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ORIGINAL ARTICLES

Acrylamide content and genotoxicity of baby food

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ABSTRACT

Arylamide (AA) is a monomer that can form in heated starchy food as a result of Maillard reaction. The adverse effects of AA in humans are neurotoxicity and carcinogenicity. The present study was carried out to cover the lack information of AA content and its genotoxicity in baby food. Sixty samples of baby food were collected from local markets ,its involved 12 types with five replicates as raw potato tubers ,potato ,corn ,sweet, chocolate and wheat processed products. AA content (µg/kg) and genotoxicity of these products were determined. The obtained results showed presence high content of AA mainly in wheat products as in Rusk (800 µg/kg),potato crisp(300 µg/kg) and chocolate(250 µg/kg).Whereas, the other products were less than 100 (µg/kg). The genotoxicity measurements showed high significant values than blank in selected baby food. The linear relationship resulted between AA content and genotoxicty ,as y= 0.1011x + 9.2047, with high significant R^2 =0.9828.The exposure assessment was carried out using analytical data and recommended daily consumption of baby food. AA concentration of baby food exposure was estimated at two levels: minimum and maximum. The calculations of AA intake daily(µg/kg per weight body)according the common consumption of baby food in Arabic area, showed that four or more types are common use daily has AA content more than safety levels. This raise alarm of human health risk for presence the toxic ,carcinogenic and neurotoxic compound in baby food.

Key words: Acryl amide, baby food, genotoxicity

Introduction

Children develop in an environment of relationships, they need also to develop in an environment free of harmful chemicals .In the same time need safe food contain nutrients in a well-balanced diet for good health. Others are toxic and carcinogenic , such as AA which occurs in foods commonly consumed by children due to its attractive appearance, taste ,color and flavor (Becalski *et al*,2003). The reducing sugars (glucose, fructose) and asparagines are natural components of plants and plant-derived ingredients used in preparation of the foods (Lingnert & Wailer 1983)(Fig.1).It has been shown that the reducing sugars are the limiting factors of AA formation in potatoes, while asparagine appears to be the limiting factor in cereal products (Stadler&Scholz, 2004).AA is a suspected carcinogenic whose presence in foods was first detected in 2002 (Lineback *et al*,2012) . A wide range of cooked foods contain AA at levels ranging from a few ppb to the excess of 1000 ppb (Friedman& Levin ,2008;Claeys *et al*, 2005). This includes potato chips, French fries, roasted coffee, and bakery products, such as bread, crisp bread, biscuits, crackers, and breakfast cereals. Maillard-type reactions, in the presence of asparagine, have proven to be a major reaction pathway of AA formation (Stadler &Schalz,2004, Biedermann *et al*,2002, Zyzak *etal*,2003, Yaylayan & Stadler (2005).(Table,1).

The Joint FAO/WHO Expert Committee on Food Additives(JECFA)estimated the mean dietary AA intake for general population including children, between 1 and 4 lg/kg bw/day. It was noted that children have dietary AA exposures at least twice as high as for adult consumers when expressed on a body weight basis (FAO/WHO, 2011). EFSA also recently performed an exposure assessment based on AA monitoring results from 2007 to 2008. The 95th percentile of AA intake for adults (>18 years) and for children (3–10 years) were estimated to range between 0.6–2.3 lg/kg bw/day and 1.5–4.2 lg/kg bw/day, for adults and children, respectively (EFSA, 2011).But,in developed countries, all citizens seem to be exposed to AA, as it is present in a broad range of staple foods (Dybing *et al*,2005). There are no regulations by authorities in Sweden or in other countries concerning allowed levels of AA in commercial foodstuffs. An exception is the maximum level in drinking water which in the European Union (EU) is 0.1 µg/L . For instance the U. S. Environmental Protection Agency (EPA)arrived at an estimate for life time risk at 4.5 cancer cases per 1000individuals at an exposure level of 1 µg AA /kg body weight (bw) and day (Duarte-Salles *et al*,2012).Average daily exposures to AA have been estimated to approximately 0.5µg/kg bw in Western countries (Dybing e al,2005).

