high value means that typical dietary exposure is about 200–50 times less than NOAEL. Though acrylamide is described as "non-threshold carcinogen" and adverse neurological effects are improbable (in normal exposure from food), some morphological changes in nerves cannot be excluded, mainly for those who have high dietary exposure [18]. For carcinogenic effect a dose-response relationship was based on animal data and Joint FAO/WHO Expert Committee on Food Additive (JECFA) proposed two different values of $BDML_{10}$ (lower limit on the benchmark dose for a 10% response): for induction of mammary tumours in rats – 0.31 mg/kg_{bw}/day and for Hardenrian gland tumours in mice – 0.18 mg/kg_{bw}/day [17]. For health risk connected with dietary exposure to acrylamide evaluation of margin of exposure (MOE) approach is used, even though it is based on animal data it is a common in risk characterization for many food chemicals [19].

Acrylamide in food is produced during heating process (Maillard reaction) [20]. The highest acrylamide content was found in heated carbohydrate-rich foods: 150–4000 μg/kg, moderate levels of acrylamide: 5–50 μg/kg in protein-rich foods and below 5 μg/kg in unheated or boiled foods [21]. Foods with high acrylamide concentration include French fries, chips, bread crust, cereal, different baked goods. The amount of acrylamide depends on processing conditions like temperature, time, nature of food matrix, oil or usage of microwave [21, 22, 23]. Save limits of acrylamide have been already determined by The Environmental Protection Agency (EPA) and the U.S. Food and Drug Administration (FDA) in water and food. In water: less than 0.5 ppb, in food: 12 μg of acrylamide per person per day are safe in terms of the nervous system [24]. The complex response of human organism to exposure of acrylamide, including exposure from food is still unclear and correlation between estimated acrylamide intake and internal biomarkers is low [17].

The aim of this study was evaluation of dietary exposure to acrylamide in chosen population with respect to different age groups in South Poland and evaluation of health risk.

MATERIAL AND METHODS

Food consumption survey was conducted between September 2011 and June 2012 in southern region of Poland. Among 3 southern provinces: Lesser Poland, Silesian, Podkarpackie random districts were chosen. Then, random households, and random individuals within were taken under consideration in survey. Information about aims of survey and way of conducting were delivered by phone call (if possible) or sent by post. For those who agreed an appointment was made. Among 1560 individuals who were asked, 1470 agreed to participate. The age of participants extended from 6 and 60 years old. Assessment of diet for a large number of individuals requires quick and simple method like questionnaire. For people from a range of various socioeconomic backgrounds, food frequency methods seems to be the most suitable therefore we adapted and validated a semi-Quantitative Food Frequency Questionnaire, conducted in a face-to-face way by trained interviewers. Individuals were asked particularly about products that are known or potential contributors to acrylamide exposure (164 different food items for which questioned individuals were choosing proper frequencies from "never" to "n times per day"). Portion sizes were estimated using Photographic Album of Products and Dishes [25]. Consumption data of individuals were calculated into $\mu g/kg_{bw}$/day. Concentrations of acrylamide in each group of food were taken from European acrylamide monitoring database from June 2006 [26]. Average, standard deviation, median, percentiles value were calculated for different age groups (children from 6 to 12 years old – 300 individuals; adolescences between 13 and 19 – 296 individuals; adults between 20 and 30 – 296 individuals; between 31 and 41 – 278 individuals; between 42 and 60 years old – 300 individuals). Distributions of consumed amounts of each product are not gaussian, so we present also median values for them.

Risk connected with dietary exposure (MOE values) to acrylamide was calculated. MOE values were obtained by comparison of average and 95th percentile intake of acrylamide to both $BMDL_{10}$ (lower limit on the benchmark dose for a 10% response) values: 0.31 mg/kg_{bw}/day and 0.18 mg/kg_{bw}/day.

