Using probiotics for mitigation of acrylamide in food products: A mini review

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Using probiotics for mitigation of acrylamide in food products: A mini review

Running title: Reduction of acrylamide by probiotics

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Abstract

Acrylamide is a process-induced food toxicant which is formed during heating of food products via Strecker or acrolein pathway. Studies have revealed that acrylamide causes DNA damage, neurotoxicity, genetic toxicity, reproductive toxicity and carcinogenicity. Therefore, efficient approaches should be applied to reduce acrylamide level in foods. However, most of these approaches are not practical, requires costly equipment and cause nutrition loss and decline of sensory properties. In this regard, using specific strains of probiotics especially Lactobacillus has been newly hypothesized and scarcely explored. The mechanism responsible for this activity is
associated with the presence of peptidoglycan components particularly carbohydrates and alanine which binds to acrylamide. Another possible mechanism is production of the enzyme asparaginase which converts L-asparagine to L-aspartic acid and ammonia and prevents acrylamide formation. It has been highlighted that some *Lactobacillus* species (*L. casei* and *L. reuteri*) possess asparaginase genes. Therefore, adding probiotics could provide a good promising approach in reducing the acrylamide in food products. This article reviews the efficacy of probiotics in mitigation of acrylamide in food products.

**Keywords:** Asparaginase, Binding, Health effects, Mitigation, Probiotic

**Introduction**

Production of safe food have become a global major concern because of the great effect on consumers’ health. Conventional thermal treatments such as frying, baking, roasting are used for production of foods with prolonged shelf-life and acceptable sensory characteristics [1•, 2]. However, these thermal processes may induce detrimental changes in the food like lipid oxidation, vitamin degradation, protein denaturation and formation of harmful compounds [3, 4]. Acrylamide is one of these process-induced food toxicants which is formed in foods such as potatoes, cereals, cookies, coffee and meat products which have been subjected to high temperature [5•, 6]. It is an unsaturated amide that is easily absorbed by animals and humans after ingestion and distributed in different organs such as thymus, heart, brain, liver and kidney [7]. Acrylamide has received wide attention in recent years due to its neurotoxic, genotoxic and reproductive-toxic effects according to various studies [8]. Moreover, International Agency for Research on Cancer (IARC) has classified acrylamide as a probable human carcinogen following
detection of this compound in different heat-treated carbohydrate-rich foods (breads, biscuits, cookies, breakfast cereals, potato chips and crisps products) by Swedish Food Administration [9].

Since dietary exposure to acrylamide has been recognized as a major health concern, different approaches have been investigated to minimize acrylamide level in food products. These strategies include utilization of raw materials with low level of precursors, controlling the process conditions (pH, temperature, time) or post processing approaches like evaporation and polymerization [10, 11•]. Regarding the last respect, it has been reported that some bacteria are capable of using acrylamide in vitro as a source of carbon and nitrogen [2]. Additionally, some probiotics can alleviate acrylamide level through binding to cell wall as well as producing the enzyme L-asparaginase. Therefore, the aim of this review is to describe what are the main pathways involved in acrylamide formation in food products? What are the factors affecting its presence in foods and what is the range of occurrence in different food products? How acrylamide affect health status and what are its toxicological consequences? Finally, considering the existing researches and findings in these contexts, what are the possible strategies to decrease acrylamide content in food products and are probiotics an efficient way for acrylamide mitigation or not?

**Acrylamide formation in food products**

Two main pathways for acrylamide formation in foods are 1) Strecker pathway or N-glycoside pathway and 2) acrolein pathway. In Strecker pathway, heat treatment of foods with high level of carbohydrates and proteins containing asparagine at temperature higher than 120°C results in formation of Schiff base. The Schiff base might be transformed into Amadori compounds or decarboxylated to azomethine ylide. Acrylamide can be formed directly from azomethine ylide
or through β-elimination of decarboxylated Amadori compound or through the hydrolysis of ylide followed by the deamination of 3-aminopropionamide (3-APA) (Figure 1). It has been emphasized in different model systems that α-hydroxy carbonyl is more effective than dicarbonyls in converting asparagine to acrylamide, and fructose, which contains two α-hydroxy carbonyl groups, increases acrylamide formation by about 2-fold in comparison with other reducing sugars such as glucose [12].