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No data are available for AA content in baby food especially due to increasing rate for ready mail of school children. The present work concerning the determination of AA on famous baby food in local markets and its genotoxicity on human blood.

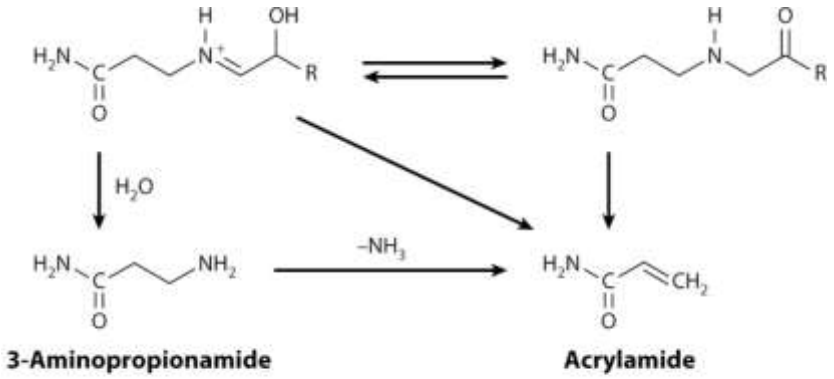

Fig. 1: Formation of acrylamide.

Table 1: Acrylamide content of different food products*.

Product/product group	Acrylamide range (μg kg^{-1})
Potatoes (raw)	<10–<50
Potato chips/crisps	117–4,215
French fries/chips	59–5,200
Bakery products and biscuits	18–3,324
Breads	<10–3,200
Bread (toast)	25–1,430
Breakfast cereals	<10–1,649
Other fruit and vegetable products	<10–70
Chocolate products	<2–826
Roasted coffee	45–935
Coffee substitute	80–5,399
Coffee extract/powder	87–1,188
Meats	<10–116
Dairy products	<10–100
Baby food and infant formula	<10–130

*Lingnert *et al* ,2002

Material and Methods

1. Sampling and treatment:

Sixty samples of baby food were collected from local markets ,its involved 12 types with five replicates as raw potato tubers ,chips potato free additives, potato crispy and potato with cheese taste. While, the others were four corn products ,as corn-rice crisp product(Sandose),popos,corn flakes and roasted corn. Also, Sweat candy(caramel candy) and chocolate .Besides, three samples of wheat products of baby food . Most all common use for baby food daily by children..

2. Acrylamide analysis:

Samples were allowed to swell adding water in an amount normally corresponding to3 times the weight of the sample (more for exceptionally dry samples). Preparation, 10 g of the homogenate was weighed into a 100 ml centrifuge glass with a screw cap and thoroughly mixed with 40 ml of 1-propanol.all analysis steps were done according to Biedermann *et al* (2002)using GC-MS according..

3. Genotoxicity test:

This technique has made to the development of the cytokinesis-block (CB) technique of human lymphocyte micronucleus assay (MN) a reliable and precise method for assessing chromosome damage. Consequently, can

now be used not only to measure whole chromosome loss or chromosome breaks but also excision repair events after using toxic human substances.

3.1. Preparation of blood samples:

Heparinized blood was obtained from six healthy, non-smoker volunteers who had no recent diagnostic or occupational exposure to ionizing radiation, laser, or chemicals and had not any experience to recentallergic responses or drug administration .Samples were prepared according to fenech (1993).

3.2. Cell Culture:

Immediately, cells were transferred into 15-ml sterile plastic round bottom tubes containing only media 199 (Sigma, Saint Loius MO, USA). Cells were incubated for 72 hours, adding cytochalasin B 48 hours before harvesting (Fenech 1993).