RESULTS

All individuals were exposed to acrylamide through diet. Average value for whole group was $0.85 \pm 0.82\ \mu g_{acrylamide}/kg_{bw}$ per day and calculated 95th percentile was 1.70 $\mu g_{acrylamide}/kg_{bw}$/day. In general total dietary exposure decreased with age from 1.51 $\mu g_{acrylamide}/kg_{bw}$/day for the youngest group (6–12 years old) to 0.67 $\mu g_{acrylamide}/kg_{bw}$/day for the oldest one (42–60 years old), data presented in Tables 1–5. Comparison of average values for men and women at different age was presented on Fig. 1. The highest differences were observed for two youngest age groups and it decreased with age. In addition men consumed more acrylamide than women.

The main contributor of acrylamide in diet in all age groups were bakery products. For most age groups second great contributor of acrylamide are different kinds of crisps. With

Table 1. Assessment of acrylamide intake among children (6-12 years old); in $\mu g_{acrylamide}/kg_{bw}$/day

age	from 6 to 12							
food category	cereals	baker's goods	crisps	French fries	cookies	coffee	meat	the overall intake
average	0.14	0.63	0.37	0.13	0.21	0.00	0.03	1.51
SD	0.11	0.29	0.68	0.22	0.31	0.00	0.01	0.86
median	0.13	0.62	0.05	0.08	0.10	0.00	0.03	1.36
25th	0.05	0.42	0.00	0.00	0.00	0.00	0.03	0.83
75th	0.22	0.75	0.44	0.10	0.28	0.00	0.04	1.78

Table 2. Assessment of acrylamide intake among children (13-19 years old); in $\mu g_{acrylamide}/kg_{bw}$/day

age	from 13 to 19							
food category	cereals	baker's goods	crisps	French fries	cookies	coffee	meat	the overall intake
average	0.04	0.41	0.04	0.05	0.10	0.04	0.02	0.89
SD	0.07	0.25	0.54	0.10	0.13	0.05	0.02	0.89
median	0.01	0.40	0.01	0.01	0.05	0.01	0.02	0.62
25th	0.00	0.26	0.00	0.00	0.00	0.00	0.01	0.35
75th	0.05	0.55	0.13	0.03	0.14	0.06	0.03	0.91

Table 3. Assessment of acrylamide intake among adults (20–30 years old); in $\mu g_{acrylamide}/kg_{bw}$/day

age	from 20 to 30							
food category	cereals	baker's goods	crisps	French fries	cookies	coffee	meat	the overall intake
average	0.04	0.33	0.05	0.02	0.09	0.06	0.01	0.61
SD	0.14	0.19	0.08	0.04	0.15	0.08	0.01	0.32
median	0.00	0.30	0.02	0.01	0.05	0.02	0.01	0.56
25th	0.00	0.22	0.00	0.00	0.00	0.00	0.00	0.41
75th	0.03	0.42	0.05	0.03	0.11	0.08	0.02	0.77

Table 4. Assessment of acrylamide intake among adults (31–41 years old); in $\mu g_{acrylamide}/kg_{bw}$/day

age	from 31 to 41							
food category	cereals	baker's goods	crisps	French fries	cookies	coffee	meat	the overall intake
average	0.03	0.31	0.01	0.03	0.08	0.09	0.01	0.56
SD	0.05	0.19	0.04	0.04	0.12	0.17	0.01	0.26
median	0.00	0.28	0.00	0.00	0.03	0.05	0.01	0.52
25th	0.00	0.17	0.00	0.00	0.00	0.02	0.01	0.40
75th	0.03	0.37	0.00	0.04	0.09	0.10	0.02	0.67

Table 5. Assessment of acrylamide intake among adults (42–60 years old); in $\mu g_{acrylamide}/kg_{bw}$/day

age	from 42 to 60							
food category	cereals	baker's goods	crisps	French fries	cookies	coffee	meat	the overall intake
average	0.04	0.25	0.15	0.05	0.05	0.11	0.01	0.67
SD	0.14	0.14	0.75	0.07	0.07	0.17	0.01	1.26
median	0.00	0.23	0.00	0.02	0.02	0.09	0.01	0.35
25th	0.00	0.17	0.00	0.00	0.00	0.00	0.01	0.33
75th	0.04	0.30	0.00	0.07	0.07	0.12	0.01	0.53

increasing age more intensive uptake of brewed coffee can be observed. Contribution of meat remains on the same level across all groups. More detailed percentage shares for different food categories and different age groups based on average values were presented on Fig. 2.