In acrolein pathway, acrolein and acrylic acids are formed through the degradation of fats and dehydration of glycerol followed by reaction with the ammonia derived from the degradation of asparagine and other amino acids and acrylamide is generated. Acrylamide formation depends on some substantial factors including primary concentration of precursors, time duration and temperature intensity of heat treatment, water activity and pH [13]. The effect of various factors on formation of acrylamide in the abovementioned food products have been reviewed in details [3, 14]. Generally, it has been recognized that increasing temperature, heating time, pH and water content increase acrylamide formation in food products. Besides these exogenous factors, endogenous factors including level of precursors (free asparagine and reducing sugars) in raw materials which are associated with on genetic and environmental factors [15••].

**Acrylamide occurrence in food products**

It has been highlighted that acrylamide is extensively found in certain foodstuffs such as potatoes, bread and cake, almonds, coffee, tea and meat products (such as breaded fried meat) [16] (Table 1).

Several analytical methods have been developed for determination the acrylamide and the most frequently used techniques in food products include high performance liquid chromatography (HPLC), gas chromatography (GC) and capillary electrophoresis (CE) [16]. Most organizations
and governments have admitted LC-MS/MS with isotope dilution, GC-MS or GC-MS/MS after derivatization as standard methods for quantification of acrylamide in thermally processed foods [17].

Acrylamide is generally detected in potato products such as French fries and potato crisps. In these food commodities, presence of acrylamide precursors (asparagine and reducing sugars) besides cooking conditions at temperature above 120°C favor Maillard reaction and acrylamide formation. The average exposure of acrylamide in potato crisps was found to be 0.052 and 0.064 μg/kg bw/day for males and females, respectively [29]. In order to reduce acrylamide formation in potato products, factors such as potato cultivars, fertilization regime, climate condition and storage condition should be considered [30] and several approaches including the use of additives (asparaginase, calcium salts, citric acid, acetic acid and antioxidants) and changes in processing conditions have been applied [3].

Breakfast cereals are the significant contributor of acrylamide intake in western countries. The level of acrylamide in cereals is in the range of 62-803 mg/kg [31•]. The main determinant factor of acrylamide formation in cereal-based products is the amount of free asparagine in cereal grains. Since agronomic and genetic tools are limited and not practical to decrease asparagine level, most suggested methods are focused on processing parameters [30].

The extent of acrylamide formation in coffee is dependent to the level of precursors in green bean as well as roasting and storage conditions. During roasting, acrylamide increases at first and then would decrease as a result of precursors’ consumption [32]. Basaran et al. (2019) [33] reported acrylamide levels of 16.5 to 79.5 ng/mL in instant coffees, from 5.9 to 38.8 ng/mL in ready-to-drink (brewed) coffees and from 5.3–54.8 ng/mL in Turkish coffee and other
traditional coffees. Acrylamide was reported to be in the range of 7.7-40 μg/L in coffee brews [32]. According to Trevisan et al. [34], the level of acrylamide in grilled, fried or roasted meats have been found to be low. Soncu et al. [35] reported the average acrylamide amount in fried chicken drumsticks, chicken wings, chicken burgers and chicken nuggets to be 174.30, 20.75, 58.60 and 71.42 μg/kg.

It has been stated that acrylamide formation in various bread types is influenced by the presence of free asparagine and reducing sugars that are associated with crop’s type and cultivar, harvest season and storage condition. In addition, high temperature during baking process leads to higher content of acrylamide [36].

**Toxicological effects of acrylamide**

After acrylamide ingestion, it is rapidly absorbed and distributed in the whole body and be found in several organs such as thymus, liver, heart, brain, kidneys as well as in human placenta [1]. It can be oxidized by the enzyme cytochrome cytochrome P450 2E1 to a highly mutagenic metabolite named as glycidamide or conjugated with glutathione. Glycidamide can form adducts with -SH, -OH and -NH₂ groups in DNA, which leads to DNA damage [37]. Acrylamide causes acute toxic effects when the oral doses are greater than 100 mg/kg of BW, and lethal doses are usually higher than 150 mg/kg of BW [38].