3.4. Harvesting of Cells:

Forty four hours after the addition of cytochalasin B, cells were collected and treated with 0.8 % sodium citrate for 3-5 minutes and then fixed in 5:1 methanol: acetic acid. Fixed cells were dropped gently onto clean microscope slides, air-dried and stained with 4% Giemsa (Sigma, Sigma, Saint Loius MO, USA) using standard procedures. (Fenech 1993).

3.4. Scoring Under the Microscope:

Slides were scored at 1000X magnification using a Leica Biomed microscope (Leica Lasertechnik GmbH, Heidelberg, Germany). Identification of cytokinesis blocked binucleatid cells and the frequencies of micronuclei in such cells were estimated according to the criteria stated by (Murtaugh *et al*,. 2004). Binucleatid cells were selected on the basis of having a well-preserved cytoplasm with two distinct nuclei of approximately equal size,which may be attached by a fine nucleoplasmic bridge or alternatively be overlapped. The micronuclei scored were therefore located within the cytoplasm and

4.0 Statstical analysis:

A Linear regression analysis was applied using Excel program; a Microsoft computer software to get value of R^2. All values were average of three replicates with standard deviation.

5.0 AA intake daily:

According the rate of baby food consumption and type of baby food as showed by workers (Erkekoğlu & Baydar, 2010, Hilbig *et al*, 2004). Besides, the values of AA as determined in selected food, the calculated range of AA as minimum /maximum rate values.

Results:

Mean daily intake of AA for adults is estimated as 1 µg/kg body weight/day. The values for infants and children are estimated to be 2- to 3-fold higher than for adults when expressed on a body weight basis (WHO/IPCS 2006).However, because toxicity studies of AA have been performed using adult animals, information on effects during fetal, infantile and pubertal periods and the relationship between the age and susceptibility to ACR-induced toxicity is insufficient.

1- Acrylamide content of Baby food

The results of AA separation using GC-MS analysis are shown in Table (2). The processed food products contained AA can classify according its concentration in two groups:

- Less than than 100 (µg kg−1), as most corn , some potatoes and wheat products. As in Table(1).
- More than 100 (µg kg−1),as in potatoes crisp(imported) ,sweets and rusk (Biksomat-in Arabic).

Most of the second group products resulted due to using high temperature for long time during food processing .Its clear ,that potatoes products from local markets contained less than 100 µg kg−1,whereas, the only one from abroad contained high content of AA as potato crisp. The controlling temperature of food processing may be can reduced AA concentration as shown between the local samples as potatoes products and

imported sample one. Unfortunately, A A can produce at low temperature (65-130 ^{0}C) as showed early detected by sweden researchers during 2005 (Eriksson, 2005).These results as obtained by Cengiz and Gündüz (2013)who showed that mean AA levels of baby biscuits, breads, baby bread-rusks, crackers, biscuits, breakfast cereals and powdered cereal-based baby foods were 153, 225, 121, 604, 495, 290 and 36µg/kg, respectively. The minimum, mean and maximum AA exposures were estimated to be 0.06, 1.43 and 6.41µg/kg BW per day, respectively. The foods that contributed to AA exposure were aligned from high to low as bread, crackers, biscuits, baby biscuits, powdered cereal-based baby foods, baby bread-rusks and breakfast cereals.

Same results were obtained by German workers (BFR, 2003) who found that children's wholemeal biscuit products were contain just under 400 microgram AA per kilogram (µg/kg) biscuit., whereas preparation of broth was up to 160 µg/kg.

Also, they showed that a toddler would take in as much AA per kilogram bodyweight as a highly exposed adult. (Hilbig *et al*, 2004). The Federal Institute for Risk Assessment (BfR) calls for the levels of AA to be reduced as far and as quickly as possible by means of good manufacturing practice. If information is available about products and their AA contents, the Federal Institute recommends that parents opt for products with low levels. (BFR, 2003, Murtaugh, *et al*, 2004). The large number of products shows that it is possible for baby and infant formula to be produced with low levels of AA.