The MOE values, shown in Table 6, calculated for average acrylamide exposure in diet was 212 for $BMDL_{10}$=0.18 mg/kg_{bw}/day and 365 for $BMDL_{10}$=0.31 mg/kg_{bw}/day. The lowest values for MOE for both average and 95th percentile were observed among the youngest group. Up to age 31–41 age group MOE vales for average exposure as well as MOE values calculated for the 95th percentile were increasing. In the oldest group both MOE values are slightly decreasing.

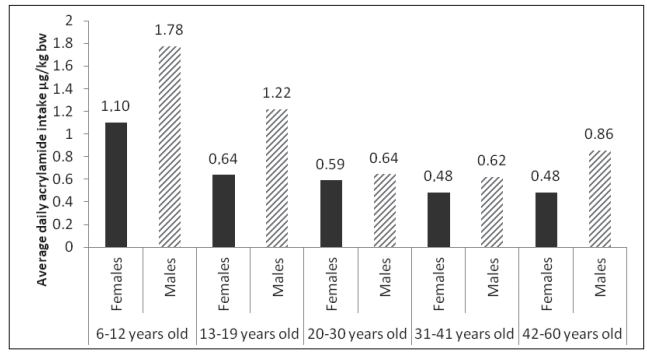

Figure 1. Average values of overall acrylamide intake in different age groups with sex classification

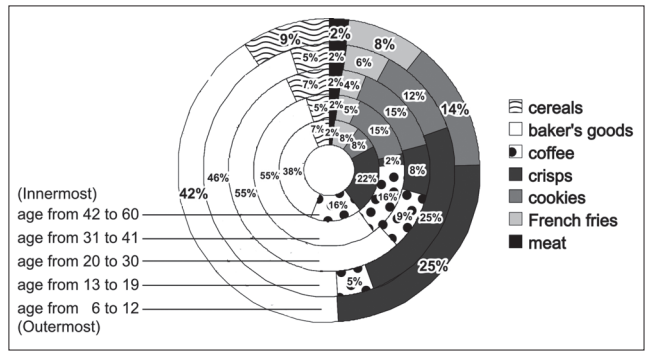

Figure 2. Percentage share of acrylamide intake in different age groups based on average values from tables 1–5. The most inner circle represents the oldest group while the other rings. successively younger categories

DISCUSSION

The latest assessment of acrylamide dietary exposure in Poland was published in 2010 [27]. Food consumption data in that study were taken from "Household Food Consumption and Anthropometric Survey in Poland" conducted in 2003 [28]. Comparing with previous studies overall intake was approximately twice bigger: in present study – 0.85±0.82 $\mu g/kg_{bw}$/day and 0.43±0.70 $\mu g/kg_{bw}$/day in previous one. Probably the main difference in exposure was the result of extrapolation to age from 1 to 99 years old done in former evaluation and the fact that previous one assessed consumption for whole Polish population. Moreover, as mentioned above, consumption data were taken from study conducted in 2003. Its unlike that the differences were due to distinctions in concentrations of acrylamide in food, cause concentrations used in present study were comparable or even lower than those assessed by Mojska et al. [27]. Present assessment of acrylamide intake was about two times higher than other national assessments conducted in such countries as: Belgium [29], France [30], China [31] or Norway [32], about 60%-70% higher than in Australian [33]

Table 6. Margins of exposure (MOE) estimated for average and 95th percentile of dietary exposure to acrylamide. in different age groups

Age (years)	6–12		13–19		20 –30		31–41		42–60		all	
	$\bar{x}$	95th	$\bar{x}$	95th	$\bar{x}$	95th	$\bar{x}$	95th	$\bar{x}$	95th	$\bar{x}$	95th
Exposure ($\mu g/kg_{bw}$/day)	1.51	2.86	0.89	3.00	0.61	1.15	0.26	1.10	0.67	1.12	0.85	1.70
MOE ($BMDL_{10}$=0.18mg/kg_{bw}/day)	119	63	203	60	295	157	702	163	269	161	212	106
MOE($BMDL_{10}$=0.31mg/kg_{bw}/day)	206	109	349	103	509	270	1209	281	464	278	365	182