Numerous health risks have been documented for acrylamide in the literature. It induces neurotoxicity possibly through inhibition of kinesin-based fast axonal transport and direct inhibition of neurotransmission [39]. Acrylamide inhibits the action of brain glutathione S-transferase and reduces the levels of brain glutathione. It oxidize proteins leading to change in their structure and function and nerve cell damage [8]. Acrylamide can cause tumors in different
organs such as lung, uterus, skin, mammalian gland and brain [40]. Carcinogenicity of acrylamide is derived from acrylamide-DNA adducts and consequent mutagenesis, reduction of glutathione store, inhibition of a mitotic/meiotic motor protein [1]. The tolerable daily intake (TDI) of acrylamide is 40 μg/kg per day for neurotoxicity and 2.6 μg/kg per day for cancer [16]. Acrylamide enhances reactive oxygen species (ROS) and early apoptosis and decrease DNA and histone methylation levels which result in reduced oocyte quality and fertility [41]. It has been reported that [42], the molecular basis of reproductive toxicity is the alkylation of -SH groups in the sperm nucleus and tail, which damages the testis DNA and reduction in glutathione.

Considering experimental data on detrimental effects of acrylamide on health, it is necessary to conduct risk assessment program for acrylamide in various populations within different food products. Furthermore, more prolonged epidemiological studies are required to improve the existing information related to the effects of acrylamide on humans.

**Probiotics as a functional ingredients in functional food products**

Probiotics are defined as “live microorganisms which when administered in adequate amounts confer a health benefit on the host” [43]. Several species of the genera *Lactobacillus*, *Bifidobacterium*, *Streptococcus*, *Lactococcus*, *Bacillus*, *Streptococcus*, *Pediococcus*, *Enterococcus*, *Bacteroides*, *Akermansia*, *Propionibacterium* and *Saccharomyces* are recognized as probiotics [43]. Probiotics regulate gastrointestinal functions and exert several health benefits such as reduction of lactose intolerance, cholesterol and atopic diseases, modulation immune system, alleviation of food allergy and prevention of cancers [44]. The basic mechanisms by which probiotics exert their health impacts include modulation of the mucosal barrier function, production of antimicrobial peptides and bacteriocins, adherence to epithelial cells, block of pro-inflammatory molecules and interfering with the quorum sensing signaling [45].
Although there is no consensus on the minimum of viable probiotic cells per gram or milliliter of probiotic product, generally, the concentrations of $10^6$ and $10^7$–$10^8$ CFU/mL or CFU/g have been accepted as the minimum and satisfactory levels, respectively. Furthermore, it has been pointed out that probiotic products should be consumed regularly with an approximate amount of 100 g/day in order to deliver about $10^9$ viable cells into the intestine [46].

Considering the growing awareness of consumers regarding the relation between diet and health status, development of functional foods containing healthful bioactive compounds such as probiotics has increased in recent years [43]. Probiotics can be incorporated into various food matrices such as dairy and non-dairy foods and create pleasant flavor, extend the shelf life and confer health-promoting effects. Additionally, probiotics can be utilized as starter cultures in fermentation of food matrices. They can enhance the level of vitamin B groups in fermented foods as well as mineral absorption through production of short chain fatty acids and pH decrease [47]. Increases of antioxidant activity during fermentation of different food matrices such as apple juice fermented with *L. plantarum* ATCC 14917 [48] and grape beverage fermented with *L. plantarum*, *L. rhamnosus* and *L. casei* [49] have been reported.

The most important challenges in probiotic food development are viability of probiotics during production and shelf life of foods and sensory characteristics of food product. Therefore, selection of suitable strains and food matrices as well as encapsulation of probiotics or addition of prebiotics can be considered as possible solutions.

**Mitigation of acrylamide by probiotics**

Several methods have been potentially explored for decrease of acrylamide level in food products (Figure 2). However, some of these methods cannot be practical due to the requirement of high energy, expensive equipment and detrimental effect on nutritional quality of products
Additionally, they do not remove acrylamide completely and may damage sensory characteristics. In this regard, application of microorganisms such as lactic acid bacteria (LAB) and probiotics has been taken into consideration and can be employed as pre- and post-process mitigation tools.