Table 2: AA content(µg kg) and mutagenicity of selected baby food.

Type of baby diet	Acrylamide(µg kg−1)	Genotoxicity value*
Blank samples	----------	2.0 ±0.71
Corn processed products		
Corn-rice crisps (Sandose*)*	10±0.2	9.0±0.9
Corn flaks	16±1.3	6.0 ±0.5
Popcorn	18±2.0	10.0±0.7
Sweat corn-roast	20±1.4	12±0.8
Processed potato products		
Potato(raw samples)	6.0±1.0	9.0±3.0
Potato chips -additives free)	40.0±3.0	15±0.2
Potato chips -cheese tasty	14.0±6.0	13±2.0
Potato crisp(Importede)	300±8.0	40±3.0
Sweets		
Sweet candy –caramel(candy)	100±12.0	13±0.2
Chocolate	250±10.0	36±0.2
Wheat processed products		
Rusk(biksomat-in arabic)	800±0.20	90.0±1.2
Loaf	21±1.7	14.0+
Roasted bread	30.0±4.0	17.0±1.2
Linear equation : y = 0.1011x + 9.2047, R^2 = 0.9828		

*Ratio of binucleotide (BN) to mononucleotide (MN) cells as influenced by treatment methods.

- *Genotoxicity of baby food:*

The obtained data are illustrated in Table (2).Its markedly clear that high AA content induced high genotoxicity values. A linear relationship, resulted between AA content and genotoxicity values with high significant R^2 = 0.9828,the linear equation as, y=0.101 x+9.204, (Fig. 2).

Same results were obtained of the genetic toxicity of AA and glycidamide has been thoroughly reviewed in recent publications, demonstrating positive effects in bacterial cells and clastogenic and mutagenic effects in mammalian cells. AA is supposed to have genotoxic and carcinogenic effects on humans from animal tests. It forms haemoglo-bin adducts (Paulsson *et al*, 2001, CIAA, 2006, HEATOX, 2007) and the formed compounds are metabolized via a direct conjugation with glutathione or via oxidation into glycidamide, that is metabolised and eliminated in urine. Glyci-damide is more effective carcinogen than AA itself. In comparison with AA it has a higher reactivity with nu-cleophilic compounds. It forms adducts with DNA that have been detected in biological fluids and placenta (EFSA, 2013).

-AA intake daily:

According the calculations of food consumptions daily intake in some countries as showed by some workers(Duarte-Salles *et al*,2013,Hirvonen *et al*,2011 and NFCA,2002).The tabulated data are illustrated in Table (3) for some Arabic countries. Most of the children must eat at least four or five samples daily of the selected children food ,which contain high levels of AA. The famous consumed food as proved by the rate of AA intake. daily (µg/kg) minimum/maximum.Usually, as in Arabic area the selected baby food as consume daily must use one or two types from corn ,potatoes, sweet and wheat processed products. AA content in these baby food more than the safety levels .Most of these products contain high levels of AA in heated, starch-containing foods like chips, crisp bread, breakfast cereals and chips. Besides negligible amounts from other

sources. In fact ,the daily intake became practically more than the recommended dose., 0.21 to 0.43 µg/kg body weight daily (Hilbig *et al*, 2004).