$BDML_{10}$ values from: JECFA. 2011; $\bar{x}$ – average

or Danish survey [34] but comparable with Hungarian [35] and the US studies data [36]. According to data gathered by The Joint FAO/WHO Expert Committee on Food Additives (JECFA) estimates of average intake of acrylamide among 17 countries ranged from 0.3 to 2.0 μg/kg_{bw}/day and in general values calculated for children were 2–3 times higher than for adults [37]. Probably differences in assessment reflect the fact that presented studies are regarding to population at limited age (6–60 years old). Moreover, methods used in studies, time when the survey was conducted and products which were taken under consideration were different. Information about food consumption was gathered using food frequency questionnaire, 24-hour dietary recall or food record. According to Sirot et al. [30] food frequency questionnaire overestimate consumption which is directly connected with exposure, though Brantsaeter et al. [32] compared results of 4-days food diary, food frequency questionnaire and probabilistic approach concluding that the differences between the methods for estimating dietary acrylamide exposure were small and food frequency questionnaire can be used for epidemiological studies. Time when surveys were conducted was different: like the year 2003 for Australian and Polish studies, or 2005–2007 in case of French survey [30].

Both Polish studies described bakery products as the main source of acrylamide. According to data enclosed in Polish Central Statistical Office report bread in Poland is consumed in amount about 60 kg per inhabitant per year [38], even higher amount (83 kg per inhabitant per year) is consumed in Germany and significant proportion of total intake of acrylamide in Germany came from the intake of bread and other bakery products (18–46%) [39]. Other studies reflect differences in consumption habits. In Norwegian assessment crispbread and coffee are the major contributors (22% and 28%) [32], while in French studies French fries takes the first place (45% for adults and 61% for children) [30]. In the Netherlands crisps are the most important contributor in overall exposure (31%) [40], in Sweden coffee is the main source of acrylamide in food (39%) [41]. In the USA potato crisps were leading among other sources of acrylamide [37].

Level of acrylamide depends on heating process (temperature), kind of fat used for frying and products that are used. Some countries (Germany, Sweden, France) decided to undertake approaches to reduce concentration of acrylamide in foodstuff [42]. Confederation of the Food and Drink Industries of the EU presented an useful approach: The Confederation of the Food and Drink Industries of the European Union (CIAA) Acrylamide "Toolbox", aimed at finding appropriate and practical solutions to reduce the overall dietary exposure to acrylamide [43].

The importance of acrylamide monitoring is reflected in Recommendations of European Union [44]. The system for supervising food safety covers all food chain and is regulated by many legal acts, both national and those of the European Union. Also the "Rapid Alert System for Food and Feed" (RASFF) is implemented in order to enhance consumers safety [45]. Besides focusing on safety, the risk for health assessment is commonly used (MOE). The lower MOE values are the higher risk indicate. According to JECFA's report from 2011 MOE values between 45 and 310 may implicate health concern [17]. In present study, the MOE values seem to be low, the lowest for young groups: 63 for 6–12 years old and 60 for 13–19 years old. MOE data obtained in present study extended from 60 to 1209. In JECFA report for 2011, values of MOE for general population ranged from 50 for 95th percentile to 200 for average intake [17].

It should be pointed out that estimation of MOE value was assessed through experiments conducted on rodents. Different toxicokinetics of rodents may influence on the extrapolation to humans [19]. In addition standard procedure of extrapolating health risk from high-dose effects in rodents (acrylamide causes increase of cancer incidents mainly connected with: brain cancer, lung adenomas, initiated skin tumorogenesis, cancers of the thyroid and other endocrine glands and reproductive organs [7, 46, 47]) to very low-dose level of human exposure may lead to an overestimation of toxicological risk.

Even though years of studies on acrylamide have passed the problem is still raising. Slight correlation between cancer risk and acrylamide exposure cannot be interpreted as a proof of no carcinogenicity of this molecule and it is advisable to reduce dietary exposure to acrylamide [48, 49]. Moreover it is advisable to conduct studies focusing on the effects caused by acrylamide exposure.