Two main mechanisms have been proposed to clarify detoxification activity of probiotics toward carcinogens. One is physical absorption of carcinogenic compounds to microorganisms and the second is metabolizing the compounds (such as biotransformation of N-nitro, C-nitro and C-nitrous type mutagens by \textit{L. delbrueckii} subsp. \textit{bulgaricus}) and reduce their health risks. The first mechanism has been more welcomed among researchers because it has been found that inactivated cells show the same ability to remove carcinogens as viable cells. The other possible mechanism by which probiotics can decrease acrylamide formation in food products, is production of L-asparaginase that can be used as a pre-process reduction strategy. The two methods are discussed as following:

\textit{Binding of acrylamide to probiotic cell walls}

It has been highlighted that some probiotic bacteria have capability of binding to carcinogenic compounds through their cell wall, therefore, these compounds would be removed together with the bacteria through the feces. Previous studies have indicated the capability of probiotics to remove toxins such as mycotoxins and heavy metals and recently polycyclic aromatic hydrocarbons, heterocyclic aromatic amines, nitrosamine and acrylamide. The number of studies in respect of removal of acrylamide by probiotics are limited, but the main mechanism of reduction has been attributed to the peptidoglycan in bacteria cell wall. Peptidoglycan is the major constituent of probiotic cell wall (90% of cell wall mass) and is composed of chains of N-acetylglucosamine (GlcNAc) and N-acetylmuramic acid (MurNAc).
that are linked via β-1, 4 bonds and peptidic chains are linked to the lactyl group of MurNAc via covalent bonds [59]. These peptidic chains are various in terms of amino acids residues in different bacterial species.

The ability of 14 strains of LAB to metabolize acrylamide (5 and 10 µg/mL) *in vitro* after 0, 4 and 12 h incubation at 37°C in different pH (3, 5 and 8) was investigated. It was noted that the degree of binding ability to acrylamide was dependent on pH, acrylamide concentration and type of strain. Binding to acrylamide was dependent on incubation time. It was proposed that binding was a rapid process and occurred passively on the bacterial surface [60]. A higher binding of pyrolyzed mutagen at pH 6-7 was reported by Zhang *et al.* [61] which is in consistent with the study conducted by Hernandez-Mendoza *et al.* [62] that pointed out, the extent of binding of *Lactobacillus reuteri* NRRL 14171 and *Lactobacillus casei* shirota was higher at pH 7 than pH 8. It was ascribed to the competition between toxic compounds and protons to bind to the negatively charged binding sites [63]. In another study by Serrano Niño *et al.* [60], the interaction of acrylamide and aflatoxin B₁ with teichoic acids (TA) in the cell wall of 14 LAB was studied. It was announced that there was a relation between components of TA and level of bound acrylamide. Lower levels of glucose, D-alanine or teichoic acid caused a higher degree of binding of acrylamide to the cell wall of bacteria. Hydrogen bonds may be formed between carbonyl oxygen and the amino group between adjacent acrylamide and D-alanine directly attached to position D-4 (L-2) of ribitol. Moreover, the amine group of D-alanine might react with acrylamide units through Michael addition reaction. Also, hydrogen bonds may occur between carbonyl (C=O) oxygen of both AFB₁ and acrylamide, and the hydroxyl groups of either glucose residues or glycerol phosphate substituent attached to the poly (ribitol phosphate) chain [60].
Zhang et al. [50] studied the ability of four strains of LAB (*Lactobacillus plantarum* 1.0065, *L. casei* ATCC393, *Lactobacillus acidophilus* KLDS1.0307 and *Streptococcus thermophilus* KLDS1.0316) to bind acrylamide. It was declared that binding ability of bacterial peptidoglycan to acrylamide ranged was in the range of 33.65-87%. The highest binding ability was recorded for *L. plantarum* 1.0065 followed by *L. casei* ATCC393, *L. acidophilus* KLDS1.0307 and *S. thermophilus* KLDS1.0316. The compositional analysis of cell wall demonstrated that *L. plantarum* 1.0065 contained the highest level of carbohydrate in peptidoglycan structure and there was a positive relation between carbohydrate content and binding ability of peptidoglycan to acrylamide. Furthermore, a positive relation between binding ability and the level of alanine, aspartic acid, glutamic acid and lysine was observed. Alanine had the highest level among amino acids in peptidoglycan and had a remarkable effect on binding of acrylamide. These results confirmed previous studies which expressed that the amine group of D-alanine probably reacts with acrylamide through Michael addition [60]. Adsorption mechanism of *L. plantarum* 1.0665, *L. plantarum* ATCC 8014, *L. plantarum* 806, *L. casei* ATCC 393, and *L. acidophilus* KLDS 1.0307 was explored by [51]. It was figured out that heat-inactivated bacteria had more binding ability compared to untreated ones because of change in the structure of cell wall and providing more binding sites. The heat-inactivated *L. plantarum* ATCC 8014 showed the highest adsorption capacity as a result of possessing the highest specific surface and specific volume. Consistent with aforementioned results obtained by [50, 60], C=O, C-O, and N-H groups were the principal functional groups involved in adsorption of acrylamide by bacterial cell wall. Rivas-Jimenez et al. [2] assessed the ability of *L. reuteri* NRRL 14171 and *L. casei* Shirota in removing dietary acrylamide under simulated gastrointestinal conditions. The results demonstrated that toxin reduction was associated with acrylamide level and bacterial cell concentration. Although
viability reduced in the food model and in simulated digestive system, both bacteria survived in enough concentrations to remove the toxin (32-73%), being *L. casei* Shirota the most effective (70% removal). Moreover, toxin removal was higher in products with lower levels of acrylamide. It was deduced that strains of the *Lactobacillus* genus could be applied in order to decrease the absorption of dietary acrylamide in gastrointestinal tract.