Since children and adolescents eat more food in relationship to their bodyweight, relative consideration of AA intake referred to bodyweight reveals a different picture. Here, the relative intake level decreases with age and increasing body mass. Children aged between four and six belong to the group with the highest AA level. On average, they take in more than 1.2 µg/kg bodyweight daily from the foods with high acrylamide content considered in the study. In the case of adolescents it is on average 0.9 µg/kg bodyweight daily. By contrast, the daily intake of young adults aged between 19 and 24 amounts to 0.7 µg/kg bodyweight and the daily intake of adults is only 0.6 µg/kg body weight. (BFR,2002).Also, due to the concerns about the possible public health risks from dietary exposure to AA, a consultation was held by the FAO/WHO in June 2002(Health Implications of AA in Food, Report of the FAO/WHO Consultation, 2002). In rats exposed to AA in drinking water for 90 d the NOEL for morphological changes in nerves detected using electron microscopy was 0.2 mg/kg bw/d (FAO/ WHO ,2005)and no exposure related non-neoplastic lesions were found at other tissues at dose levels below 5 mg/kg bw/d. In reproduction studies, male rodents showed reduced fertility, dominant lethal effects, and adverse effects on sperm count and morphology at oral doses of AA > 7mg/kg bw/d. In female rodents, no adverse effects on fertility or reproduction have been observed, apart from slight reductions in rat offspring body weight at oral doses of 2.5 mg/kg bw/d and above.(FAO/ WHO, 2005).

Our results proved that tested food children may be contain high AA content are particularly popular with young people in Arabic area which consider alarm from healthy side.

Table 3: AA daily intake (µg/kg)minimum/maximum(Min./Max.)of tested food children.

Type of baby diet	AA (µg kg−1)	Rate of consumption(gm)		AA content (µg kg−1)	
		Min.	Max.	Min.	Max.
Corn processed products					
Corn flaks	16±1.3	50	100	800	1500
Popcorn	18±2.0	50	200	900	3600
Sweat corn-roast	20±1.4	50	300	1000	6000
Processed potato products					
Potato chips -additives free)	40.0±3.0	50	200	2000	8000
Potato crisp(Imported)	300±8.0	50	100	15,000	30,000
Sweets					
Sweet candy –caramel(candy)	100±12.0	25	50	2500	5000
Chocolate	250±10.0	50	100	12,500	25,000
Wheat processed products					
Rusk(biksomat-in arabic	800±0.20	100	400	80,000	320,000
Loaf	21±1.7	200	400	4200	8400
Roasted bread	30.0±4.0	100	200	3000	6000
Total				121,900	413,50

Discussion:

The present obtained results showed that selected baby food contain high levels of AA. The famous consumed bay food as proved by the rate of AA intake daily (µg/kg) minimum/maxiumum. Same results were obtained in Finnish children diets especially rich starch exposure among the 3-year-old children. (Hirvonen *et al*, 2011). AA, and α,β-unsaturated (conjugated) reactive molecule, can be detected as a contaminant in several foodstuffs including baby foods and infant formulas. It is anticipated that children will generally have intakes that are two to three times those of adults when expressed on a body weight basis.Though exposure to AA is inevitable, it is necessary to protect infant and children from high exposure. Besides its adverse health effects as mutagenicity, genotoxicity, carcinogenicity, neurotoxicity and reproductive toxicity, and the possible outcomes of childhood exposure from baby foods and infant formulas (Erkekoğlu and Baydar, 2010).

Usually, as in Arabic area the selected baby food as consume daily must use one or two types from corn, potaoe, sweet and wheat processed products. AA content in these baby food more than the safety levels. To avoid the toxicity of AA must use food high content antioxidants besides decrease food processing treatment as temperature or sugars in food content to prevent AA production. Practical suggestions to lower the risk of AA exposure by food without decreasing the quality of the nutrition in the diet are given (Hilbig *et al*, 2007). The international public concern followed since AA is known to cause cancer by damaging DNA. The determination of AA intake for German infant showed that children and adolescents will have higher exposures to AA than adults (Hilbig *et al*, 2007).