CONCLUSIONS

1. Young population consume the highest amount of acrylamide thus any efforts should be done to rise their knowledge about importance of nutrition and problems related with non-appropriate nutrition. The need for promotion of balanced diet and education aiming at selecting proper products is still needed, regardless of age.
2. Efforts aiming at reduction of acrylamide content in food products, both commercial and home-made, must be carried on, because elimination of acrylamide from diet with proper and healthy nutrition is very unlikely.

REFERENCES

1. Moorman WJ, Reutman SS, Shaw PB, Blade LM, Marlow D, Vesper H, Clark JC, Schrader SM. Occupational Exposure to Acrylamide in Closed System Production Plants: Air Levels and Biomonitoring. J Toxicol Environ Health A. 2012; 75(2): 100–111.
2. Hogervorst JG, Baars BJ, Schouten LJ, Konings EJ, Goldbohm RA, van den Brandt PA. The carcinogenicity of dietary acrylamide intake: a comparative discussion of epidemiological and experimental animal research. Crit Rev Toxicol. 2010; 40(6): 485–512.
3. McLean D, Agarwal V, Stack K, Horne J, Richardson D. Synthesis of guar gum-graft-poly(acrylamide-co-diallyldimethylammonium chloride) and its application in the pulp and paper industry. BioResources. 2011; 6(4): 4168–4180.
4. Zhuang X, Wang Y, Li Q, Yan S, He N. The production of bioflocculants by Bacillus licheniformis using molasses and its application in the sugarcane industry. Biotech Bioproc Eng. 2012; 17(5): 1041–1047.
5. Adamsa A, Hamdania S, Van Lanckera F, Méjrib S, De Kimpe N. Stability of acrylamide in model systems and its reactivity with selected nucleophiles. Food Res Int. 2010; 43(5): 1517–1522.
6. Fuhr U, Boettcher MI, Kinzig-Schippers M, Weyer A, Jetter A, Lazar A, Taubert D, Tomalik-Scharte1 D, Pournara P, Jakob V, Harlfinger S, Klaassen T, Berkessel A, Angerer J, Sörgel F, Schömig E. Toxicokinetics of acrylamide in humans after ingestion of a defined dose in a test meal to improve risk assessment for acrylamide carcinogenicity. Cancer Epid Biom Prev. 2006; 15: 266–271.
7. Capuano E, Fogliano V. Acrylamide and 5-hydroxymethylfurfural (HMF): A review on metabolism, toxicity, occurrence in food and mitigation strategies. Food Sci Technol. 2011; 44(4): 793–810.
8. Turkington C, Mitchell D. The encyclopedia of poisons and atidots, 2009. Copyright by Estate of Carol Turkington.
9. Xie J, Terry KL, Poole EM, Wilson KM, Rosner BA, Willett WC, Vesper HW, Tworoger SS. Acrylamide hemoglobin adduct levels and ovarian