*Reduction of acrylamide by L-asparaginase of probiotics*

It has been elucidated that using the enzyme L-asparaginase (L-asparagine amido hydrolase, E.C. 3.5.1.1.) can be a useful processing aid for reduction of acrylamide formation in food products. It is extensively distributed in animals, plants and living organisms [11]. L-asparaginase hydrolyses L-asparagine to L-aspartate and ammonia, thus suppressing acrylamide formation. It is obtained from several microorganisms including *Escherichia coli*, *Erwinia carotovora*, *Bacillus* sp., *Enterobacter aerogenes*, *Corynebacterium glutamicum*, *Pseudomonas stutzeri* and *Candida utilis* [64]. There are two commercial asparaginase currently available for acrylamide reduction named PreventASe™ from DSM (Heerlen, Netherlands) and Acrylaway from Novozymes A/S (Bagsvaerd, Denmark) obtained from *Aspergillus niger* and *A. oryzae*, respectively [11] [12]. Asparaginase to be used in food industry should be stable over a wide range of pH and temperature as well as high specificity and conversion rate [65].

Recently, it has been stated that *Lactobacillus* species also possess the asparaginase genes that are yet to be characterized [66]. Aishwarya *et al.* [64, 67] cloned the gene of asparaginase from *L. casei* subsp. *casei* ATCC 393 and *L. reuteri* DSM20016 which were expressed in *E. coli*. Onishi *et al.* [68] used L-asparaginase from *Bacillus subtilis* for reducing acrylamide formation in fried potato chips. It was reported that by treating sliced potatoes with the enzyme, 40% of L-
asparagine was converted to L-aspartic acid. Performing pre-treatments including freeze-thawing, drying at 90ºC for 20 min, and vacuum treating for 10 min under decompressed condition resulted in 90% hydrolysis of L-asparagine to L-aspartic acid. Correspondingly, Sanghvi et al. [69] isolated L-asparaginase from B. subtilis KDPS1 with stability over a wide range of physiological conditions like temperature, pH and exposure to metal ions and decreased acrylamide formation in sliced potato by 90-95% compared to untreated potato slices. Considering the limited findings in respect of the presence of asparaginase in probiotics and its activity, more researches are needed to ensure the efficacy of this mechanism in probiotics.