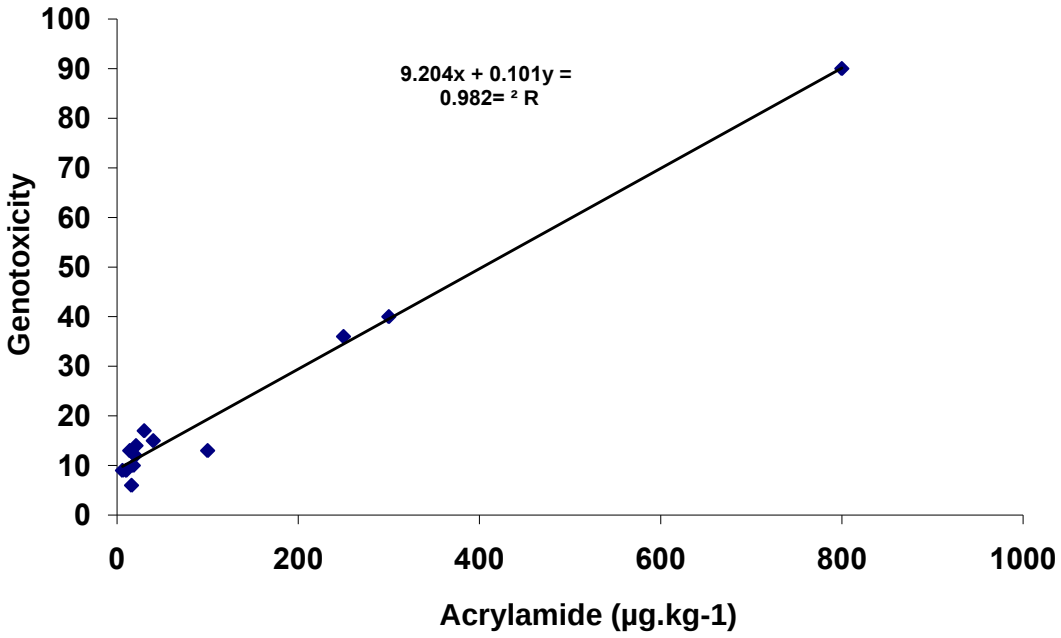

Fig. 2: The linear relationship between acylamide content and the genotoxicity of baby food.

Finally, the neurotoxicity of AA in humans is well-known from occupational and accidental exposures. In addition, experimental studies with AA in animals have shown reproductive, genotoxic and carcinogenic properties. Studies conducted in Sweden in 2002 showed that high levels of AA were formed during the frying or baking of a variety of foods (Reynolds, 2002). Most of the major countries have advised consumers to follow the dietary recommendations for a balance diet issued by their food regulatory agency. But all the available data still need more research to reduce that level to minimum values for more safe food .Cancer risk assessment of AA via food is challenging, as the whole population is exposed by low daily intakes.

Our results proved that present tested food children may be contain high AA content are particularly popular with young people in Arabic area raise alarm, due to high levels of AA in tested baby food. The famous consumed bay food as proved by the rate of AA daily intake.(µg/kg) minimum/maxiumum. Same results were obtained before by many workers (Hirvonen *et al*, 2011). It is anticipated that children will generally have intakes that are two to three times those of adults when expressed on a body-weight basis. Though exposure to AA is inevitable, it is necessary to protect infant and children from high exposure.

Mitigating the content of AA in foods, which is formed during heating of foods is recommended (Shipp *et al*, 2006, and Tareke *et al*, 2002). Although there is a 500-fold margin between the estimated average intake of dietary AA and the noael-level (no observable adverse effect level) for AA of 0.5mg/kg bodyweight per day, the health effects associated with especially the long-term intake of dietary AA are still unclear (Joint FAO/WHO Expert, 2005). Epidemiological studies about the health risks associated with dietary AA have shown contradictory outcomes, although recent studies by Hogervorst *et al* (2007) have given more indications about a positive association between high AA intake and various types of cancer (Hogervorst *et al* (2007,2008).

Practical suggestions to lower the risk of AA exposure by food without decreasing the quality of the nutrition in the diet are given. By Hilbig *et al* (2004). To face the AA content practically by minimizing the formation of AA or mitigation its content as reducing cooking temperature or using chemical methods. Also, increasing consuming of natural antioxidant sources from fruits ,spices or fruits directly in diets of children or as additives is recommended to avoid risk AA risk of toxicity.

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