cancer risk: a nested case-control study. Cancer Epidemiol Biomarkers Prev. 2013; Feb 15 (available in the Internet).
10. Clement FC, Dip R, Naegeli H. Expression profile of human cells in culture exposed to glycidamide, a reactive metabolite of the heat-induced food carcinogen acrylamide. Toxicology 2007; 240(1–2): 111–124.
11. Busk L. Acrylamide – A case study on risk analysis. Food Control. 2010; 21(12): 1677–1682.
12. Parka HR, Kima M-S, Kima SJ, Parka M, Konga KH, Kima HS, Kwackb SJ, Kangb TS, Kimb SH, Kima HS, Leea J. Acrylamide induces cell death in neural progenitor cells and impairs hippocampal neurogenesis. Toxicol Lett. 2010; 193(1): 86–93.
13. Lin Y, Lagergren J, Lu Y. Dietary acrylamide intake and risk of oesophageal cancer in a population-based case-control study in Sweden. Int J Cancer. 2011; 128: 676–681.
14. Pelucchi C, La Vecchia C, Bosetti C, Boyle P, Boffetta P. Exposure to acrylamide and human cancer – a review and meta-analysis of epidemiologic studies. Ann Oncol. 2011; 22: 1487–1499.
15. IARC Monographs on the evaluation of carcinogenic risk to humans. Acrylamide, summary of data reported and evaluation. IARC, Lyon, France 1994, p.289.
16. Yaminia Y, Ghambariana M, Esrafilia A, Yazdanfarb N, Moradia M. Rapid determination of ultra-trace amounts of acrylamide contaminant in water samples using dispersive liquid–liquid microextraction coupled to gas chromatography-electron capture detector. Int J Environ Anal Chem. 2012; 92(13): 1493–1505.
17. JECFA Evaluation of certain food additives and contaminants. 72nd report of the joint FAO/WHO expert committee on food additive. WHO Technical Report Series 959, 2011
18. Bonneck S. Acrylamide risk governance in Germany. 2007. http://www.researchgate.net/publication/227047240_Acrylamide_Risk_Governance_in_Germany (accessed: 2012.12.07).
19. Bolger PM, Leblancb J-C, Setzerc RW. Application of the Margin of Exposure (MoE) approach to substances in food that are genotoxic and carcinogenic: EXAMPLE: Acrylamide (CAS No. 79-06-1). Food Chem Toxicol. 2010; 48: S25–S3.
20. Capuanoa E, Ferrignoa A, Acampaa I, Serpenb A, Açarb ÖÇ, Gökmenb V, Foglianoa V. Effect of flour type on Maillard reaction and acrylamide formation during toasting of bread crisp model systems and mitigation strategies. Food Res Int. 2009; 42(9): 1295–1302.
21. Lasekan O, Abbas K. Investigation of the roasting conditions with minimal acrylamide generation in tropical almond (Terminalia catappa) nuts by response surface methodology. Food Chem. 2010; 125(2): 713–718.
22. Yuan Y, Chen F, Zhao GH, Liu J, Zhang XH, Hu XSA. A comparative study of acrylamide formation induced by microwave and conventional heating methods. J Food Sci. 2007; 72(4): 212–216.
23. Sannya M, Jinapb S, Bakkerc EJ, van Boekela MA, Luninga PA. Possible causes of variation in acrylamide concentration in French fries prepared in food service establishments: An observational study. Food Chem. 2012; 132(1): 134–143.
24. Center for Science in the Public Interest. FDA Urged to Limit Acrylamide in Food June 4, 2003. http://www.cspinet.org/new/200306041.html (accessed: 2012.12.07).
25. Szponar L, Wolnicka K, Rychlik E. Album fotografii produktów I potraw. Prace IŻŻ 96. Warszawa 2000.
26. U.S. Food and Drug Administration. Survey Data on Acrylamide in Food: Individual Food Products. http://www.fda.gov/Food/FoodSafety/FoodContaminantsAdulteration/ChemicalContaminants/Acrylamide/ucm053549.htm (accessed: 2012.12.07).
27. Mojska H, Gielecińska I, Szponar L, Ołtarzewski M. Estimation of the dietary acrylamide of the Polish population. Food Chem Toxicol. 2010; 48: 2090–2096.
28. Szponar L, Sekuła W, Rychlik E, Ołtarzewski M, Figurska K. Badania indywidualnego spożycia żywności i stanu odżywienia w gospodarstwach domowych. Prace IŻŻ 101. Warszawa 2003.
29. Claeysa W, Baerta K, Mestdaghb F, Vercammenc J, Daenensd P, De Meulenaerb B, Maghuin-Rogistere G, Huyghebaertb A. Assessment of the acrylamide intake of the Belgian population and the effect of mitigation strategies. Food Add Contam. 2010; 27(9): 1199–1207.