Conclusions and future research

Researches on acrylamide reduction in food products have an attracted attention due to its occurrence in variety of food products and detrimental health effects. In this regard, various approaches have been suggested amongst using probiotics has been found as an efficient method for acrylamide mitigation. Probiotics can detoxify acrylamide through binding to the cell wall which is dependent on factors such as strain type, bacterial population, incubation time, pH, growth phase, and acrylamide concentration. It can be assumed that as detoxification process is associated with cell wall components, probiotics cell fragments obtained from inactivated bacteria can also be effective in reducing the absorbed acrylamide level in the human body. In this regard, probiotics can be consumed along with food products to decrease the health risk of this toxicant. Also, probiotic species which inhabit in GI tract can be incorporated into food products and consumed in order to modulate intestinal permeability and absorption procedures. Although few studies have characterized the compounds involved in binding mechanism, more in vivo researches are required for supporting the obtained results and fill the existing gaps in respect of probiotic action in digestive system and the exact underlying mechanisms. In addition,
it is necessary to assess and confirm the detoxification ability of probiotic in contaminated food products and not only in aqueous solution of toxins. Furthermore, they are a few reports in the literature regarding the presence, identification and characterization of L-asparaginase in probiotics which can be new sources of this enzyme and a promising approach in decrease of acrylamide in food products which would motivate the researches to be oriented toward this field.

Conflict of interest

The authors declare no conflict of interest.

Conflict of interest

The authors declare that there is no conflict of interest.

Acknowledgement

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References and recommended reading

Papers of particular interest, published within the period of review, have been highlighted as:

• of special interest
•• of outstanding interest

References


In this review article, two major carcinogenic compounds formed during maillard reaction are considered regarding their metabolism and toxicity in body, their formation pathways in foods and the ways to reduce their formation in food products.


In this review, acrylamide mitigation strategies including additives and enzymatic approaches have been investigated. The application of asparaginase in acrylamide mitigation, its sources and its activity under different conditions have been reviewed.

A very detailed and well-classified description of acrylamide mechanism of formation in foods, mitigation strategies and risk assessment are presented in this article.


Akgün B, Arci M: Evaluation of acrylamide and selected parameters in some Turkish coffee brands from the Turkish market. Food Addit Contam A 2019, 36(4):548-60.


This review article focuses on formation of acrylamide in cereal products and genetic and agronomic strategies to reduce acrylamide formation in cereal products have been discussed.


[53] Lili Z, Junyan W, Hongfei Z, Baoqing Z, Bolin Z: Detoxification of cancerogenic compounds by lactic acid bacteria strains. *Crit Rev Food Sci 2018*, 58(16): 2727-42. This review describes mechanism of toxic compounds binding to bacterial cell wall and presents a molecular dynamic computer model to stimulate the binding behaviour of *Lactobacillus acidophilus* to a toxic compound.


Figure 1. Mechanism of acrylamide formation in food.
Figure 2. Strategies for mitigation of acrylamide in food products.
Table 1. Occurrence of acrylamide in some food products

<table>
<thead>
<tr>
<th>Food product</th>
<th>Determination method</th>
<th>Analytical range</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coffee</td>
<td>LC-MS/MS</td>
<td>31.1-323.4 µg/kg</td>
<td>[18]</td>
</tr>
<tr>
<td>Iranian traditional bread (Sangak)</td>
<td>LC-MS/MS</td>
<td>5.8-60.6 ng/g</td>
<td>[19]</td>
</tr>
<tr>
<td>Cracker</td>
<td>GC-MS</td>
<td>194-1271 µg/kg</td>
<td></td>
</tr>
<tr>
<td>Corn chips</td>
<td></td>
<td>78-441 µg/kg</td>
<td></td>
</tr>
<tr>
<td>Wafer</td>
<td></td>
<td>687-2497 µg/kg</td>
<td></td>
</tr>
<tr>
<td>Commercial flat breads</td>
<td>LC-MS/MS</td>
<td>24.2-2070 µg/kg</td>
<td>[20]</td>
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<tr>
<td>Cocoa powder</td>
<td>LC-MS/MS</td>
<td>40-440 µg/kg</td>
<td>[21]</td>
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<tr>
<td>Roasted cocoa bins</td>
<td></td>
<td>50-380 µg/kg</td>
<td></td>
</tr>
<tr>
<td>White chocolate</td>
<td></td>
<td>30-90 µg/kg</td>
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</tr>
<tr>
<td>Milk chocolate</td>
<td></td>
<td>&lt; 30 µg/kg</td>
<td></td>
</tr>
<tr>
<td>Fried potatoes</td>
<td>LC-ESI-MS/MS</td>
<td>20-1068 µg/kg</td>
<td>[22]</td>
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<tr>
<td>Potato crisps and snack</td>
<td>LC-MS/MS</td>
<td>529-3300 µg/kg</td>
<td>[23]</td>
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<tr>
<td>French fries</td>
<td>LC-MS/MS</td>
<td>299-750 µg/kg</td>
<td>[23]</td>
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<tr>
<td>Cocoa biscuit</td>
<td>GC-MS</td>
<td>313-383 ng/g</td>
<td>[24]</td>
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<td>Sponge cake</td>
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<td>107.4-198.6 ng/g</td>
<td>[24]</td>
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<td>Dried prunes</td>
<td>LC-ESI-MS/MS</td>
<td>14.7-124.3 µg/kg</td>
<td>[25]</td>
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<tr>
<td>Dried peanuts</td>
<td></td>
<td>10-42.9 µg/kg</td>
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<tr>
<td>Bread</td>
<td>UPLC-MS</td>
<td>119-263 µg/kg</td>
<td></td>
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<tr>
<td>Fried potato dish (rösti)</td>
<td>GC-MS</td>
<td>702 µg/kg</td>
<td></td>
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<tr>
<td>Breads</td>
<td>GC-MS/MS</td>
<td>7.6-165.6 µg/kg</td>
<td>[26]</td>
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<tr>
<td>Biscuits</td>
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<td>4.63-2405 µg/kg</td>
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<td>Sandwich biscuits with cream</td>
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<td>112.6-570.4 µg/kg</td>
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<tr>
<td>Biscuits for infants</td>
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<td>4.63-801.7 µg/kg</td>
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<td>349.5-955.5 µg/kg</td>
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<td>Crackers</td>
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<td>347.8-366.1 µg/kg</td>
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<td>73-108 µg/kg</td>
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<td>Coffee</td>
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<td>152-682 µg/kg</td>
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</tr>
<tr>
<td>Tea</td>
<td></td>
<td>10-97 µg/kg</td>
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<tr>
<td>Potato crisps</td>
<td>GC-MS</td>
<td>174-3444 ng/g</td>
<td>[27]</td>
</tr>
<tr>
<td>Corn-based extruded snacks</td>
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<td>123-447 ng/g</td>
<td></td>
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<tr>
<td>Roasted peanuts</td>
<td></td>
<td>21 ng/g</td>
<td></td>
</tr>
<tr>
<td>Savoury snacks</td>
<td></td>
<td>44-813 ng/g</td>
<td></td>
</tr>
<tr>
<td>Bread</td>
<td>GC-MS</td>
<td>165.62-289-72 µg/kg</td>
<td></td>
</tr>
<tr>
<td>Breakfast cereal</td>
<td>GC-MS</td>
<td>7.46 548 µg/kg</td>
<td>[28]</td>
</tr>
<tr>
<td>Infant food</td>
<td></td>
<td>7.46-174 µg/kg</td>
<td></td>
</tr>
<tr>
<td>Baby bread</td>
<td></td>
<td>7.46-660 µg/kg</td>
<td></td>
</tr>
</tbody>
</table>
*LC-MS/MS: liquid chromatography with tandem mass spectrometry; GC-MS: gas chromatography-mass spectrometry; LC-ESI-MS/MS: Liquid Chromatography Electrospray Ionization Tandem Mass Spectrometry; UPLC-MS: ultra-performance liquid chromatography-mass spectrometry.