30. Sirot V, Hommetb F, Tarda A, Leblanca JC. Dietary acrylamide exposure of the French population: Results of the second French Total Diet Study. Food Chem Toxicol. 2012; 50: 889–894.
31. Chen F, Yuan Y, Liu J, Zhao G, Hu X. Survey of acrylamide levels in Chinese food. Food Add Contam: Part B Surveillance 2008; 1, 85–92.
32. Brantsaeter AL, Haugen M, Mul A, Bjellaas T, Becher G, Klaveren JV, Alexander J, Meltzer HM. Exploration of different methods to assess dietary acrylamide exposure in pregnant women participating in the Norwegian Mother and Child Cohort Study (MoBa). Food Chem Toxicol. 2008; 46: 2808–2814.
33. Croft M, Tong P, Fuentes D, Hambridge T. A survey of acrylamide in carbohydrate-based foods. Food Add Contam. 2004; 21(8): 721–736.
34. Olesen PT, Olsen A, Frandsen H, Frederiksen K, Overvad K, Tjønneland A. Acrylamide exposure and incidence of breast cancer among postmenopausal women in the Danish Diet, Cancer and Health Study. Int J Cancer. 2008; 122(9): 2094–2100.
35. European Food Safety Authority. Results on acrylamide levels in food from monitoring years 2007–2009 and Exposure assessment. EFSA Journal 2011; 9(4):2133–2181.
36. Vesper HW, Caudill SP, Osterloh JD, Meyers T, Scott D, Myersand GL. Exposure of the U.S. Population to Acrylamide in the National Health and Nutrition Examination Survey 2003–2004. Environ Health Perspect. 2010; 118(2): 278–283.
37. Joint FAO/WHO Expert Committee on Food Additives (JECFA). Evaluation of certain food contaminants, 2006. 64th Report
38. GUS. Analiza zmian cen towarów i usług konsumpcyjnych w ujęciu terytorialnym w pierwszym półroczu 2009.
39. Hilbig A, Freidank N, Kersting M, Wilhelm M, Wittsiepe J. Estimation of the dietary intake of acrylamide by German infants, children and adolescents as calculated from dietary records and available data on acrylamide levels in food groups. Int J Hyg Environ Health. 2004; 207(5): 463–71.
40. Konings EJM, Baars AJ, van Klaveren DJ, Spanjer MC, Rensen PM, Hiemstra M, van Kooij JA, Peterset PWJ. Acrylamide exposure from foods of the Dutch population and an assessment of the consequent risks. Food Chem Toxicol. 2003; 41: 1569–1579.
41. Lin Y, Lagergren J, Lu Y. Dietary acrylamide intake and risk of esophageal cancer in a population-based case-control study in Sweden. Int J Cancer. 2011; 128(3): 676–681.
42. Segovia Bravo K, Ramírez R, Durst R, Escobedo-Avellaneda ZJ, Welti-Chanes J, Sanz PD, Torres JA. Formation Risk of Toxic and Other Unwanted Compounds in Pressure-Assisted Thermally Processed Foods. J Food Sci. 2012; 77(1): R1-10.
43. The Confederation of the Food and Drink Industries of the EU. http://www.fooddrinkeurope.eu/uploads/publications_documents/Toolboxfinal260911.pdf (accessed: 2012.12.07).
44. Recommendations Commission recommendation of 2 June 2010 on the monitoring of acrylamide levels in food. Official Journal of European Commission http://eur-lex.europa.eu/LexUriServ/LexUriServ.do?uri=OJ:L:2010:137:0004:0010:EN:PDF (accessed: 2012.12.07).
45. Wojtyła A, Biliński P, Jaworska-Łuczak B. Regulatory strategies to ensure food and feed safety in Poland—update review. Ann Agric Environ Med. 2010; 17(2): 215–220.
46. Błochowiak K, Sidorowicz K, Sokalski J, Witmanowski H. Er:YAG laser evaluation in the treatment of benign neoplasms and tumorous lesions of the oral mucosa. PostEP Derm Alergol. 2012; XXIX, 3: 143–147.
47. Witmanowski H, Lewandowicz E, Sobieszek D, Rykała J, Łuczkowska M. Facial skin cancers: general information and an overview of treatment methods. Postep Derm Alergol. 2012; XXIX, 4: 240-255.
48. EFSA/WHO international conference with support of ILSI Europe on risk assessment of compounds that are both genotoxic and carcinogenic, 2005. 16–18 November 2005, Brussels, Belgium.
49. EFSA Results on acrylamide levels in food from monitoring year 2008. EFSA Journal 2010; 8(5):1599–1630.